CLINICAL PROFILE OF CHILDREN WITH ADENOTONSILLAR HYPERTROPHY AT MOI TEACHING AND REFERRAL HOSPITAL, ELDORET, KENYA

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THIS THESIS IS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS OF THE MASTERS IN MEDICINE PROGRAMME, M.MED (CHILD HEALTH AND PEDIATRICS), MOI UNIVERSITY, SCHOOL OF MEDICINE

DECLARATION

Student's declaration

This research thesis is my original work done during my studies in the Masters of Medicine in Child Health and Pediatrics degree course of Moi University, and has not been presented in any other university or academic forum.

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ACRONYMS AND ABBREVIATIONS

BMI	Body Mass Index
ENT	Ear, Nose and Throat
IREC	Institutional Research and Ethics Committee
MTRH	Moi Teaching and Referral Hospital
MUAC	Mid-Upper Arm Circumference
OSA	Obstructive Sleep Apnea
STOP-BANG	The Loud Snoring, Tiredness, Observed apnea, high blood Pressure (STOP)-Body mass index (BMI), Age, Neck circumference, and gender (Bang)
WHO	World Health Organization

OPERATIONAL DEFINITIONS

Adenotonsillar Hypertrophy	Enlargement of the adenotonsillar tissue diagnosed by the Ear, Nose and Throat surgeon on Postnasal Space X-Ray or posterior rhinoscopy
Pulmonary Hypertension	A mean pulmonary artery pressure greater than 25 mmHg (millimetres of mercury) by Echocardiography; mild pulmonary hypertension: 25-40 mmHg, moderate pulmonary hypertension: 40-55mmHg, severe pulmonary hypertension >55mHg
Adenoid Facies	Facial features that are found on a child with adenotonsillar hypertrophy, characterised by two or more of the following features: i. Underdeveloped thin nostrils ii. Short upper lip and hypoplastic maxilla iii. Prominent upper teeth iv. Crowded teeth v. A narrow upper alveolus vi. A high arched palate
Dennie's lines	Horizontal creases under both lower eyelids
Nasal pleat	The horizontal crease just above the tip of the nose produced by the recurrent upward wiping of nasal secretions
Allergic shiners	Bilateral shadows under the eyes, produced by chronic venous congestion

ABSTRACT

Background: Adenotonsillar hypertrophy is a leading cause of upper airway obstruction in children and a common reason for paediatric referrals to Ear, Nose and Throat (ENT) specialists. It can lead to complications such as pulmonary hypertension and failure to thrive. Most of the children admitted to the paediatric wards in Moi Teaching and Referral Hospital (MTRH) with adenotonsillar hypertrophy are admitted when complications have already set in. There is paucity of local data on children with this condition.

Objective: To determine the clinical profile of children presenting with adenotonsillar hypertrophy at MTRH.

Study Methods: This was a cross-sectional descriptive study conducted in the ENT Clinic and the Paediatric Ward at MTRH, from December 2014 to May 2016. The study population included children aged between 2 months and 13 years clinically diagnosed with adenotonsillar hypertrophy. Data was collected using a structured data collection tool. Echocardiography was done on all patients to determine the pulmonary pressures. Statistical analysis was performed using SAS version 9, and a p-value of less than 0.05 was used to define statistical significance. Demographic and clinical characteristics were summarized using descriptive statistics. Categorical variables were summarized as frequencies and percentages. Continuous variables were summarized as mean and standard deviation. The test for association between categorical variables was conducted using Pearson's Chi Square. Data was presented using table shells and graphs.

Results: A total of 105 participants were recruited into the study. There were 59 males (56.2%). The mean age was 3.5 years. The most common presenting symptoms were nasal congestion and obstruction in 100 (95%) of the participants, mouth breathing while asleep in 96 (91.4%), recurrent sore throat in 76 (72.4%) and snoring in 64 (61.0%) participants. The most common physical examination findings were mouth breathing, adenoid facies, tachycardia, tachypnea and tonsillar enlargement. Pulmonary hypertension occurred in 24.8% of the patients, and was associated with mouth breathing while asleep (OR 3.11, CI 95% 1.43-8.97; p=0.024), adenoid facies (OR 2.45, CI 95% 0.57-10.12; p=0.029), hypoxia (OR 4.11, CI 95% 2.56-8.91; p=0.037) and tonsils Grade 4 (OR 2.55, CI 95% 0.47-3.22; p=0.043).

Conclusion: The most common presenting complaints of adenotonsillar hypertrophy are nasal blockage, mouth breathing while asleep, snoring and recurrent sore throat. A quarter of the children with adenotonsillar hypertrophy had features of pulmonary hypertension on echocardiography.

Recommendation: Early recognition and treatment of adenotonsillar hypertrophy is recommended so as to avert the development of long-term complications. Echocardiography should form part of the routine investigations for children with adenotonsillar hypertrophy.

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CHAPTER ONE

INTRODUCTION

1.1 Background

The adenoids are anatomically located on the posterior nasopharyngeal wall and develop from a sub-epithelial infiltration of lymphocytes in the 16th week of gestation (Blum and McGowan, 1997). Adenoid hypertrophy is the unusual enlargement of the adenoidtonsils, as first described by the Danish physician Wilhelm Meyer (1824-1895) in Copenhagen in 1868, (Sie, Perkins and Clarke 1997). Upper airway obstruction in childhood is due to several causes, and among them, the commonest is the adenotonsillar hypertrophy. Adenotonsillar hypertrophy also represents one of the main causes of pulmonary hypertension and corpulmonale in children.

The adenoids are a component of the Waldeyer's ring, alongside the palatine and the lingual tonsils. Adenoids are present at birth and enlarge, along with the tonsils, until children are aged 5-7 years, (Henry and Thompson, 2005).The adenoids usually enlarge or undergo hyperplasia during these earlier years, and begin to shrink in size by adolescence. The prevalence of adenotonsillar hypertrophy decreases with age. Due to this physiological atrophy of the tissue, adenotonsillar hypertrophy is rare in children in their teen years (Eziyi, Amusa and Nwawolo, 2014).

1.1.1 Clinical presentation

The major predisposing factors for upper airway obstruction are anatomic narrowing, muscle weakness and abnormal neural regulation (Jones, Hilgen and Phillips, 1998). The adenoids are in the midline within the nasopharynx, and the Eustachian tubes open laterally to them on either ear. Obstruction due to adenotonsillar hypertrophy can lead to both chronic sinusitis and recurrent otitis media. The child may also

present with an irritating cough as a result of the involvement of the mucosa over the larynx and vocal cords (Jones, et. al, 1998)

Enlarged adenoids can become nearly the size of a ping pong ball and completely block airflow through the nasal passages (Anton and Prescott, 2009). Children thus have increased work of breathing as they are breathing against resistance. These children revert to mouth-breathing. Adenotonsillar hypertrophy leads to a dentofacial growth anomaly that is defined as "adenoid facies" (Jones, Hilgen and Phillips, 1998).

In the otherwise healthy child, parents principally report snoring during sleep (Brockmann and Bertrand, 2012). Some children snore loudly and have audible intermittent gasps (Sogut, Altin, Uzun and Tomac, 2012). Some demonstrate paradoxical chest and abdominal wall movements, laboured breathing with retractions, cyanosis, sweating, and restlessness, (Friendmann and La Rossa, 1999). Often, these children prefer sleeping with their head and neck extended and their mouth wide open (Davis, Gance-Cleveland, 2007). Other recognized complaints amongst children with adenotonsillar hypertrophy include restlessness, frequent nightmares, enuresis, difficulty getting up in the morning, excessive daytimesleepiness, hyperactivity and/or behaviour problems, daytime mouth breathing and altered sleep patterns (Dayyat, Sans Capdelliva and Gozal, 2009).

1.1.2 Clinical Evaluation

Children with adenotonsillar hypertrophy should undergo a complete physical examination with special attention to structures of the upper airway (Aydin, Sanli and Celebi, 2008). Accurate vital signs, including measurement of blood pressure; plots of the child's height, weight, and body mass index (BMI) by age on a gender-specific growth chart should be obtained. Height and weight should always be measured. The

child's growth should also be defined as whether normal or abnormal, (Benga Norte, 2008)

The child's face should be examined for any craniofacial anomalies. Inspection should be done for midfacial hypoplasia, a flat nasal bridge, or facial asymmetry; the jaw should also be examined for micrognathia (an abnormally small jaw) or retrognathia (the jaw is recessed), (Chinawa, Onuorah, and Awoere, 2015). Adenoid facies should also be looked for, with mouth breathing, nasal speech, and periorbital swelling. Nasal patency should be assessed and evaluation done for signs of allergic rhinitis, nasal polyps and growths, and septal deviation, (Chinawa, et.al, 2015). The nasal passages are examined for mucosal swelling, cobblestone pattern of the mucosa, and reduced nasal airflow, (Gebber, O'Connor and Haler, 1996). The size and position of tonsils and uvula should also be assessed, particularly noting hypertrophy or malformation. The adenoids are assessed by posterior rhinoscopy, lateral neck radiographs, and Postnasal Space (PNS) radiographs, (Tatlipinar, Duman, Uslu and Egeli, 2011). Neck CT Scans are also useful in evaluation of soft tissue masses in the neck, (Granzotto, Aquino, Florence and Lubianca, 2010).

Once the diagnosis has been established and its severity assessed, adenotonsillectomy is usually the first line of treatment, (Santos and Cipolotti, et. al, 2005). Adenoidectomy is most often performed because of nasal obstruction, but is also performed to reduce recurrent or chronic otits media (Budey, Arroliga and Matthay, 2003).

1.1.3 Associated Comorbidities

Major morbidities associated with upper airway obstruction in childhood include obstructive sleep apnea, failure to thrive, difficulty concentrating and/or developmental delay, behavioural problems, hypertension, pulmonary hypertension, and, ultimately, corpulmonale. Chronic upper airway obstruction with laboured breathing may result in the development of a pectusexcavatum. Concomitant gastroesophageal reflux is likely to be exacerbated by obstructive sleep apnea. In children with failure to thrive (FTT), treatment of obstructive sleep apnea leads to resolution of the somatic growth disturbance. Similarly, pulmonary hypertension resolves, (Urshitz and Guenther,et. al, 2003).

1.2 Problem Statement

Diseases of the tonsils and adenoids are among the most common problems seen by physicians who care for children and adolescents. Adenotonsillar hypertrophy is a common condition in childhood, whose serious complications of pulmonary hypertension and cor-pulmonale are common and devastating. They are also associated with failure to thrive but local prevalence is unknown[5]. In a study by Marangu D. et al, the prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH was 21%.

Children with adenoid and adenotonsillar hypertrophy are treated daily either in an outpatient set up, in an inpatient setting, and are also seen as frequent referrals to otolaryngologists. A full sociodemographic and clinical assessment, including their anthropometric measurements and screening for possible complications is not usually part of routine clinical evaluation. Most of these complication are therefore either missed or diagnosed late

Most of these children initially visit the paediatric clinics and are seen by paediatrician. The long term complication of Pulmonary Hypertension, Failure to Thrive and Corpulmonale are also managed by paediatrician, hence it is crucial that the paediatrician is conversant with the clinical presentation, management, complications and prevention of upper airway obstruction from adenoidal hypertrophy.

1.3 Study Justification

Adenotonsillar hypertrophy is the most common cause of upper airway obstruction in children and a common reason for paediatric referrals to otolaryngologists (ENT specialists), yet local statistics are unknown.

Age prevalence shows that highest rates of adenotonsillectomy are done in children aged 2-4 years due to upper airway obstruction, associated with failure to thrive. This is a crucial point in the growth and development of any child, hence the need to carry out thorough physical assessment, including anthropometric measures so that complications such as Failure to Thrive are detected and managed early enough.

No similar study has been carried out to assess the possible complications of chronic obstruction of the upper airways. There is paucity of data on complications of adenotonsillar hypertrophy. This study also seeks to establish the importance of routine echocardiography to detect existence or absence of pulmonary hypertension in children with adenotonsillar hypertrophy.

1.4 Research Question and Objectives

1.4.1 Research Question

1. What is the clinical profile of pediatric patients presenting with adenotonsillar hypertrophy at MTRH?
2. What is the prevalence of pulmonary hypertension in these patients?

1.4.2 Study Objectives

Broad Objective

To determine the clinical profile of patients presenting with adenotonsillar hypertrophy at Moi Teaching and Referral Hospital

Specific Objectives

i. To describe the presenting clinical characteristics of children with adenotonsillar hypertrophy at MTRH

ii. To determine the prevalence of pulmonary hypertension in children with adenotonsillar hypertrophy at MTRH

CHAPTER TWO
LITERATURE REVIEW

Adenotonsillar disorders are among the most common problems seen by paediatricians (Yates, 1988). Understanding the classification, pathophysiology, evaluation, and treatment of adenotonsillar hypertrophy and obstructive sleep disorder is not only important for practicing otolaryngologists but also for all health care providers caring for children and adolescents such as primary care physicians, dentists, and orthodontists (Sebusiani, Pignatari, Arminio and Stamm, 2003).

Upper airway obstruction in children may result from different causes, such as craniofacial anomalies, subglottic stenosis (a narrowing of the subglottic airway), choanal atresia and more commonly adenotonsillar hypertrophy, (Blum, et. al, 2004). Chronic obstruction of the upper airways is usually associated with sleep apnea syndrome and, in more severe cases, development of pulmonary hypertension and cor pulmonale (Sebusiani, et. al, 2003)

Children with obstructive disorders, especially due to adenotonsillar hypertrophy are predisposed to hypopnea or apnea (Xu, Jiaguing, Shen, 2008). During apnea, there is a progressive decrease in oxygen levels and increase in Co_2 levels, (Fujioka, Young and Girdany, 1979). Hypercapnia and hypoxemia provoke respiratory acidosis and consequently vasoconstriction of the pulmonary artery. In addition to increased venous return due to increased pulmonary resistance, there is also increased venous return to the right cardiac chambers, which is facilitated both by the decubitus horizontal position during sleep and by intra-thoracic pressure which becomes more negative due to respiratory effort against the obstructive area, (Miman and Kirzali, 2000). Signs and symptoms of pulmonary hypertension may not be obvious at first,

but they can be worsened over time and can begin to limit daily activities. Symptoms of Pulmonary hypertension include: breathlessness, chronic fatigue, dizziness, faintness, swollen ankles and legs, chest pain, especially during physical activity (Vivianne, Feller, et. al, 2013). Functional classification of pulmonary hypertension include as follows, (Er, Ederer, Nia, Caglayan, 2012).

- (I) Without limited physical activity and comfortable at rest; ordinary physical activity does not cause dyspnea or fatigue, chest pain or near syncope.
- (II) Mild limited physical activity but comfortable at rest; ordinary physical activity cause undue dyspnea or fatigue, chest pain or near syncope.
- (III) Marked limited physical activity but comfortable at rest; less than ordinary activity cause undue dyspnea or fatigue, chest pain or near syncope.
- (IV) Inability to carry out any physical activity without symptoms; discomfort is increased by any physical activity

Pulmonary hypertension, regardless of the cause, is a feature of advanced disease. For children who have had symptoms for a short duration of time, this remodeling of the pulmonary vasculature due to chronic hypoxia has not yet began (Yilmaz, Onrat, Atlunta, 2005). However, as the symptoms progress and the secondary changes begin to set in, pulmonary hypertension becomes evident, (Moghaddam, Bavil, et. al, 2010).

2.1 Clinical presentation

Adenotonsillar hypertrophy can present in a number of ways in the paediatric population, ranging from habitual or primary snoring, to failure to thrive due to obstructive sleep apnea and other neurocognitive disturbances, (Wood, 1992). Children with adenotonsillar hypertrophy may also present with recurrent tonsillar infections, nasal obstruction; mouth breathing, snoring and, in more severe cases,

sleep apnea, irritability, poor progression in school and daytime somnolence (Davis et. al, 2007). Children can also present with characteristic facial features called adenoid facies. Chinawa et. al recorded the prevalence of adenoid hypertrophy as 1.3% in a study carried out to demonstrate the prevalence of nasal diseases among school going children in Nigeria. The peak age of diagnosis of adenotonsillar hypertrophy as shown by Chinawa is between 13-36 months while the mean age of presentation is 32.6±17.9 months. The commonest symptoms in children with adenotonsillar hypertrophy as found by Chinawa in their study were cough, catarrh and snoring and mouth breathing especially at night, (Chinawa, et.al, 2015). Eziyi J.A and Amusa Y.B, et al found the prevalence of adenoid hypertrophy in Nigerian children to be 7.7%. Aydin in his study of 10,298 primary school children aged 6-13 years in Brazil reported a prevalence of degree II and III adenoid hypertrophy as 49.4%[53] whereas, Santos reported a higher prevalence of 66.4% in primary school children in Turkey.

In a population based cross-sectional study carried out by Brockmann PE and Bertrand et al, to assess the prevalence of habitual snoring and associated neurocognitive consequences among Chilean school aged children, the prevalence of habitual snoring was 18%. Children with habitual snoring showed significantly lower school grades.

Sogut A, Altin R. and Uzun et. al carried out a cross-sectional study to investigate the prevalence of sleep-disordered breathing (SDB) and obstructive sleep apnea (OSAS) in Zonguldak, northwestern Turkey and associated symptoms in 3 to 11-year-old Turkish children. Symptomatic children were identified by using a self-administered questionnaire and were classified into three groups: nonsnorers, occasional snorers,

and habitual snorers. All habitual snoring children were invited to undergo polysomnography (PSG). Nine hundred fifty-four children (79.5%) were nonsnorers, 205 (17.2%) were occasional snorers, and 39 (3.3%) were habitual snorers.

Kara C.O et al carried out a study to investigate the prevalence of tonsillar hypertrophy and associated oropharyngeal symptoms in primary school children. The study was performed in two primary schools which were chosen randomly in Denizli. The study population consisted of 1211 (636 boys, 575 girls) primary school children between 6 and 13 years old (mean 9.3+/-2 years). Prevalence of tonsillar hypertrophy in school children was found as 11% in the school children. There was a statistically significant association between tonsillar hypertrophy and history of frequently having tonsillitis, habitual snoring, observed apnea, oral breathing during sleep and difficulty eating.

2.2 Evaluation of Complications

The most important diagnostic tool for upper airway obstruction is a thorough history and physical examination, (Jacobsons, 1989). Many times, management of a patient with upper airway obstruction must start simultaneously with the diagnostic process. Airway resistance varies inversely with the fourth power of the radius at the point of obstruction, thus small changes in the underlying pathology may dramatically worsen respiratory airflow, (Khosh and Lebovics, 2001). Lateral neck radiographs and postnasal space radiographs are important in the evaluation of upper airway obstruction due to adenotonsillar hypertrophy, (King and Sheehan). Posterior rhinoscopy, nasopharyngoscopy and CT Scans of the neck are also important investigations that should be carried out in the evaluation of a child with upper airway obstruction, (Aboussouan and Stoller, 2009).

Doppler echocardiogram is a accurate method to diagnose pulmonary hypertension, (Sebusiani, et. al, 2003). Electrocardiograms have also been used in the assessment of pulmonary hypertension in infants with adenotonsillar hypertrophy associated with sleep apnea, (Jacobs, Teague, and Bland, 1981). Nevertheless, these have revealed low sensitivity. According to Sebusiani, (2003), Doppler echocardiography is highly useful when diagnosing pulmonary hypertension in infants with adenotonsillar hypertrophy, as it is considered a very safe, practical and non-invasive investigation, (Friedberg and Feinstein, 2007).

A detailed echocardiographic assessment should be performed in all patients with suspected pulmonary hypertension. Transthoracic echocardiography provides a number of measures and views that are essential for the assessment of the hemodynamics of the heart, (Simmoneau, et.al, 2009).

The M-mode provides one-dimensional information allowing fine measurement of the heart s' dimensions. Features of pulmonary hypertension on this mode include increased right ventricular wall dimension and thickness, (Aduen and Castello, et.al, 2011).

2-dimensional echocardiography permits structures to be viewed moving in real time in a cross-section of the heart. Right atrial enlargement, right ventricular dilatation and hypertrophy, paradoxical septal motion, and dilatation of pulmonary valve and trunk are features of pulmonary hypertension that are appreciated using 2-dimensional echocardiography, (Arcasoy, et. al, 2003).

Doppler echocardiography assesses blood flow with regard to direction and velocity. The practice of cardiology has been greatly transformed by the use of this modality in that pulmonary arterial pressures can be assessed non-invasively. Doppler

echocardiography has shown excellent correlation with cardiac catheterization (correlation co-efficient r = 0.93, standard estimate error 8mmHg) demonstrating its utility in the monitoring and screening of pulmonary hypertension, (Yock and Popp, 1984).

Estimation of systolic pulmonary arterial pressures (sPAP) is based on the peak velocity of the jet of tricuspid regurgitation arrived at by employing the simplified Bernoulli equation. The reproducibility and reliability for estimating sPAP using echocardiography is well established, (Chemla and Castelain, 2009).

Following the current consensus definition of pulmonary hypertension of mPAP> 25 mmHg at rest, numerous studies have looked at formulae for evaluating mPAP that correlate with cardiac catheterization. A well investigated equation proposed by Aduen et al and Chemla et al to estimate mPAP (mPAPest) is calculated as systolic PAP (sPAP) x 0.61 +2 mmHg. Other equations documented in the literature that have been used to estimate mPAP include the Syyed and the Mahan formulae which are-comparable with regard to sensistivity, specificity and predictive values when compared to cardiac catheterization. The Chemla equation is the simplest to apply, (Chemla, Castellain, Provencher, et. al, 2009).

A cross-sectional study carried out by Marangu D. et al revealed that the prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH was 21.1%. Clinical screening and echocardiography evaluation was concluded as extremely vital for early identification and prevention of pulmonary hypertension.

In a study conducted at São Lucas Hospital, by Viviane Feller Martha and José da Silva Moreira et al.,(2013), thirty-three pediatric patients with adenotonsillar hypertrophy and evidence of obstructive upper airways complaints were treated with

adenotonsillectomy. All 33 patients underwent echocardiogram before and after the surgery with measurement of the pulmonary arterial pressure (PAP). Similar measurements were taken in 10 normal controls. Pulmonary hypertension was confirmed in 12 (36%) of the 33 patients with adenotonsillar hypertrophy.

El-Hoshy, et. al, in a study done in Egypt in 2003, detected pulmonary hypertension in 12 out of 60 children, representing a point prevalence of 20%. In 2005, in a study carried out to assess the effect of adenotonsillectomy on the pulmonary pressures in children with adenotonsillar hypertrophy, Elmofty et al found that eight out of thirty (26.7%) children had pulmonary hypertension. In this study, pulmonary hypertension was defined as sPAP>30mmHg.

In Turkey, two studies were carried out by Naiboglu et al (2008) and Yilmaz and colleagues, (2005), on 39 children aged 3 to 10 years and 52 children aged 4 to 11 years respectively. Naiboglu established that every child with adenotonsillar hypertrophy had some probability of having pulmonary hypertension regardless of his or her disease severity, and thus made a recommendation of echocardiographic examination in all children with adenotonsillar hypertrophy.[34] Yilmaz and colleagues found a point prevalence of pulmonary hypertension 51.9% , that is in 27children out of 52 children. In this study, pulmonary hypertension was defined as an mPAP> 20mmHg using the Mahan formula. The mPAP in these children significantly decreased after adenotonsillectomy.

In a study carried out in Iran by Moghaddam et. al, (2010), evaluating subclinical pulmonary hypertension in 55 children with adenotonsillar hypertrophy with symptoms of upper airway obstruction, 7.3% of children had pulmonary hypertension preoperatively. Pulmonary hypertension in this study was defined as mPAP> 25mmhg determined by the Mahan formula. In a study carried out in Brazil, Granzotto et al

reported a prevalence of pulmonary hypertension of 13% in 45 children with sleep disturbed breathing as a result of adenotonsillar hypertrophy scheduled for adenotonsillectomy. Pulmonary hypertension was defined as sPAP>30mmHg or an estimated mPAP> 20mmHg determined using the Chemla formula. A recommendation was made for these children to have supplementary studies with echocardiography or be given preference for surgery.

CHAPTER THREE

METHODOLOGY

3.1 Study Design

This is a cross-sectional study

3.2 Study Site

The study was conducted in The ENT Clinic and the Paediatric Ward at Moi Teaching and Referral Hospital.Moi Teaching and Referral Hospital.

3.3 Study Period

The study was carried out from December 2014 to May 2016

3.4 Study Population

The study population consisted of children aged between 2 months and 13 years clinically diagnosed with adenotonsillar hypertrophy from both the ENT clinic and pediatric wards of MTRH. These were recruited consecutively until the desired sample size was attained.

3.4 Inclusion and Exclusion Criteria

3.4.1 Inclusion Criteria

1. Children aged 2 months to 13 years attending the ENT Clinic or admitted to the wards with adenotonsillar hypertrophy

3.4.2 Exclusion Criteria

1. Children who have had adenotonsillectomy done
2. Children with congenital anomalies- craniofacial malformations and congenital heart disease

3.5 Sample Size:

Sample size was computed using the Fisher's formula.

$$n = \frac{Z^2{}_{\alpha/2} * p(1-p)}{d^2}$$

Where;

n = Is the anticipated sample size to be considered for the study

$z_{\alpha/2}$= 1.96, the standard normal variate

p= estimated prevalence of children who have adenotonsillar hypertrophy and are diagnosed with pulmonary hypertension; the prevalence of pulmonary hypertension in children with adenotonsillar hypertrophy at KNH is 21.1 % (95% CI 14.3% to 29.4%), as revealed by Marangu D., et al.

d= margin of error at 5%

Calculating sample size yielded the following figure;

$$n = \frac{1.96^2 \times 0.21 \times 0.79}{0.05^2}$$

n= 254 subjects

Adjusting for finite population (n_f): out of 433 patients seen at the ENT clinic in 2013, 253 had a diagnosis of adenotonsillar hypertrophy

(253/12) x 8= 169

N=169 (an estimated 169 patients will be seen in the 8 months that the study will be carried out)

$n_f = n_0/(1+n_0/N)$

$n_f = 254/(1+254/169)$

$n_f = 254/2.5 = 101.6$

$n_f = 102$

Therefore the sample size was calculated as a minimum sample of 102 subjects.

All the children with the diagnosis of adenotonsillar hypertrophy were consecutively selected until the desired sample size was achieved

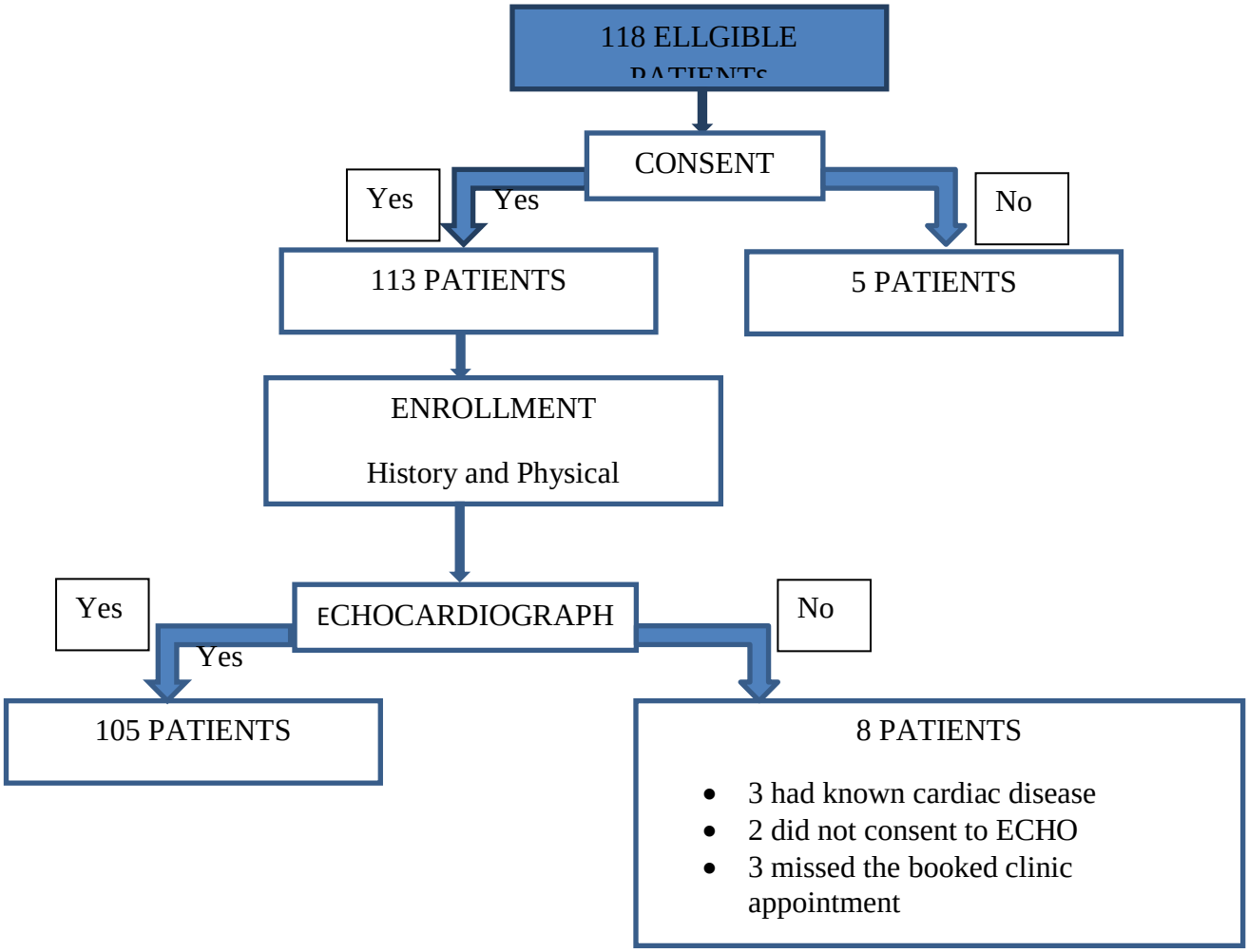

Figure 1: representation of the study participant Enrolment on flow chart

3.6 Study Procedure

At the beginning of the study period, the primary investigator underwent a training and orientation session on the basics of echocardiography and how to measure pulmonary arterial pressures and right ventricular function, including right ventricular systolic pressures, by echocardiography. Moi Teaching and Referral Hospital has an echocardiograph machine that is specific for research purposes. This was the machine that was used for echocardiography. The echocardiographs were thus done on the children at no cost on the parent or guardian. This was done with the assistance of the Echocardiography Technicians, and guidance on interpretation sought from the cardiologist.

The clinical members of staff working at the ENT department and the Paediatric ward were then sensitized on the study. This involved giving a talk to the clinicians and nurses to inform them on the main purpose of the study, on how children with adenotonsillar hypertrophy were to be recruited for the study and on how the study was going to benefit the children. All children seen at the ENT department or admitted to the Paediatric Wards with adenoid or adenotonsillar hypertrophy during the study period and who met the inclusion criteria were recruited and data on the same collected using a standardized data collection tool.

History and Physical Examination

The main signs and symptoms included snoring, observed apnea while sleeping, hyponasal speech, regressed milestones or failure to thrive were captured in the data collection tool as obtained by direct questions to the mother or guardian of the child. The child's biodata was also recorded, and clinical evaluation done, with accurate recording of the age, weight, height, mid-upper arm circumference, and for the older children, the body mass index. The systemic examination findings were also

documented by the principal investigator and the research assistant. Physical examination was done from head to toe, and the findings of inspection, palpation, percussion and auscultation of the chest for respiratory and cardiovascular systems documented in the data collection tool. The same was also done for the abdominal examination. The patient's height or length, weight, and Blood Pressure were also taken and recorded, then charted on the appropriate anthropometric growth chart and Blood Pressure chart, and the corresponding percentile derived. Examination for adenoid facies was also done by the primary investigator and the research assistant. The features assessed for adenoid facies included underdeveloped thin nostrils, short upper lip, prominent upper teeth, crowded teeth, narrow upper alveolus, a high arched palate and a hypoplastic maxilla, Dennie's lines, a nasal pleat and allergic shiners.

Recumbent length was measured for the children less than 2 years. This was done by the principle investigator, assisted by 2 people, one of whom included the research assistant. One person positioned the child supine on the measuring board, and the other took the measurement, reported it verbally, and recorded it in the data collection tool, to the nearest 0.1 cm.

In children older than two years, the standard height was measured. This measurement was taken using a heightometer, with the participant having removed their shoes. The measurement was reported verbally, recorded to the nearest 0.1 cm and recorded in the data collection tool.

The weight was measured using a properly calibrated weighing scale. The infants and toddlers were weighed without clothing, but with a diaper, the measurement reported verbally, and recorded in the data collection tool, to the nearest 0.1 kg.

The MUAC was measured on the left upper arm, at the midpoint between the tip of the acromion process and the olecranon process on the elbow. This was done for children between 6 months and 5 years. The measurement was also noted verbally, and recorded on the data collection tool, to the nearest 0.1 cm.

Blood pressure was measured using a standard aneroid blood pressure machine with an appropriately sized paediatric cuff and a stethoscope to auscultate the Kortkoff sounds. The blood pressures were then recorded and plotted on separate standardized blood pressure charts for boys and girls.

The oxygen saturations were measured using a hand held pulse-oximeter and recorded as a percentage. Oxygen saturations of less than 90% were defined as hypoxia.

Echocardiography

The research assistant, who had been trained earlier, identified children with a confirmed diagnosis of adenotonsillar hypertrophy as they attended the ENT Clinic. After history taking, physical examination and investigations done at the ENT clinic, he would direct them to the special Echocardiography room, where the echocardiograph was done and the results entered in the data collection tool. The Echocardiograph was used to determine the prevalence of pulmonary hypertension in these children by measuring the right ventricular systolic pressure and mean pulmonary arterial pressure and recording it accordingly.

Echocardiography was performed by two trained Echorcardiography Technologists, assisted by the principle investigator, and reviewed by the paediatric cardiologist, who read the images separately, and were all blinded to the state of the patient, with regard to their presenting complaints, the history as taken by the principle investigator and

the research assistant, and the physical examination findings. The echocardiographs were either reported as normal or abnormal.

Transthoracic echocardiography was performed on all patients with the use of a portable Phillips CX50 Color Ultrasound System echocardiography machine. Cardiac measurements were performed according to the guidelines of the American Society of Echocardiography. M-mode, 2D echo and Doppler echocardiography were employed *(See Appendix 11)*

Systolic pulmonary artery pressure (sPAP) was measured using the modified Bernoulli equation applied using the tricuspid regurgitation jet. Mean pulmonary artery pressure (mPAP) was then derived using the Chemla equation = (0.61* sPAP) +2mmHg.M Pulmonary hypertension was defined as a an estimated mPAP of >25mmHg. Any other additional findings were also recorded. Majority of the young infants were pacified with breastfeeding by the mother or distraction by the clinician.

Assessment of the Right Heart Function and the Tricuspid Valve

The Assessment of the Tricuspid Valve function was further carried out in the assessment for pulmonary hypertension. This was through the measurement of the following parameters:

- Tricuspid Velocity (TRV)
- End Diastolic Pulmonary Regurgitant Velocity (PRV)
- Right Ventricular Outflow Tract Acceleration Time (RVOT)
- TAPSE- The Tricuspid Annular Plane Systolic Excursion
- The Right VentricuarIsovolumetric Relaxation Time (IVRT)
- Inferior Vena Cava Diameter
- Right Atrial Volume

- The Right Ventricle Myocardial Performance Index
- Tissue Dopple Index (TDI) of the Right Ventricle free wall
- Cardiac Output Pressure
- Right Ventricular Systolic Pressure (RVSP)
- The mean pulmonary arterial pressure (mPAP) was derived after subjecting the ECHO-derived systolic pulmonary pressure sPAP, as $4(TRV)^2$ to the Chemla Equation:

$$(0.61*SPAP + 2\ mmHg)$$

When pulmonary stenosis is absent, the Right Ventricular Systolic Pressure (RVSP) is assumed to be equivalent to the systolic pulmonary artery pressure, sPAP, and can be calculated from the TRV using the Bernoulli equation: systolic PAP= $RVSP=4(TRV)^2 + P_{ra}$.

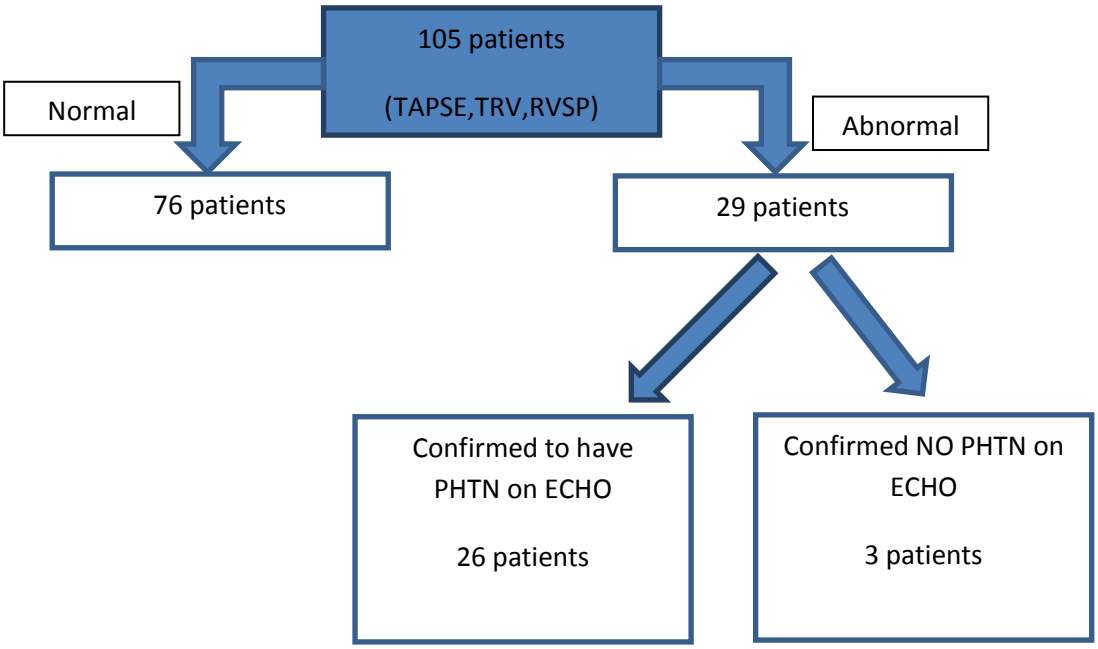

Figure 2: The Interpretation of Echocardiographs As Done On Study Participants

3.7 Data Management

3.7.1 Data Analysis

Statistical analysis was performed using SAS version 9.3 (SAS Institute, Cary, North Carolina, USA). Demographic, clinical and radiological characteristics were summarized using descriptive statistics. Frequency listings and percentages were used to describe categorical variables. For continuous variables descriptive statistics such as mean and median were used. . Categorical variables were summarized as frequencies and percentages while the continuous variables were summarized as mean and standard deviation or median and the corresponding inter quartile range. The test for association between categorical variables was conducted using Pearson's Chi Square and a p-value of less than 0.05 was used to define statistical significance. Correlation analysis was done to determine the association between the variables. Multinomial logistic regression was performed to explain the impact of predictor variables in terms of odds ratios. The prevalence of pulmonary hypertension was

calculated as a percentage of the number of confirmed cases by echocardiography, against the total number of participants. Data was presented using table shells, pie charts, bar graphs and line graphs.

3.8 Ethical considerations

Approval was sought from the Institutional Research and Ethics Committee at Moi University. Informed and written consent was obtained from the mothers or guardians of the children, and assent from children aged 7 years and above.

Confidentiality was upheld in data handling. Patients were de-identified using serial numbers. Data was collected and entered into the data collection tool by the principal investigator and the research assistant. The data collection tool did not have any information to identify the participants or to connect the individual subjects to their responses. Data collection tools were filed and stored in lock and key drawers accessible only to the principal investigator and the research assistant. Data was keyed and entered into a spreadsheet and stored accordingly in a password protected database and laptop, accessible only to the principal investigator and the research assistant. Information was only transferred to persons who were authorized to access it.

Study participants received standard treatment, and no incentives were given to convince participants. The primary investigator would share the findings and recommendations of the study accordingly.

CHAPTER FOUR
RESULTS

4.1 Patient Characteristics

A total of 105 participants were recruited into the study. The sample consisted of 59 males (56.2%) and 46 females (43.8%), with a male to female ratio of 1.3:1. The mean age was 3.5 ±3 years, with a minimum of 0.3 and a maximum of 7.0 years. On nutritional status, 86 of the participants (81.9%) were well-nourished, with the weight for height in the 5^{th}-95^{th} percentile. 15 children (14.3%) were underweight, and 4 children (3.8%) were overweight (Table 2).

The mean duration of symptoms was 3.0 (±3.4) months. Eighty six of the patients (81.9%) were referred from elsewhere. Forty percent of the participants were referred by a clinical officer. Twenty three percent were referred by a doctor- the cadre of which was not specified, 24 (28.2%) were referred by a nurse, 1 (1.2%) was refereed by a paediatrician, and 6 (7.1%) were referred by a physician (Figure 3).

Forty three participants (41.0%) were on medication at the time of contact in this study. 33 of these (76.7%), were on antihistamines, 53.5% on antibiotics, 11.6% on nasal drops and paracetamol, 7.0% were on other cough syrups, and 4.7% were on herbal medication.

Table 1: Patient Characteristics in Children with Adenotonsillar Hypertrophy at Mtrh (N = 105)

CHARACTERSTIC	DETAIL
Sex: Male Female	59 (56.2%) 46 (43.8)
Age in years Under 2 years 2-5 years 5-10 years Above 10 years	Mean age:3.5 ±3 years; (0.3 - 7.0 years) 24 (22.9%) 58 (55.2%) 20 (19.0%) 3 (2.9%)
Nutritional status	Normal: 86 (81.9%) Underweight: 15 (14.3%) Overweight: 4 (3.8%)

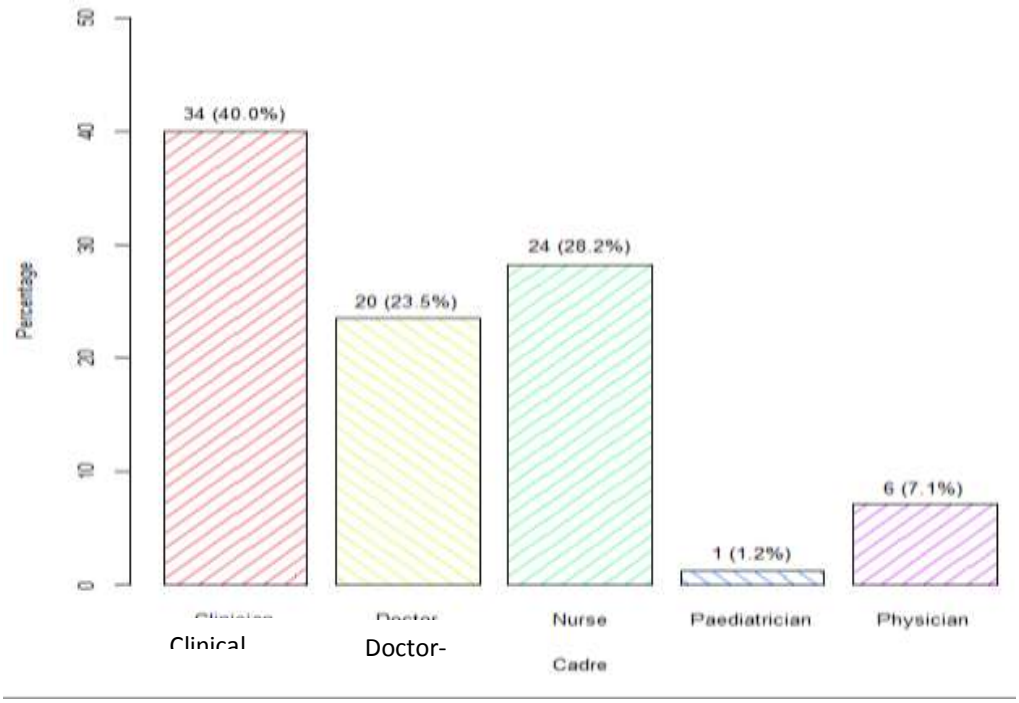

Figure 3:Cadre of referring clinician

4.2 Clinical Presentation

The most common presenting symptom was nasal congestion and obstruction in 95%, mouth breathing while asleep in 91.4%, recurrent sore throat in 76%, snoring in 61%, throat pain in 44.8%, restless sleep in 41.9%, fatigue in 36.2%, difficulty in swallowing in 31.4%, sneezing and allergic symptoms in 26.7%, weight loss in 15.2%, and fever and shortness of breath in 8.6% and 6.7% of the participants, respectively (Table 3).

Table 2: Reasons for referral /presenting symptoms at the hospital

Symptoms	**n (%) ;N=105**
Nasal congestion and obstruction	100(95.2%)
Mouth breathing while asleep	96(91.4%)
Recurrent sore throat	76(72.4%)
Snoring	64(61.0%)
Nasal discharge	62(59.0%)
Throat pain	47(44.8%)
Restless sleep	44(41.9%)
Fatigue	38(36.2%)
Difficulty in swallowing	33(31.4%)
Sneezing, allergic symptoms	28(26.7%)

Table 3: Neurocognitive disturbances in children of school-going age

Neurocognitive disturbances	***(n=63)***
Inattentiveness	50 (79.4%)
Difficulty playing with other children or engaging in leisure activities	43 (68.3%)
Irritability	35 (55.6%)
Short attention span	31 (49.2%)
Troubled concentration	23 (36.5%)
Daytime somnolence	18 (28.6%)
Hyperactivity	5 (7.9%)

Sixty three parents/guardians with children of school-going age responded to neurocognitive disturbances. Of these, 79.4% said that their children lacked attentiveness, 68.3% said that their children had difficulty in playing with other

children or in engaging in leisure activities. There were 55.6% who were reported to be irritable, while another 49.2% had short span of attention. Other neurocognitive disturbances reported include poor concentration (36.5%), daytime somnolence (28.6%), and hyperactivity (7.9%), anger (1.6%) and being fidgety (1.6%).

Table 4: Smoking and family history of clinical conditions

Variable		**Sample size**	**n (%)**
History of smoking	Never	105	105 (100.0%)
Exposed daily to smoke from other family members		105	2 (1.9%)
Family history			
	Asthma		28 (26.2%)
	Allergies		22 (20.6%)
	Diabetes	105	20 (18.7%)
	High Blood Pressure		11 (10.3%)
	Bleeding symptoms		1 (0.9%)
	Cancer		1 (0.9%)

Two of the guardians (1.9%) reported that the children were exposed to smoking from other family members on a daily basis.

Family history of asthma, allergies, and diabetes were reported by 26.2%, 20.6%, and 18.7% respectively. High blood pressure was reported by 10.3% of the participants. There was one who reported history of cancer in the family.

Table 5: Presenting symptoms- Risk of OSA

Variable	n (%)	N
Snore loudly	64 (61.0%)	105
Feeling tired/fatigued/sleepy during daytime	65 (61.9%)	105
Observed that the child at times stops breathing while asleep	81 (77.1%)	105
Child has high blood pressure	2 (1.9%)	105

61.0%, 61.9%, 77.1%, and 1.9% were reported to be snoring loudly, feeling tired/sleepy during day time, to stop breathing while asleep, and have high blood pressure respectively. Over half (56.2%) of the participants had a high risk for OSA, having scored more than 3 in the above parameters that are addressed in the STOP-BANG questionnaire.

4.3 Clinical Examination Findings

The clinical examination findings were documented on general examination of the patients, the cardiovascular system examination, the respiratory system examination, and the ENT examination.

4.3.1 General Examination Findings

The general examination findings were normal in 91 of the patients (86.7%). 12 patients (11.4%) had adenoid facies.

4.3.2 The Cardiovascular System Examination Findings

The cardiovascular system assessment involved assessment for tachycardia, oedema, hypoxia, high blood pressure, distended neck veins, palpable P2, hepatomegaly.

The most common finding was tachycardia in 16 (15.2%) patients. Hepatomegaly was found in 12 (11.4%) patients, and a murmur was found in 9 (8.6%) patients. Oedema and hypoxia were found in 4 (3.8%) patients, a palpable P2 in 6 (5.7%) patients, and distended neck veins in 2 (1.9%) patients and high blood pressure in 2 (1.9%) patients.

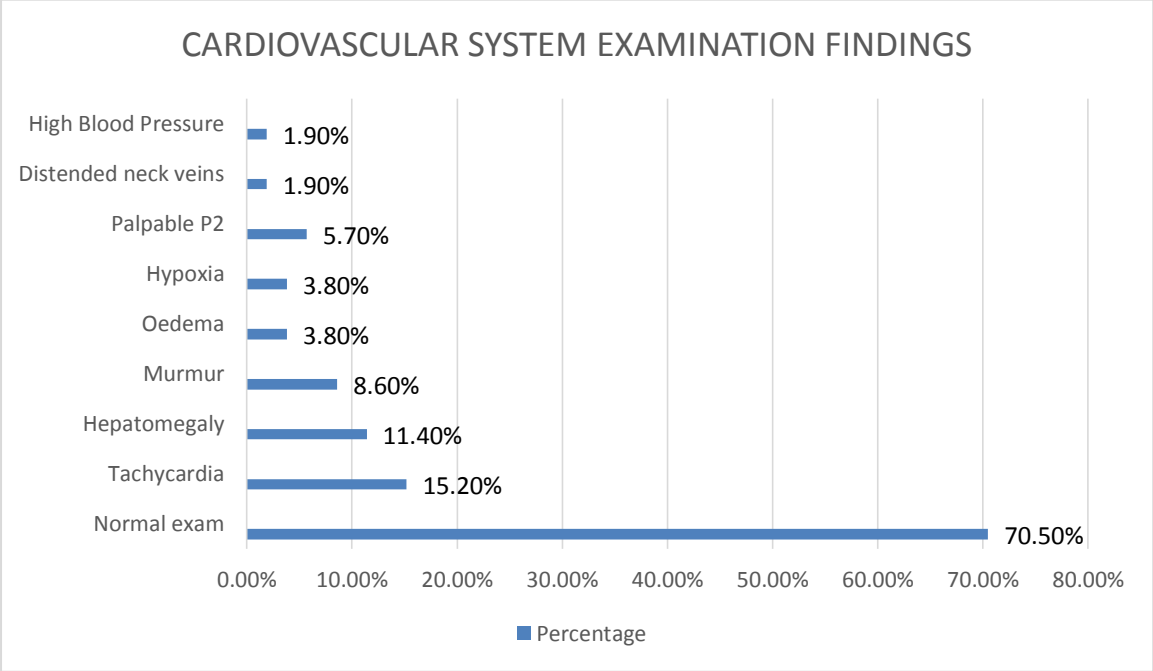

Figure 4: Cardiovascular System Examination Findings

4.3.3 The ENT Examination Findings

On ENT evaluation, mouth breathing was observed in 84 (80%) of the children. Tonsil size was graded using the Brodsky scale. Majority of the children 77 (74.3%) had tonsil size grade 2+. Grade 0-1 tonsils were examined in 11 patients (10.5%), grade 3+ tonsils in 13 (12.4%) and 4 children (3.8%) children had tonsils assessed to be grade 4.

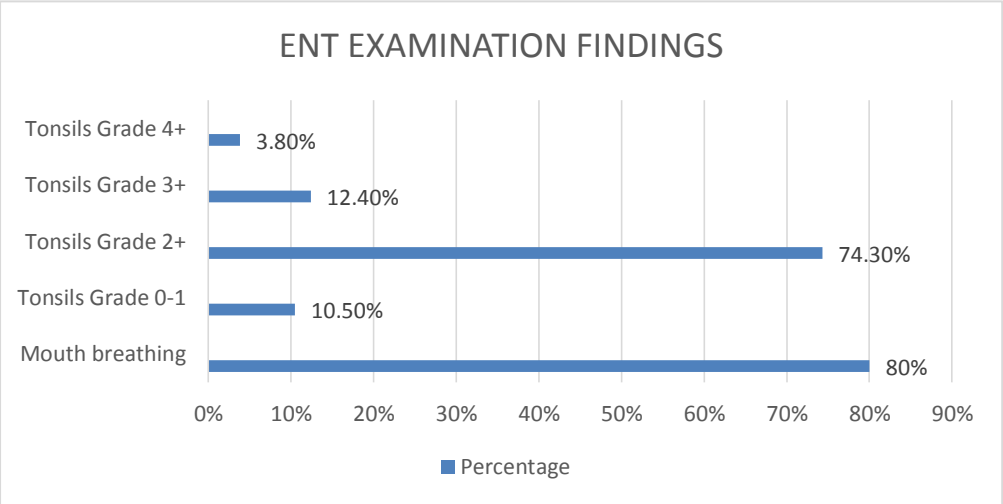

Figure 5: ENT Examination Findings

Table 6: Association between high risk of OSA and growth parameters

	High Risk of OSA No (Score < 3)	Yes (Score >= 3)	Fisher's Exact P
Length/Height-for-age percentile			
<5.0%	16 (34.8%)	29 (49.2%)	
5.0 – 95.0%	29 (63.0%)	29 (49.2%)	0.312
>95.0%	1 (2.2%)	1 (1.7%)	
Weight-for-age percentile			
<5.0%	4 (8.7%)	11 (18.6%)	
5.0 – 95.0%	41 (89.1%)	45 (76.3%)	0.262
>95.0%	1 (2.2%)	3 (5.1%)	
BMI-for-age percentile			
<5.0%	4 (9.1%)	7 (11.9%)	
5.0 – 85.0%	30 (68.2%)	34 (57.6%)	0.707
85.0 - 95.0%	6 (13.6%)	9 (15.3%)	
>95.0%	4 (9.1%)	9 (15.3%)	

There was no sufficient evidence from the data to link high risk of OSA to poor growth among the participants.

4.3.4 Assessment For Pulmonary Hypertension

A total of 26 out of 105 children, which is 24.8% (95% Cl 12.9-28.2), had pulmonary hypertension. Of these, 19 (73%) had mild pulmonary hypertension, 6 (23%) had moderate pulmonary hypertension, and 1 (4%) had severe pulmonary hypertension. The mean mPAP of the study population was 24.3 ±3.9 mm Hg.

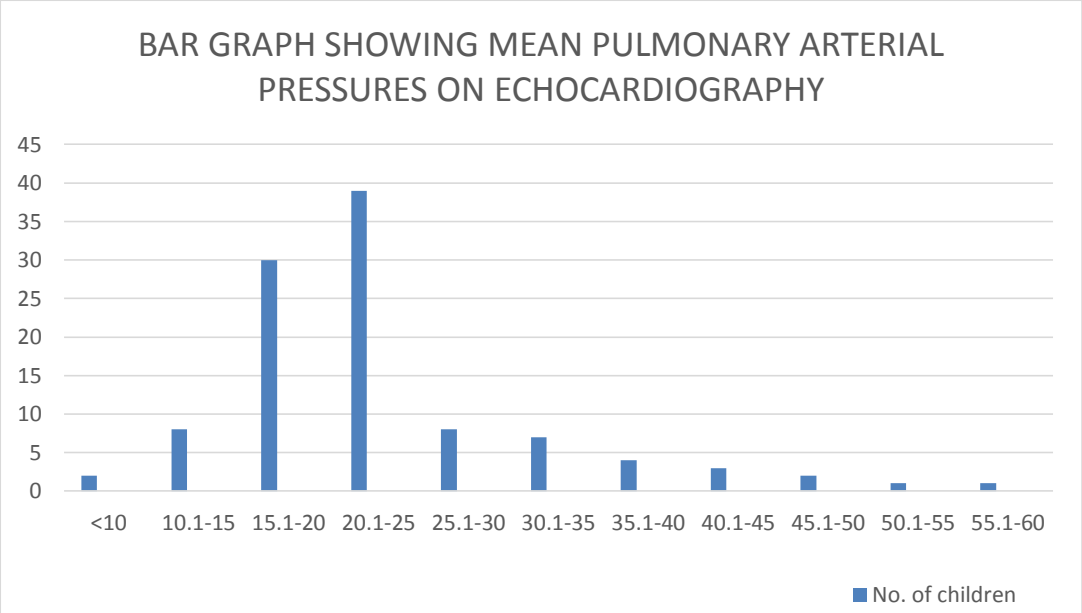

Figure 6: Mean pulmonary artery pressures on echocardiography

Table 7: Association between pulmonary hypertension and growth parameters

	Pulmonary Hypertension		
	No PHTN <= 25 mm Hg	Yes PHTN 25 mm Hg	Fisher's Exact P
Length/Height-for-age percentile			
<5.0%	29 (36.7%)	10 (38.5%)	
5.0 – 95.0%	48 (60.8%)	15 (57.7%)	0.692
>95.0%	2 (2.5%)	1 (3.9%)	
Weight-for-age percentile			
<5.0%	9 (11.4%)	4 (15.4%)	
5.0 – 95.0%	65 (82.3%)	22(84.6%)	0.629
>95.0%	5 (6.3%)	0 (0.0%)	
BMI-for-age percentile			
<5.0%	10 (12.7%)	4 (15.4%)	
5.0 – 85.0%	41 (51.9%)	13(50.0%)	0.695
85.0 - 95.0%	17 (21.5%)	3 (11.5%)	
>95.0%	11 (13.9%)	6 (23.1%)	

The results do not suggest any difference in the growth rates between those who had pulmonary hypertension compared to those who did not have. The height-for-age, weight-for-age, and BMI-for-age parameters did not differ significantly between the two groups of participants.

Table 8: Association between duration of symptoms and pulmonary hypertension

	Pulmonary Hypertension		
	No PHTN (n = 79)	PHTN (n = 26)	**Fisher's Exact P**
Duration of symptoms, (mean duration-months)	3.0 ±1.9	4.6 ±2.3	**0.037**

The mean duration of symptoms among those who were diagnosed of pulmonary hypertension was longer, 4.6 ±2.3 months, compared to among those who did not have pulmonary hypertension, 3.0 ±1.9 months. The test for difference between the two groups showed statistical significance, with a p value of 0.037

Table 9: Association between patient characteristics and pulmonary hypertension

Patient Characteristics	**Pulmonary hypertension N=26 (%)**	**Normal pulmonary pressure N= 79 (%)**	**OR (95% Cl)**	**P Value**
Sex: Male	17(65.4%)	42(53.1%)	2.23(0.81-7.22)	0.131
Medication Use: On medication	14(53.8%)	29(36.8%)	1.51(0.65-8.12)	**0.045**
Referral: Referrals	19(73.1%)	67(84.8%)	2.11(0.48-8.89)	0.412
Inpatient:	18(69.2%)	5(6.3%)	2.4(1.67-4.66)	**0.038**

Bivariate analysis of patient characteristics associated with pulmonary hypertension showed that the odds of pulmonary hypertension in male children was 2.23 times greater as compared to female children, OR=2.23(0.81-7.22), p=0.131. Children who were on medication had a 1.5 fold increased risk of having pulmonary hypertension compared to those who were not on medication, OR=1.51(0.65-8.12), P=0.045. Children who were sent to the clinic or to the ward as referrals had a 2.11 fold

increased risk of pulmonary hypertension in comparison to those who were not referrals. However, this was not statistically significant. Children who were inpatients with adenotonsillar hypertrophy were also at a 2.4 fold risk of having pulmonary hypertension, as compared to children who were seen as outpatient, OR=2.4(1.67-4.66), p=0.038.

Table 10: Association between patients' presenting symptoms and pulmonary hypertension

Symptom	**Pulmonary HPTN N=26(%)**	**No Pulmonary HPTN N=79(%)**	OR (95% Cl)	p value
Mouth breathing while asleep	20(76.9%)	76(96.2%)	3.11(1.43-8.97)	**0.024**
Recurrent sore throat	13(50%)	63(79.8%)	0.54(0.23-3.42)	0.543
Snoring	9(34.6%)	55(69.6%)	0.49(0.38-1.03)	0.118
Nasal discharge	6(23.1%)	56(70.1%)	0.61(0.34-6.55)	0.309
Throat pain	12(46.2%)	23(29.1%)	0.74(0.21-4.33)	0.285

Children with adenotonsillar hypertrophy who presented with mouth breathing while asleep had a three-fold increase of having pulmonary hypertension, OR=3.11(1.43-8.97), p=0.024. This was statistically significant.

Table 11: Association between physical examination findings and pulmonary hypertension

GENERAL EXAMINATION	PHTN N=26(%)	NO PHTN N=79(%)	OR (95% CL)	p value
Pallor	1(3.8%)	1(1.3%)	0.43(0.12-1.61)	0.621
Adenoid facies	8(30.8%)	4(5.1%)	2.45(0.57-10.12)	**0.029**
CARDIOVASCULAR SYSTEM EXAMINATION				
Tachycardia	9(34.6%)	7(8.9%)	0.74(0.26-2.76)	0.815
Hepatomegaly	8(30.8%)	4(5.1%)	1.43(0.76-2.96)	0.203
Murmur	7(26.9%)	2(2.5%)	0.98(0.34-1.19)	0.912
Oedema	3(11.5%)	1(1.3%)	1.89(0.60-1.93)	0.114
Hypoxia	3(11.5%)	1(1.3%)	4.11(2.56-8.91)	**0.037**
Palpable P2	2(7.7%)	4(5.1)	1.01(0.74-3.54)	0.119
Distended neck veins	2(7.7%)	0(0%)	1.95(0.14-7.95)	0.441
High Blood Pressure	1(3.8%)	1(1.3%)	0.98(0.97-1.90)	0.774
ENT EXAMINATION FINDINGS				
Tonsils Grade 1	1(3.9%)	10(12.6%)	0.57(0.22-5.35)	0.681
Tonsils Grace 2	9(34.6%)	68(86%)	1.99(0.36-3.41)	0.237
Tonsils Grade 3	9(34.6%)	4(5.1%)	0.58(0.36-2.98)	0.448
Tonsils Grade 4	2(7.7%)	2(2.53%)	2.55(0.47-3.22)	**0.043**

The physical examination findings that were associated with pulmonary hypertension in children with adenotonsillar hypertrophy included adenoid facies, on general examination OR=2.45 (0.57-10.12), p=0.029. This was statistically significant. Children with adenotonsillar hypertrophy who had adenoid facies had a 2.45-fold higher chance of having pulmonary hypertension than those who did not have adenoid facies. On the cardiovascular system examination, pulmonary hypertension was associated with finding of hypoxia, OR=4.11(2.56-8.91), p=0.037. Children with adenotonsillar hypertrophy who had hypoxia on physical examination had a 4 fold increase in the chance of having pulmonary hypertension. Those with tonsils Grade 4 had a 2 fold increase in the chance of having pulmonary hypertension.

CHAPTER FIVE
DISCUSSION

5.1 Patient Characteristics

Most of the children with adenoid hypertrophy were between 3 and 7 years. The mean age of presentation of the children in this study was 3.5±3 years. The age group of the participants in Chinawa's study ranged from birth to 70 months. The mean age of presentation in his study was 32.6±17.9 months. The mean age in years in Marangu's study, for those who had pulmonary hypertension was 2.3 years and 2.91 years for those without pulmonary hypertension. The adenoids are part of the lymphoid tissue in the body, and they proliferate and grow continuously within the first few years of life. By late childhood, which is about 10 to 13 years, the lymphoid tissue begins to atrophy. Adenotonsillar hypertrophy is thus most common in the earlier years of life, due to lymphoid tissue proliferation, and the cases then reduce towards the teen years, as the lymphoid tissue begins to atrophy.

Majority of the patients seen in this study had been sent as referrals, either by a clinical officer, a nurse, a paediatrician, a physician, or other healthcare worker of another cadre. This is evidence that adenotonsillar hypertrophy is a common reason for paediatric referrals to otolaryngologists, hence each cadre of health care worker needs to have a high index of suspicion when a child presents to them, so that further treatment and consultation is sought early enough. The highest referrals were from non-specialized cadres of health care givers, including clinical officers and nurses; fewer referrals came from paediatricians. This could be because the clinical officers and nurses first encounter these patients right at the grass root level facilities, or at the outpatient departments and send them for referral for specialized consultation and treatment as soon as they record that the patient is complaining of ENT symptoms.

The reason for the few referrals from paediatricians could be that these patients do not go to the consultant clinics or specialist clinics, right from the onset. This could be due to the high cost of private consultation. None of the studies done by either Marangu, Eziyi or Chinawa looked at the number of patients who had come to the ENT departments by way of referrals.

About half of the patients in my study were also on medication, which ranged from antibiotics, to antihistamines, antipyretics and analgesics, nasal drops and even herbal medication. This study demonstrated that children who were on medication for adenotonsillar hypertrophy were at an increased risk of developing pulmonary hypertension, OR. 1.51(0.65-8.12), p=0.045. This is because these children are put on drugs that simply provide symptomatic relief, but the primary cause of the upper airway obstruction still remains. This is different from what was demonstrated by Marangu D. in her study. Ninety two children out of the 123 (74.8%) that she studied were on intranasal steroids, sixty seven (72.8%) of whom were using intranasal steroids intermittently as opposed to continuously. This difference can be explained from the fact that she zeroed in on the use of intranasal steroids. An intranasal steroid will most likely have been prescribed by a clinician, as opposed to the classes of drugs that the children in my study were on, which are largely available as over the counter drugs, and some mothers actually confirmed self- presciptions and purchase of these drugs. Steroids act as anti-inflammatory drugs and work to reduce the process of inflammation that occurs within the lymphoid tissue with hyperplasia, and further worsened in case of upper respiratory tract infections or exposure to allergens. Steroids can thus reduce the obstructive symptoms and the sleep disordered breathing that occurs especially at night.

5.2 Clinical Characteristics

The most common presenting symptom among the children were nasal congestion and obstruction, mouth breathing while asleep, recurrent sore throat, snoring, throat pain, restless sleep, fatigue, difficulty in swallowing, sneezing and allergic symptoms, weight loss, fever, and shortness of breath. In Chinawa's study, he found that cough 19(73.1%), catarrh 18(69.2%), history of allergy 15(57.7%), fever 13(50.0%), Snoring 10(38.4%), expiratory rhonchi 5(19.2%), and mouth breathing 4(15.4%) were the most common symptoms.

In this study, we defined the duration of time for which the children have had these symptoms and did not characterize them in terms of frequency. Marangu's study was able to enlist these symptoms as either day or night symptoms, and to look at the severity and frequency in terms of days or nights per week. Marangu's study found that the children had fairly advanced disease with 6 of 10 symptoms reported to occur every day. Similarly, Kara, in their study, showed that there was a statistically significant association between tonsillar hypertrophy and history of frequently having tonsillitis, habitual snoring, observed apnea, oral breathing during sleep and difficulty eating.

None of the patients had chest pain during physical activity, chronic fatigue, dizziness or fainting. These findings were similar to my study, as snoring, open mouth breathing and agitated sleep were the common symptoms that were registered by the participants in my study. None of the parents or guardians of the patients in this study complained of chest pain during physical activity, dizziness or fainting. Jabbari et al also evaluated 47 children aged between 3 and 11 years, with adenotonsillar hypertrophy. In their study, all children (100%) had night snoring with open mouth

breathing and agitated sleep. Eleven (20%) had a history of respiratory pause similar to hypopnea, but none of them suffered from apnea.

Children with adenotonsillar hypertrophy may present with other complaints, other than the usual upper airway obstruction complaints, or symptoms to do with breathing. We found that over half of the parents/guardians of children of school going age responded to the question of whether their child had an element of neurocognitive disturbances. This is similar to what Brockmann and Bertrand found in their study. This may be an indicator that children with adenotonsillar hypertrophy have troubled sleep because of the upper airway obstruction, and this may lead to neurocognitive disturbances. Brockmann and Bertrand were able to do an objective assessment of their study participants using the SDB questionnaire. My assessment of the sleep disorder was mainly subjective as reported by the parents and the guardians. They were also able to objectively assess hyperactivity and inattentive behaviour using the Conner's rating scale. The neurocognitive disturbances found by Marangu in her study were mainly hyperactivity and enuresis.

In this study, family history of asthma, allergies, and diabetes were reported. Similarly, Chinawa found features suggestive of atopy in majority of children with adenoids hypertrophy. These children presented with cough, snoring, expiratory wheeze, nasal blockage, expiratory rhonchi and prolonged expiratory phase on examination of the chest.

5.3 Physical Examination Findings

On the assessment of the anthropometric measurements, most of the children were of normal nutritional status. This was similar to Marangu's study, in which she found that majority of the participants with weight for height Z-scores > -2. Marangu's

study did not include children who were obese. Children with a body mass index (BMI) > 95th percentile for the age were excluded from her study. Children with chronic upper airway obstruction can present with failure to thrive due to the chronicity of the symptoms and the chronic hypoxic state, presenting with malnourishment due to the chronic illness. [24] Symptoms may be worse in obese children. Sogut A, Altin R. and Uzun demonstrated this by carrying out polysomnography on school going children in Turkey. These they had classified as: non-snorers, occasional snorers, and habitual snorers. They found the prevalence of habitual snoring to be 3.3%, and the minimum estimated prevalence of OSAS was found to be 1.3%. In this study, polysomnograhy was not carried out.

Adenoid facies were seen on general examination of some of the patients. These are characteristic features on the face of children who have had chronic upper airway obstruction. They come about as a result of these children becoming mouth-breathers, in a bid to breathe in more oxygen, and compensated for hypoxia. Marangu, in her study, did not categorically assess for adenoid facies on general examination of the participants in her study. Generally, studies have documented and it has been demonstrated, according to Jones and Phillips, and according to Urschitz et. al., adenotonsillar hypertrophy leads to these dentofacial growth anomalies.

In this study, the findings on examination of the cardiovascular system included tachycardia, hepatomegaly, an auscultated murmur, oedema, hypoxia, a palpable P2, distended neck veins and high blood pressure. Of these, hypoxia had a positive association with pulmonary hypertension. As the right ventricle becomes hypertrophic, elevated right-sided pressures can produce a prominent a wave (corresponding to atrial contraction) in the jugular venous pulse, as well as a right-sided fourth heart sound and either a left parasternal heave or a downward subxiphoid

thrust. When the right ventricle becomes dilated and fails, the resultant systemic venous hypertension can produce an elevated jugular venous pressure with a prominent v wave, a right ventricular third heart sound, peripheral edema and, rarely, ascites. According to Han and McLaughlin, the liver can become enlarged and pulsatile, reflecting tricuspid insufficiency. Night-time hypoxia has a stronger correlation with obstructive sleep apnea syndrome, compared to the daytime oxygen saturations, according to Brouillette and Morielli. We did not measure the night time oxygen saturations, as this would require a sleep laboratory with appropriate monitors attached to each child, which the hospital does not have. Night time oxygen saturations should be measured for this correlation to be objectively drawn. In Marangu's study, the nighttime saturations were also not measured. In her study, both bivariate and multivariate analysis of daytime oxygen saturations were significantly lower in the children with pulmonary hypertension compared to those with without.

On ENT evaluation, a large percentage of the children had mouth breathing. Majority of the children also had tonsil grade 2+, according to the Brodsky scale.Routine ear, nose and throat (ENT) examination, lateral neck radiography and direct visualization of adenoid tissue by nasal endoscopy can be used to evaluate adenoid and tonsil size. This is according to King and Sheehan. These are all diagnostic modalities that are accessible and affordable in our setting. We evaluated the patients based on clinical evaluation and grading of the tonsils. Mouth breathing was positively associated with the presence of pulmonary hypertension. Marangu demonstrated that daily mouth breathing singly or in combination with restless sleep showed the highest sensitivity (88.5%) and negative predictive value (86.4%). In my study, <50% of the children had tonsils size grade > 3 on the Brodsky scale.

5.4 Pulmonary Hypertension and Associated Factors

One quarter of participants in my study had pulmonary hypertension. In Marangu D. et al's study, 27 patients had pulmonary hypertension giving a prevalence of 21.9%. In terms of the patients' characteristics, age was not significantly associated with pulmonary hypertension. Males with adenotonsillar hypertrophy had a 2-fold increased chance of having pulmonary hypertension than the females with adenotonsillar hypertrophy. This is comparable to other studies by Moghaddam et al, Abdel-Aziz et al, Yilmaz et al, Granzotto et al and Tatlipinar et al.

Children who were on medication were more likely to have pulmonary hypertension as demonstrated in this study. This was different from Marangu, et al's finding, who found that most of the children in her study were using intranasal steroids. Children who did not use intranasal steroids had a 3-fold increased odds of having pulmonary hypertension, implying that intranasal steroids confer protection. For our study, the evidence of medication use included use of antibiotics, antihistamines, analgesics and anti-inflammatory drugs, and not intranasal steroids. This may explain the differing findings. For our study population, medication was to be taken for symptomatic relief, but the process and the pathophysiology leading up to the pulmonary hypertension is still on-going .Children with adetonsillar hypertrophy that required admission as inpatients into the padiatric ward were also found to have an over two-fold increased chance of having pulmonary hypertension than those who were not inpatient. This is because at the point of admission, the children must be very sick, with very severe signs and symptoms. No further studies were found from which to further comparison on the medications used children with the adenotonsillar hypertrophy, and the effect of whether a patient is inpatient or not.

The patients' presenting symptoms associated with pulmonary hypertension were mouth breathing The physical examination findings associated with pulmonary hypertension on general examination included the adenoid facies, and on cardiovascular examination were hypoxia. There was a positive association between the duration of symptoms and pulmonary hypertension. There was also a positive association between Grade 4 tonsils on the Brodsky scale and pulmonary hypertension. The children who had pulmonary hypertension had had symptoms for a longer time period of time. Obstruction at any site along the upper airway causes pulmonary hemodynamic disturbances. Repeated episodes of obstruction result in increased pulmonary vascular resistance as a result of hypoxic vasoconstriction.

In this study, one echocardiograph reading was obtained, pre-operatively. Viviane and José da Silva Moreira et al, evaluated the pre-operative and post-operative echocardiographs in 33 paediatric patients with adenotonsillar hypertrophy. Pulmonary hypertension was verified in 12 (36%) of the 33 patients with adenotonsillar hypertrophy. Adenotonsillectomy was associated with a significant 27% decrease in pulmonary pressures. Jabbari Moghaddam, in their study, evaluated pulmonary pressures in 47 patients with adenotonsillar hypertrophy. They found pulmonary pressures that were higher than 25mmHg in 12.7% of the patients. They followed up patients preoperatively and post-operatively, with the first month and sixth month post-operative echocardiography.

The prevalence of pulmonary hypertension in our study is close to three and a half times higher than this. This could be due to different population characteristics, different pattern of disease in our population, or varying degrees of the severity of symptoms and range of findings on physical evaluation of the patients. In Brazil, Granzotto, Aquino and Lubianca et al carried out a study to evaluate children who

were scheduled for adenotonsillectomy. The indication for the adenotonsillectomy was due to sleep disordered breathing. A point prevalence of 13% was reported. [37,60] The Chemla formula used is similar to our study. In our study, the prevalence of pulmonary hypertension is almost twice as high.

This study seems to have different prevalences for pulmonary hypertension compared to the studies done in the Middle East and in South America. This could be attributed to the difference in geographical location and environmental exposures, and difference in patient dynamics.

5.5 Study Limitation

Nocturnal, laboratory-based polysomnography (PSG) is the most commonly used test in the diagnosis of obstructive sleep apnea syndrome, which is a possible complication as a result of upper airway obstruction. Polysomnography can directly monitor and quantify the number of respiratory events and the resultant hypoxemia and arousals related to these respiratory events.

Our limitation in this study was the lack of availability of equipment and laboratory for polysomnography. This would have been an important test for these patients with upper airway obstruction due to adenotonsillar hypertrophy.

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

1. The most common presenting complaints in children with adenotonsillar hypertrophy are nasal congestion and obstruction, mouth breathing while asleep, recurrent sore throat, snoring, nasal discharge.
2. About a quarter of the children with adenotonsillar hypertrophy have pulmonary hypertension.

6.2 Recommendations

Early recognition of adenotonsillar hypertrophy is important, as these children need to be treated promptly, before long term effects of the illness, including pulmonary hypertension set in.

Echocardiography should form part of the routine investigations for children with adenotonsillar hypertrophy.

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APPENDICES

Appendix 1: Consent Form (English)

CONSENT TO PARTICIPATE IN A RESEARCH STUDY

Serial Number:……

BACKGROUND

I, Dr. Grace Nyambura, a registrar in Child Health and Pediatrics, am carrying out a study to describe the clinical profile of children with adenotonsillar hypertrophy. I will take a full history from you as the parent or guardian of the child, do a full physical examination and then perform an Echocardiograph on your child. An echocardiograph is anultrasound device that is used to examine the working heart and display moving images of its action. It is a safe and non-invasive investigation. Our study is for research purposes.

There are no risks involved in this study. Your child will receive standard treatment as per the diagnosis and his or her clinical presentation. Your participation in this study is purely on a voluntary basis.

There are no direct medical benefits to your child for participating in this study. A potential benefit of the study will be improved healthcare service delivery based on the recommendations of this study.

All information in this study will be treated with utmost confidentiality and will not be divulged to any unauthorized persons. As a study participant, you will be identified by a serial number, and not by name.

If you have questions, complaints or concerns about this study, you can contact the primary investigator on this contact: Dr. Grace Nyambura, Telephone Number: 0722-372008.

There is no cost to you, and there is no compensation to subjects for participation in this study.

I truly appreciate your support and your co-operation.

CONSENT

By signing this consent form, I confirm that the researcher has clearly explained to me what will be involved in this study and I have clearly understood.

I hereby do voluntarily agree to take part in this study, and append my signature/fingerprint.

Caregiver's Name:…….……. Signature/Mark…….. Date……..

Investigator's Name:…… Signature/Mark……. Date……..

Appendix 2: Assent for Echocardiography for Children above 7 Years

Dr. Grace Nyambura has explained to me that she is a doctor in training, a registrar in Child Health and Pediatrics. She has explained to me that she is carrying out a study to describe the clinical profile of children with adenotonsillar hypertrophy. She has explained to me that part of the study to perform an Echocardiograph on me. An echocardiograph is a scan to see how my heart is functioning. She has assured me that it is a safe test, there is no pain involved, and no pricking involved.

By writing my name/making a mark on this form, I confirm that I have clearly understood and I agree to have the echocardiograph done.

Child's Name:……...…….…… Signature/Mark…….. Date……..

Caregiver's Name:…….…….…… Signature/Mark…….. Date……..

Investigator's Name:…… Signature/Mark……. Date……..

Appendix 3: Consent Form (Swahili)
FOMU IDHINISHO

IDHINI YA KUSHIRIKI KATIKA UTAFITI

NambariyaFomu:……

UTANGULIZI

Mimi, Dr. Grace Nyamburanimwanafunzikatikachuokikuu cha Moi University .Ninafanyautafitinikiangaziawatotoambaowanashidayamafindo (*yaani tonsils kwakimombo)*kuwakubwakulikoinavyofaanakuzibanjiayahewakwenyemapua*(yaaniad enotonsillar hypertrophy kwakimombo).*

Piatutaangaliakamahuuugonjwaunafanyawatotohawakupunguzakimo au kuwanashidayapreshakuwajuukwenyemapafu.

Hiitutafanyakwakutimiapichamaalumyarohoinayojulikanakama*Echocardiograph* kwakimombo.

Hakunamadharayoyoteyatakayohusishwakatikautafitihuu.Mtotoatapatamatibabuyaug onjwakulingananamatokeo au sera zinazokubaliwanahospitalihiipamojanataratibuzawizarayaAfya.

Hakutakuweponafaidayamojakwamojakwakuhusikakatikautafitihuu.

Habarizotezitakazopatikanazitahifadhiwakisirinahabarikuhusuafyayamwanaohaitatum iwabilaidhini. Hakunamajina au habarizakukutambulishazitakazotolewa.

Iwapounaswalilolote, malalamishi au jambolisilokuridhishakuhusiananautafitihuu, mjulishemtafitimkuukwenyeanwaniifuatayo: Daktari Grace Nyambura, nambariyasimu 0722372008.Hakuna malipoyoyotekwakonaridhaakwayeyoteanayehusikakatikautafitihuu.

Asante sanakwakuruhusumwanaokushirikikatikautafitihuu.

IDHINI:

Kwakutiasahihifomuhii, ninakubalikuhusikakatikautafitihuu.

Jina la mzazi:…….…….……Sahihi/Alama…….. Tarehe……..

Jina la shahidi:……Sahihi/Alama……. Tarehe.……...

Appendix 4: Data Collection Tool
SERIAL NUMBER: _____

Tick Accoringly:

- **Ward**
- **ENT Clinic**

Each of the following items is important in helping us find out about the illness that brought you to see us. It will be helpful in managing the symptoms that your child is having.

Please answer each question as completely and as accurately as you can.

If you are unsure about a question, you can ask for clarification.

1. In a few words, please describe why you brought the child to see the doctor today._____
_____
2. How long has your child had this problem?_____
3. Were you refereed here by another doctor?Yes_____ No_____. If yes, please specify the cadre_____
4. Has yaour child ever had any of the following, or have you ever been told that your child has any of the following? (Tick all that apply)

o High blood pressure o Heart attack o Abnormal heart rhythm o Stroke o Heart failure o Heart murmur o Seizures o Asthma o Symcope/fainting spells o Kidney disease	o Liver disease/jaundice o Cancer (type_____) o Hepatitis o Thyroid problems o Pneumonia o Anaemia o Tuberculosis (TB) o Arthritis o HIV	o Acid reflux o Latex allergy o Diabetes o Blood transfusion o Bleeding problems o Depression o Needing radiation treatment o Substance abuse o Deep vein thrombosis/ blood clots

a. Other conditions that the child has been/is being treated for?_____

b. How many admissions has the child had so far due to this problem?_____

5. Does your child have any allergies to medications? (Specify)_____
6. Is the child currently on any medication?(Specify)
   - No medications
   - Medications (Please include non-prescription medications, such as aspirin, herbal treatments and vitamins that the child is taking): _____ _____ _____
   - Reason for taking the medications:_____
   - Were the medications prescribed by a doctor or purchased as Over-The Counter Drugs?_____
7. Are the child's immunizations up to date? Yes_____ No_____ If not, why?_____ _
8. Do you as the guardian/parent of the child smoke?
   - No, I have never smoked
   - No, I quit smoking ___ years ago. At that time, I was smoking___ packs per day for ___ years
   - Yes, I smoke cigarettes or a pipe
   - Yes, I have smoked ____ packs of cigarettes per day for _____ years
9. Is the child exposed to smoke from other members of your family on a daily basis?
   - Yes
   - No
10. Please tick all the symptoms that you have noted in the child today, or which the child has complained to you about

GENERAL ○ Fatigue ○ Chills ○ Fever ○ Night sweats ○ Weight loss/gain	**EYES** ○ Change in vision ○ Double vision ○ Wearing glasses	**EARS** ○ Hearing loss ○ Ear pain ○ Ear drainage ○ Ringing ○ Dizziness	**NOSE** ○ Nasal discharge ○ Nasal congestion ○ Nasal bleeding ○ Sinus pain
THROAT ○ Change in voice ○ Lump in throat ○ Throat pain ○ Difficulty swallowing	**LUNGS** ○ Shortness of breath ○ Frequent cough ○ Wheezing ○ Bloody cough	**CARDIOVASULAR** ○ Chest pain ○ Irregular heart beat ○ Ankle swelling	**GASTIROINTESTINAL** ○ Heart burn ○ Nausea ○ Vomiting ○ Diarrhoea ○ Constipation ○ Vomiting blood ○ Abdominal pain
GANITOURINARY ○ Difficulty urinating ○ Blood in urine	**NEUROLOGIAL** ○ Depression ○ Memory loss ○ Weakness ○ Numbness ○ Tingling	**MUSCULO-SKELETAL** ○ Back pain ○ Joint pain ○ Arm/leg pain ○ Muscle weakness	**SKIN** ○ Skin cancer ○ Skin disease
ENDOCRINE ○ Increased appetite ○ Excessive thirst ○ Heat/cold intolerance	**ALLERGIES** • Sneezing • Itchy/watery eyes • Facial swelling • Hives		

PHYSICAL EXAMINATION TARGETED TOWARDS ADENOTONSILLAR HYPERTROPHY

Age:	**Gender: Male/ Female**	**Height: (cm)**	**Weight: (kgs)**
MUAC: (cm)	**Calculated BMI:** **Height/Length for age (percentile):** **Weight for Height/Length (percentile):** **Weight for Age (percentile):**		
Blood Pressure: (mm Hg)	**Systolic:**	**Diastolic:**	

A. What are the presenting symptoms that have been noted in the child?

- **Snoring**

Do the child snore loudly (louder than talking or loud enough to be heard through closed doors)? **Yes/No**

Is the child a non-snorer, habitual snorer or an occasional snorer?

- **Tired**

Is the child often feeling of looking *t*ired, fatigued, or sleepy during daytime? **Yes/ No**

- **Observed**

Have you observed that the child stops breathing when they are sleep? **Yes /No**

- **Blood Pressure**

Does the child have a high blood Pressure? **Yes/ No**

- **BMI**

Is the BMI more than 35 kg/m^2? **Yes/ No**

- **Gender**

Is the child male? **Yes/No**

B. As a parent/ guardian, which of the other symptoms listed below have been noted in the child? *(Tick as appropriate)*

Other discomforts of breathing	**Neurocognitive disturbances**	**Frequent upper airway infections**
— Mouth breathing while asleep — Restless sleep — Difficulty in eating — Failure to thrive — Other: (please specify)	i. Inattentiveness ii. Hyperactivity iii. Anger iv. Resentment v. Fidgety vi. Short attention span vii. Daytime somnolence viii. Irritability ix. Troubled concentration x. Difficulty playing with other children or engaging in leisure activities i. Other: (please specify)	i. Recurrent sore throats ii. Recurrent ear infections iii. Nasal obstruction iv. Tonsillar infections v. Nasal drip vi. Other: (please specify)

C. As a parent/ guardian of a school going child (above 3 years), which of the other symptoms listed below have been noted in the child? *(Tick as appropriate)*

Other discomforts of breathing	**Neurocognitive disturbances**	**Frequent upper airway infection**
o Mouth breathing while asleep o Restless sleep o Difficulty in eating o Failure to thrive o Other (please specify)	o Inattentiveness o Hyperactivity o Anger o Resentment o Fidgety o Short attention span o Daytime somnolence o Irritability o Troubled concentration o Difficulty playing with other children or engaging in leisure activities o Other (please specify)	o Recurrent sore throat o Recurrent ear infection o Nasal obstruction o Nasal discharge o Others (please specify)

PHYSICAL EXAMINATION FINDINGS:

ENT:

RESPIRATORY EXAMINATION:

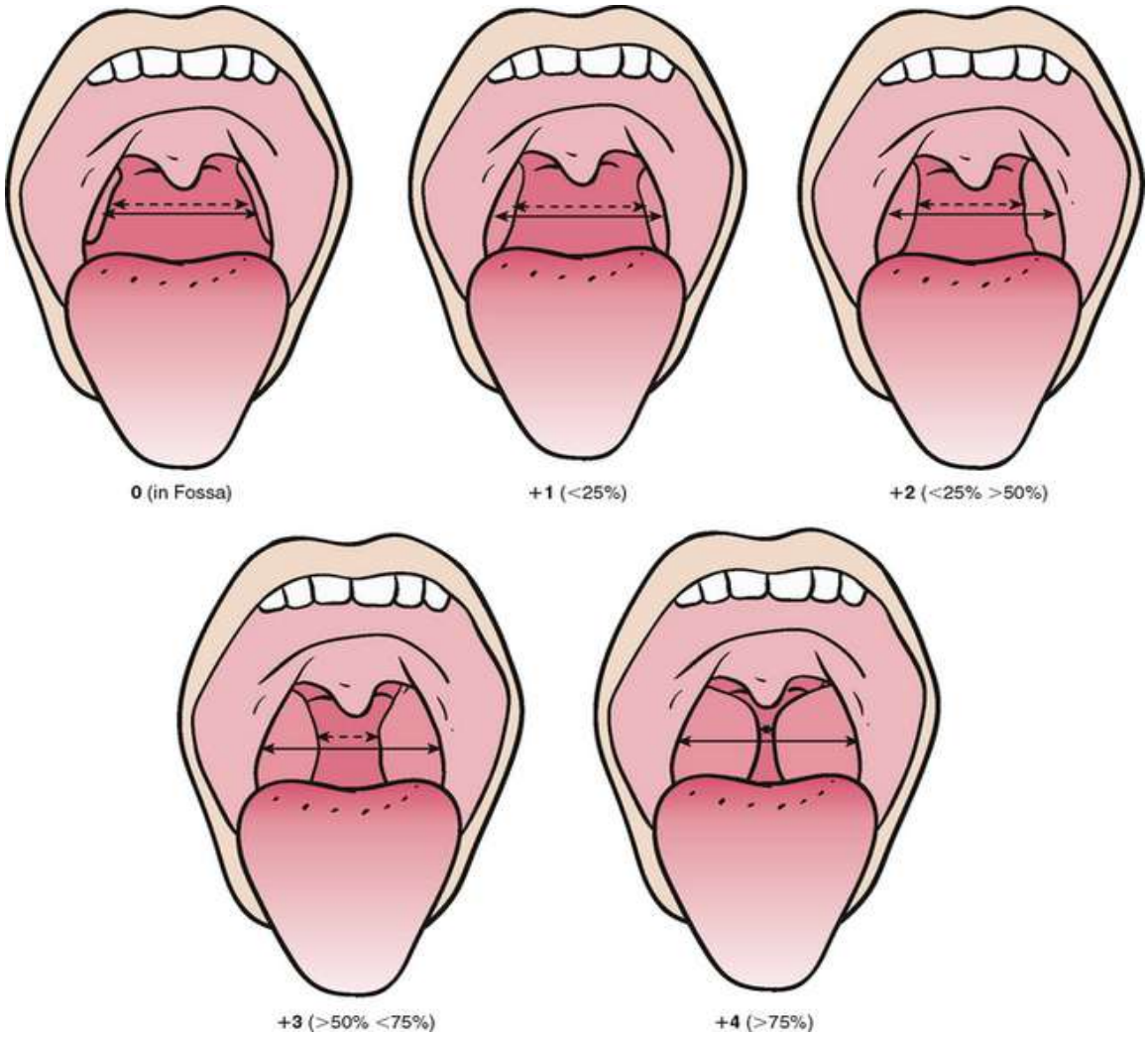
CARDIOVASCULAR EXAMINATION:

ADENOID FACIES:

ASSESSMENT FOR PULMONARY HYPERTENSION AND COR PULMONALE

Right ventricular systolic pressure (RVSP)	**Mean Pulmonary Arterial Pressure (mPAP)**	**Right Ventricular Function**

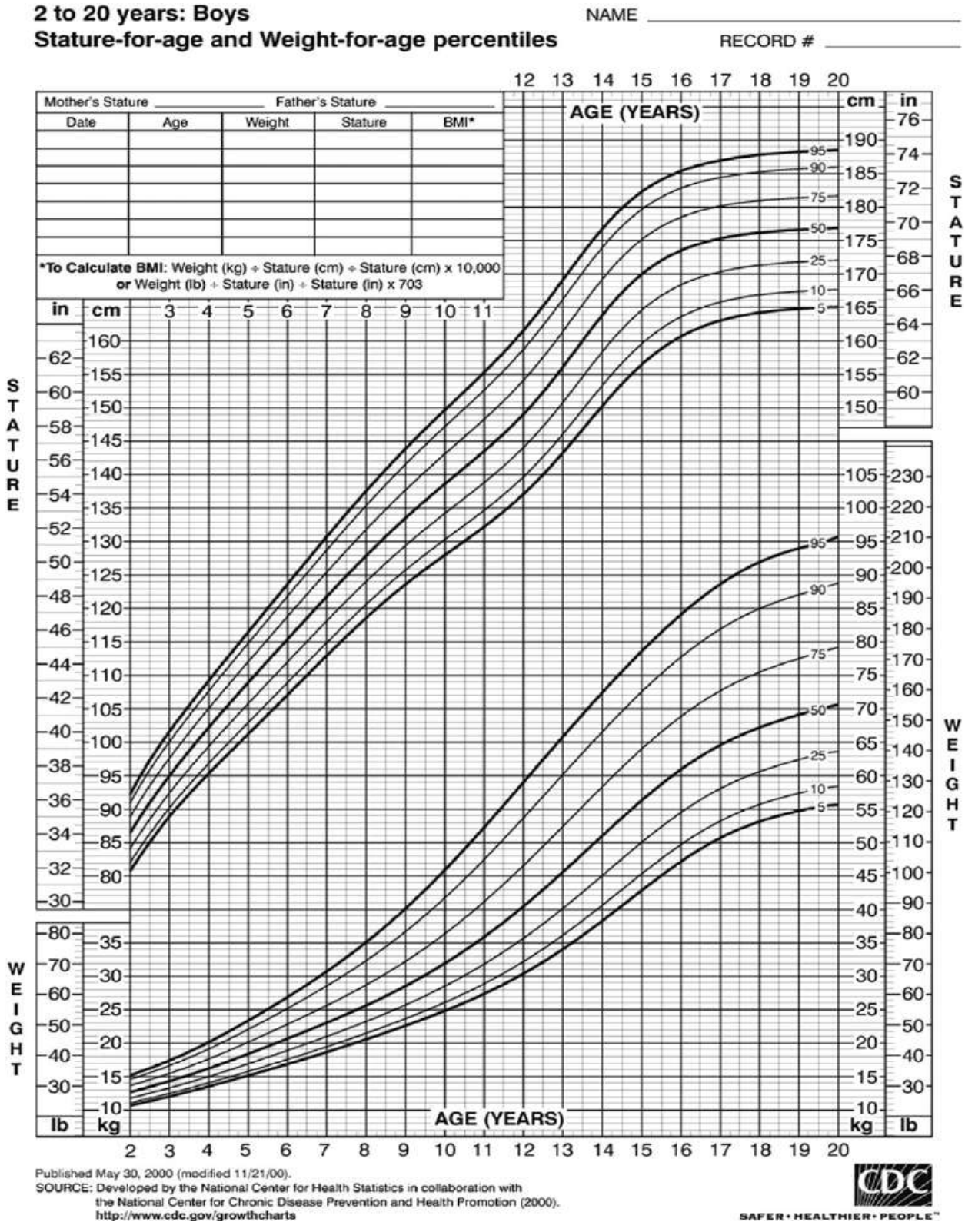
APPENDIX 5: BRODSKY CLASSIFICATION OF TONSILLAR ENLARGEMENT



DEGREE OF TONSILS BLOCKAGE	RATIO OF TONSILS IN THE OROPHARYNX
Degree 0	**Tonsils in the Fossa**
Degree 1	**Tonsil occupies less than 25% of the oropharynx**
Degree 2	**Tonsil occupies from 25-50% of the oropharyx**
Degree 3	**Tonsil occupies from 50% to 75% of the oropharynx**
Degree 4	**Tonsil occupies more than 75% of the oropharynx**

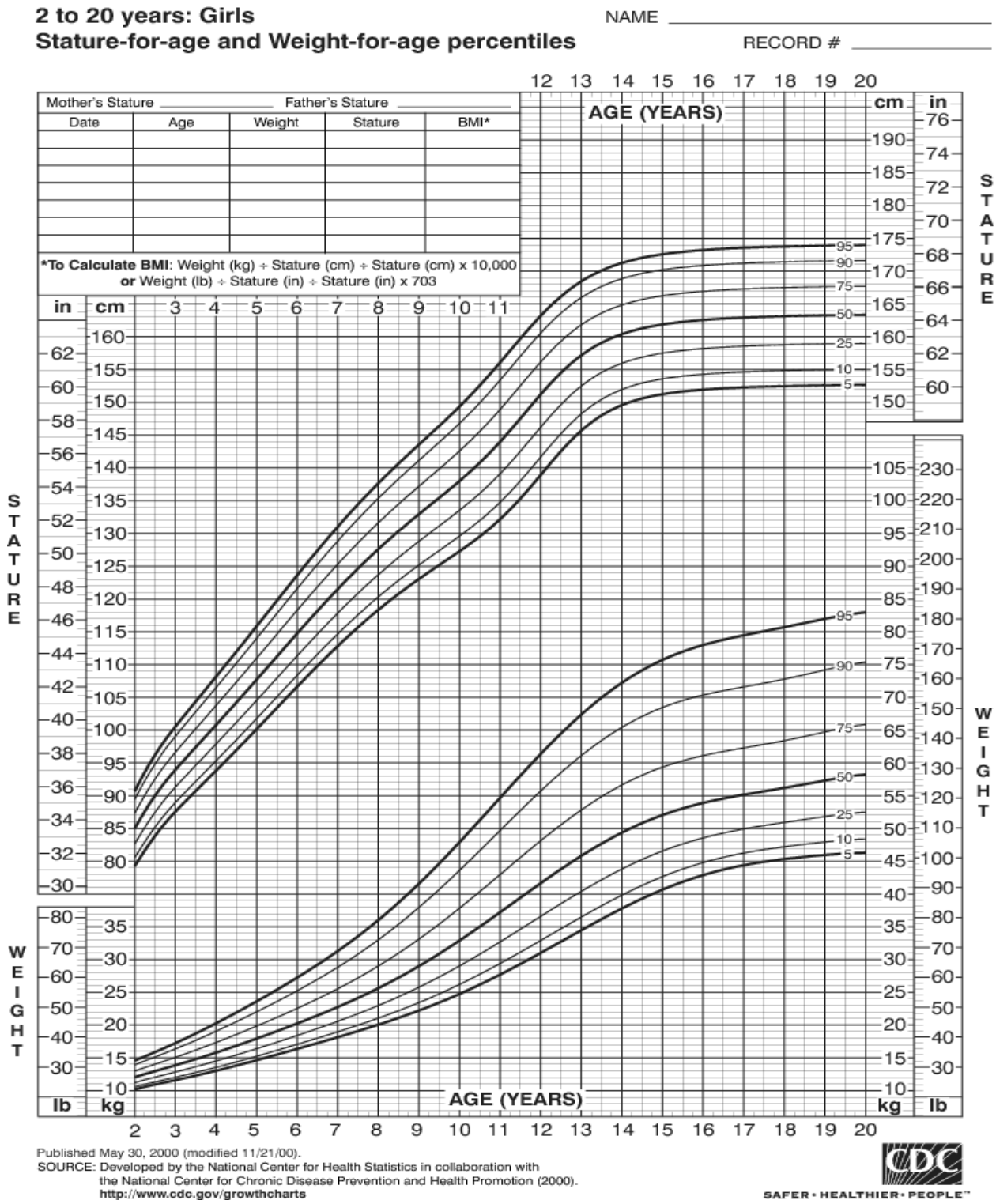
APPENDIX 6: WHO/CDC CHARTS- Stature for Age/Weight for Age

Percentiles: Boys

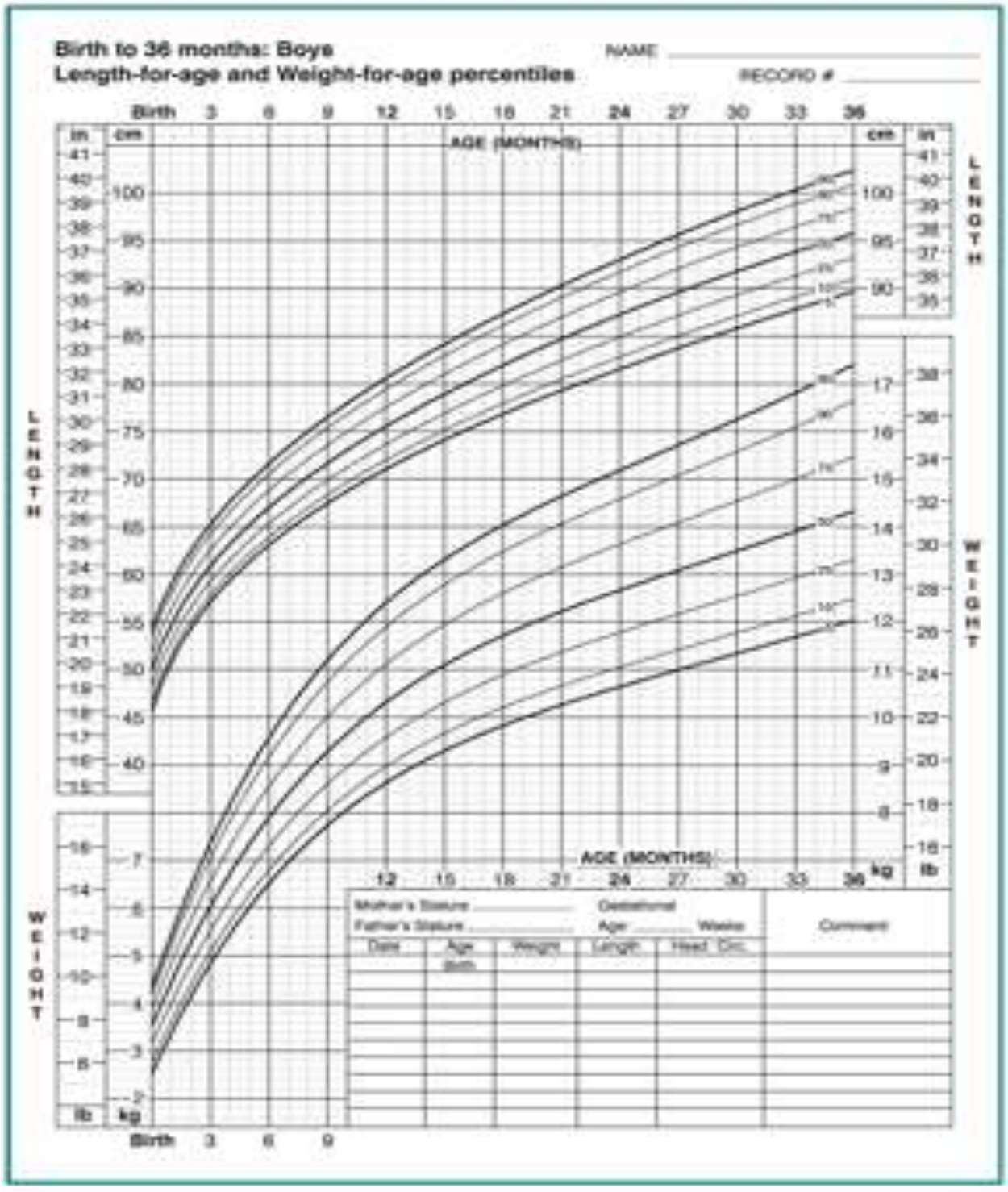


APPENDIX 7: WHO/CDC CHARTS- Stature for Age/Weight for Age

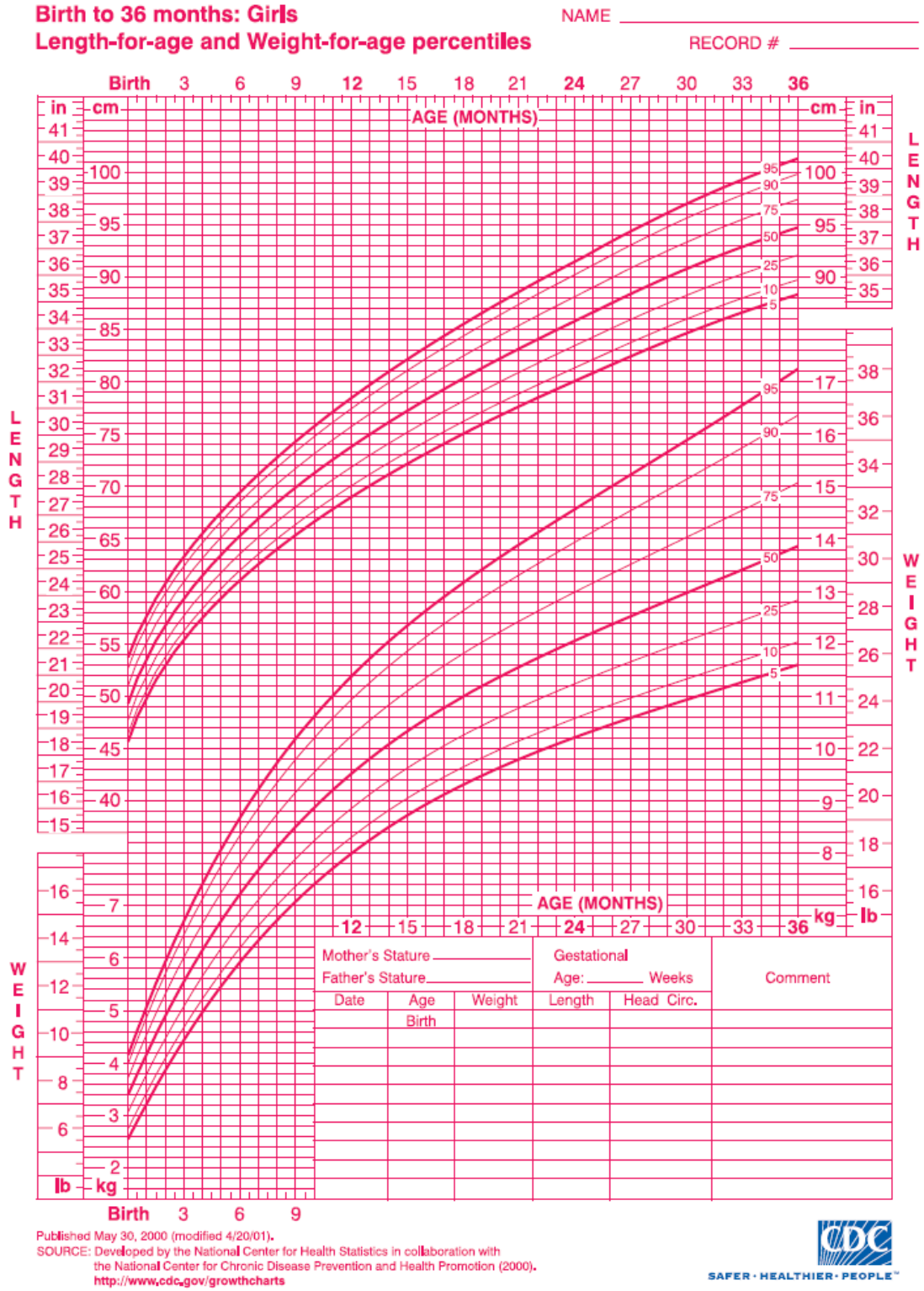
Percentiles: Girls



APPENDIX 8: LENGTH FOR AGE AND WEIGHT FOR AGE PERCENTILES:BOYS



APPENDIX 9: LENGTH FOR AGE AND WEIGHT FOR AGE PERCENTILES: GIRLS



APPENDIX 10: BLOOD PRESSURE LEVELS FOR BOYS

Blood Pressure Levels for Boys by Age and Height Percentile

Age (Year)	BP Percentile ↓	Systolic BP (mmHg) ← Percentile of Height →							Diastolic BP (mmHg) ← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
1	50th	80	81	83	85	87	88	89	34	35	36	37	38	39	39
	90th	94	95	97	99	100	102	103	49	50	51	52	53	53	54
	95th	98	99	101	103	104	106	106	54	54	55	56	57	58	58
	99th	105	106	108	110	112	113	114	61	62	63	64	65	66	66
2	50th	84	85	87	88	90	92	92	39	40	41	42	43	44	44
	90th	97	99	100	102	104	105	106	54	55	56	57	58	58	59
	95th	101	102	104	106	108	109	110	59	59	60	61	62	63	63
	99th	109	110	111	113	115	117	117	66	67	68	69	70	71	71
3	50th	86	87	89	91	93	94	95	44	44	45	46	47	48	48
	90th	100	101	103	105	107	108	109	59	59	60	61	62	63	63
	95th	104	105	107	109	110	112	113	63	63	64	65	66	67	67
	99th	111	112	114	116	118	119	120	71	71	72	73	74	75	75
4	50th	88	89	91	93	95	96	97	47	48	49	50	51	51	52
	90th	102	103	105	107	109	110	111	62	63	64	65	66	66	67
	95th	106	107	109	111	112	114	115	66	67	68	69	70	71	71
	99th	113	114	116	118	120	121	122	74	75	76	77	78	78	79
5	50th	90	91	93	95	96	98	98	50	51	52	53	54	55	55
	90th	104	105	106	108	110	111	112	65	66	67	68	69	69	70
	95th	108	109	110	112	114	115	116	69	70	71	72	73	74	74
	99th	115	116	118	120	121	123	123	77	78	79	80	81	81	82
6	50th	91	92	94	96	98	99	100	53	53	54	55	56	57	57
	90th	105	106	108	110	111	113	113	68	68	69	70	71	72	72
	95th	109	110	112	114	115	117	117	72	72	73	74	75	76	76
	99th	116	117	119	121	123	124	125	80	80	81	82	83	84	84
7	50th	92	94	95	97	99	100	101	55	55	56	57	58	59	59
	90th	106	107	109	111	113	114	115	70	70	71	72	73	74	74
	95th	110	111	113	115	117	118	119	74	74	75	76	77	78	78
	99th	117	118	120	122	124	125	126	82	82	83	84	85	86	86
8	50th	94	95	97	99	100	102	102	56	57	58	59	60	60	61
	90th	107	109	110	112	114	115	116	71	72	72	73	74	75	76
	95th	111	112	114	116	118	119	120	75	76	77	78	79	79	80
	99th	119	120	122	123	125	127	127	83	84	85	86	87	87	88
9	50th	95	96	98	100	102	103	104	57	58	59	60	61	61	62
	90th	109	110	112	114	115	117	118	72	73	74	75	76	76	77
	95th	113	114	116	118	119	121	121	76	77	78	79	80	81	81
	99th	120	121	123	125	127	128	129	84	85	86	87	88	88	89
10	50th	97	98	100	102	103	105	106	58	59	60	61	61	62	63
	90th	111	112	114	115	117	119	119	73	73	74	75	76	77	78
	95th	115	116	117	119	121	122	123	77	78	79	80	81	81	82
	99th	122	123	125	127	128	130	130	85	86	86	88	88	89	90

Blood Pressure Levels for Boys by Age and Height Percentile (Continued)

Age (Year)	BP Percentile ↓	Systolic BP (mmHg) ← Percentile of Height →							Diastolic BP (mmHg) ← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
11	50th	99	100	102	104	105	107	107	59	59	60	61	62	63	63
	90th	113	114	115	117	119	120	121	74	74	75	76	77	78	78
	95th	117	118	119	121	123	124	125	78	78	79	80	81	82	82
	99th	124	125	127	129	130	132	132	86	86	87	88	89	90	90
12	50th	101	102	104	106	108	109	110	59	60	61	62	63	63	64
	90th	115	116	118	120	121	123	123	74	75	75	76	77	78	79
	95th	119	120	122	123	125	127	127	78	79	80	81	82	82	83
	99th	126	127	129	131	133	134	135	86	87	88	89	90	90	91
13	50th	104	105	106	108	110	111	112	60	60	61	62	63	64	64
	90th	117	118	120	122	124	125	126	75	75	76	77	78	79	79
	95th	121	122	124	126	128	129	130	79	79	80	81	82	83	83
	99th	128	130	131	133	135	136	137	87	87	88	89	90	91	91
14	50th	106	107	109	111	113	114	115	60	61	62	63	64	65	65
	90th	120	121	123	125	126	128	128	75	76	77	78	79	79	80
	95th	124	125	127	128	130	132	132	80	80	81	82	83	84	84
	99th	131	132	134	136	138	139	140	87	88	89	90	91	92	92
15	50th	109	110	112	113	115	117	117	61	62	63	64	65	66	66
	90th	122	124	125	127	129	130	131	76	77	78	79	80	80	81
	95th	126	127	129	131	133	134	135	81	81	82	83	84	85	85
	99th	134	135	136	138	140	142	142	88	89	90	91	92	93	93
16	50th	111	112	114	116	118	119	120	63	63	64	65	66	67	67
	90th	125	126	128	130	131	133	134	78	78	79	80	81	82	82
	95th	129	130	132	134	135	137	137	82	83	83	84	85	86	87
	99th	136	137	139	141	143	144	145	90	90	91	92	93	94	94
17	50th	114	115	116	118	120	121	122	65	66	66	67	68	69	70
	90th	127	128	130	132	134	135	136	80	80	81	82	83	84	84
	95th	131	132	134	136	138	139	140	84	85	86	87	87	88	89
	99th	139	140	141	143	145	146	147	92	93	93	94	95	96	97

BP, blood pressure

* The 90th percentile is 1.28 SD, 95th percentile is 1.645 SD, and the 99th percentile is 2.326 SD over the mean.

For research purposes, the standard deviations in Appendix Table B–1 allow one to compute BP Z-scores and percentiles for boys with height percentiles given in Table 3 (i.e., the 5th,10th, 25th, 50th, 75th, 90th, and 95th percentiles). These height percentiles must be converted to height Z-scores given by (5% = -1.645; 10% = -1.28; 25% = -0.68; 50% = 0; 75% = 0.68; 90% = 1.28%; 95% = 1.645) and then computed according to the methodology in steps 2–4 described in Appendix B. For children with height percentiles other than these, follow steps 1–4 as described in Appendix B.

APPENDIX 11: BLOOD PRESSURE LEVELS FOR GIRLS

Blood Pressure Levels for Girls by Age and Height Percentile

Age (Year)	BP Percentile ↓	Systolic BP (mmHg) ← Percentile of Height →							Diastolic BP (mmHg) ← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
1	50th	83	84	85	86	88	89	90	38	39	39	40	41	41	42
	90th	97	97	98	100	101	102	103	52	53	53	54	55	55	56
	95th	100	101	102	104	105	106	107	56	57	57	58	59	59	60
	99th	108	108	109	111	112	113	114	64	64	65	65	66	67	67
2	50th	85	85	87	88	89	91	91	43	44	44	45	46	46	47
	90th	98	99	100	101	103	104	105	57	58	58	59	60	61	61
	95th	102	103	104	105	107	108	109	61	62	62	63	64	65	65
	99th	109	110	111	112	114	115	116	69	69	70	70	71	72	72
3	50th	86	87	88	89	91	92	93	47	48	48	49	50	50	51
	90th	100	100	102	103	104	106	106	61	62	62	63	64	64	65
	95th	104	104	105	107	108	109	110	65	66	66	67	68	68	69
	99th	111	111	113	114	115	116	117	73	73	74	74	75	76	76
4	50th	88	88	90	91	92	94	94	50	50	51	52	52	53	54
	90th	101	102	103	104	106	107	108	64	64	65	66	67	67	68
	95th	105	106	107	108	110	111	112	68	68	69	70	71	71	72
	99th	112	113	114	115	117	118	119	76	76	76	77	78	79	79
5	50th	89	90	91	93	94	95	96	52	53	53	54	55	55	56
	90th	103	103	105	106	107	109	109	66	67	67	68	69	69	70
	95th	107	107	108	110	111	112	113	70	71	71	72	73	73	74
	99th	114	114	116	117	118	120	120	78	78	79	79	80	81	81
6	50th	91	92	93	94	96	97	98	54	54	55	56	56	57	58
	90th	104	105	106	108	109	110	111	68	68	69	70	70	71	72
	95th	108	109	110	111	113	114	115	72	72	73	74	74	75	76
	99th	115	116	117	119	120	121	122	80	80	80	81	82	83	83
7	50th	93	93	95	96	97	99	99	55	56	56	57	58	58	59
	90th	106	107	108	109	111	112	113	69	70	70	71	72	72	73
	95th	110	111	112	113	115	116	116	73	74	74	75	76	76	77
	99th	117	118	119	120	122	123	124	81	81	82	82	83	84	84
8	50th	95	95	96	98	99	100	101	57	57	57	58	59	60	60
	90th	108	109	110	111	113	114	114	71	71	71	72	73	74	74
	95th	112	112	114	115	116	118	118	75	75	75	76	77	78	78
	99th	119	120	121	122	123	125	125	82	82	83	83	84	85	86
9	50th	96	97	98	100	101	102	103	58	58	58	59	60	61	61
	90th	110	110	112	113	114	116	116	72	72	72	73	74	75	75
	95th	114	114	115	117	118	119	120	76	76	76	77	78	79	79
	99th	121	121	123	124	125	127	127	83	83	84	84	85	86	87
10	50th	98	99	100	102	103	104	105	59	59	59	60	61	62	62
	90th	112	112	114	115	116	118	118	73	73	73	74	75	76	76
	95th	116	116	117	119	120	121	122	77	77	77	78	79	80	80
	99th	123	123	125	126	127	129	129	84	84	85	86	86	87	88

Blood Pressure Levels for Girls by Age and Height Percentile (Continued)

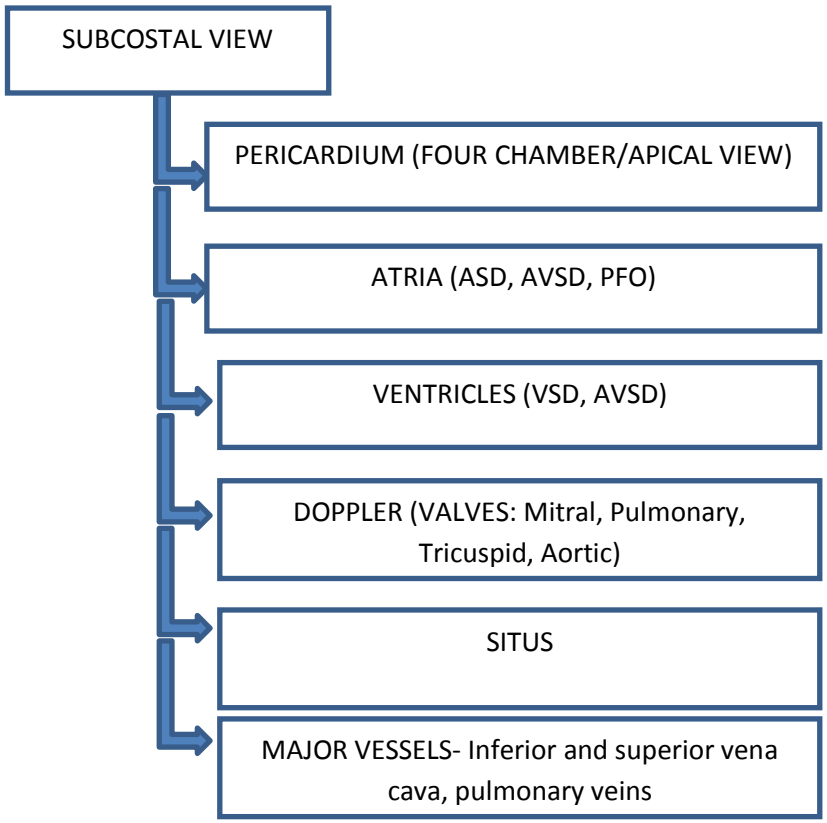
Age (Year)	BP Percentile ↓	Systolic BP (mmHg) ← Percentile of Height →							Diastolic BP (mmHg) ← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
11	50th	100	101	102	103	105	106	107	60	60	60	61	62	63	63
	90th	114	114	116	117	118	119	120	74	74	74	75	76	77	77
	95th	118	118	119	121	122	123	124	78	78	78	79	80	81	81
	99th	125	125	126	128	129	130	131	85	85	86	87	87	88	89
12	50th	102	103	104	105	107	108	109	61	61	61	62	63	64	64
	90th	116	116	117	119	120	121	122	75	75	75	76	77	78	78
	95th	119	120	121	123	124	125	126	79	79	79	80	81	82	82
	99th	127	127	128	130	131	132	133	86	86	87	88	88	89	90
13	50th	104	105	106	107	109	110	110	62	62	62	63	64	65	65
	90th	117	118	119	121	122	123	124	76	76	76	77	78	79	79
	95th	121	122	123	124	126	127	128	80	80	80	81	82	83	83
	99th	128	129	130	132	133	134	135	87	87	88	89	89	90	91
14	50th	106	106	107	109	110	111	112	63	63	63	64	65	66	66
	90th	119	120	121	122	124	125	125	77	77	77	78	79	80	80
	95th	123	123	125	126	127	129	129	81	81	81	82	83	84	84
	99th	130	131	132	133	135	136	136	88	88	89	90	90	91	92
15	50th	107	108	109	110	111	113	113	64	64	64	65	66	67	67
	90th	120	121	122	123	125	126	127	78	78	78	79	80	81	81
	95th	124	125	126	127	129	130	131	82	82	82	83	84	85	85
	99th	131	132	133	134	136	137	138	89	89	90	91	91	92	93
16	50th	108	108	110	111	112	114	114	64	64	65	66	66	67	68
	90th	121	122	123	124	126	127	128	78	78	79	80	81	81	82
	95th	125	126	127	128	130	131	132	82	82	83	84	85	85	86
	99th	132	133	134	135	137	138	139	90	90	90	91	92	93	93
17	50th	108	109	110	111	113	114	115	64	65	65	66	67	67	68
	90th	122	122	123	125	126	127	128	78	79	79	80	81	81	82
	95th	125	126	127	129	130	131	132	82	83	83	84	85	85	86
	99th	133	133	134	136	137	138	139	90	90	91	91	92	93	93

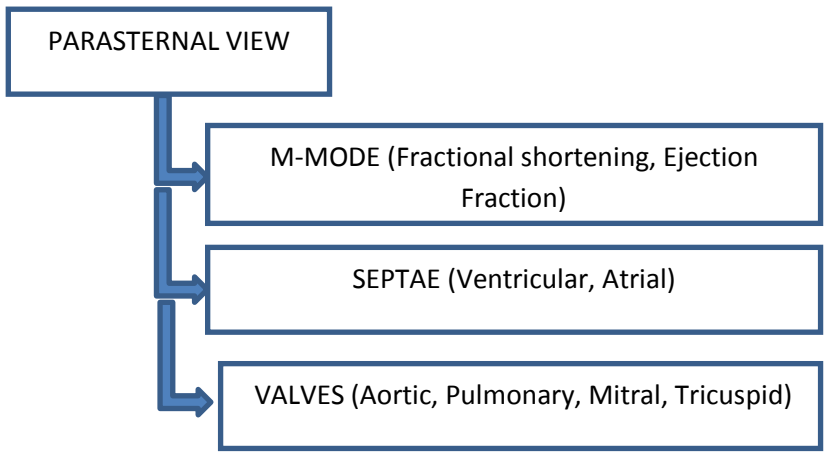
BP, blood pressure

* The 90th percentile is 1.28 SD, 95th percentile is 1.645 SD, and the 99th percentile is 2.326 SD over the mean.

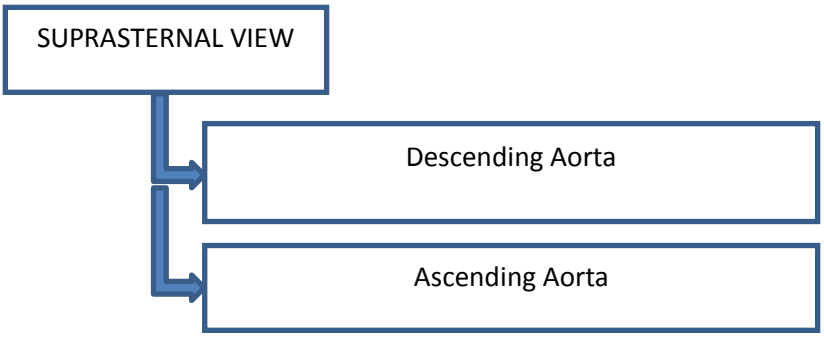
For research purposes, the standard deviations in Appendix Table B–1 allow one to compute BP Z-scores and percentiles for girls with height percentiles given in Table 4 (i.e., the 5th,10th, 25th, 50th, 75th, 90th, and 95th percentiles). These height percentiles must be converted to height Z-scores given by (5% = -1.645; 10% = -1.28; 25% = -0.68; 50% = 0; 75% = 0.68; 90% = 1.28%; 95% = 1.645) and then computed according to the methodology in steps 2–4 described in Appendix B. For children with height percentiles other than these, follow steps 1–4 as described in Appendix B.

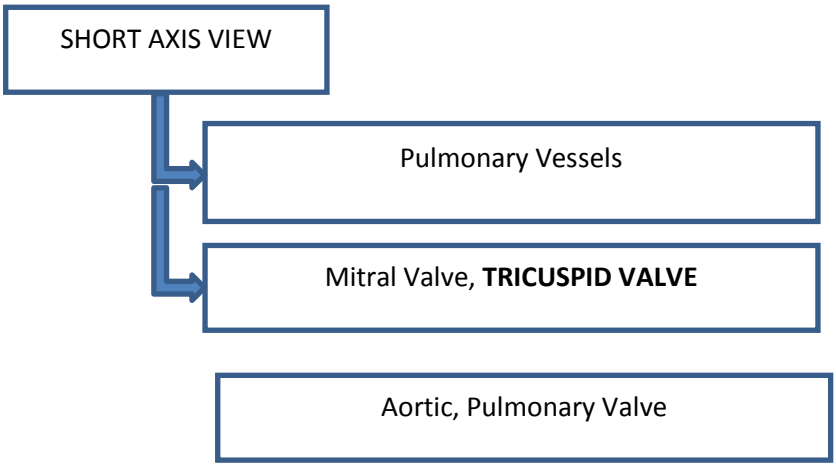
APPENDIX 12: AMERICAN ECHOCARDIOGRAPHY SOCIETY PROTOCOLS FOR FULL ECHOCARDIOGRAPHIC EVALUATION IN PAEDIATRICS





APPENDIX 12: Continued





*CHEMLA EQUATION: = (0.61*SPAP) +2 mmHg

APPENDIX 13: LIST OF EQUIPMENT AND MATEIRIAL USED FOR CLINICAL EVALUATION OF THE PATIENTS

vii. Pediatric Litmann Stethoscope

viii. Aneroid Blood Pressure Machine (Brand: Risan), with appropriate blood pressure cuffs

ix. Digital Blood Pressure Machine (Brand: Omron), with paediatic cuff

x. Hand held oxygen saturation ($SPO_{2)}$ machine

xi. Toshiba pen torch

xii. ENT Department Rhinoscopy Set

xiii. Dial pan weighing scale, and bathroom weighing scale

xiv. Heightometer and a simple length measuring board

xv. Tape measures

xvi. Phillips CX Color Ultrasound Echocardiography Machine

APPENDIX 14: IREC APPROVAL

INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 3
ELDORET
Tel: 33471//2/3

MOI UNIVERSITY
SCHOOL OF MEDICINE
P.O. BOX 4606
ELDORET

Reference: IREC/2014/175
Approval Number: 0001305

18th November, 2014

Dr. Grace Nyambura,
Moi University,
School of Medicine,
P.O. Box 4606-30100,
ELDORET-KENYA.

INSTITUTIONAL RESEARCH & ETHICS COMMITTEE
18 NOV 2014
RECEIVED
P. O. Box 4606-30100 ELDORET

Dear Dr. Nyambura,

RE: FORMAL APPROVAL

The Institutional Research and Ethics Committee has reviewed your research proposal titled:-

"Clinical Profile of Children with Adenotonsillar Hypertrophy at Moi Teaching and Referral Hospital."

Your proposal has been granted a Formal Approval Number: ***FAN: IREC 1305*** on 18th November, 2014. You are therefore permitted to begin your investigations.

Note that this approval is for 1 year; it will thus expire on 17th November, 2015. If it is necessary to continue with this research beyond the expiry date, a request for continuation should be made in writing to IREC Secretariat two months prior to the expiry date.

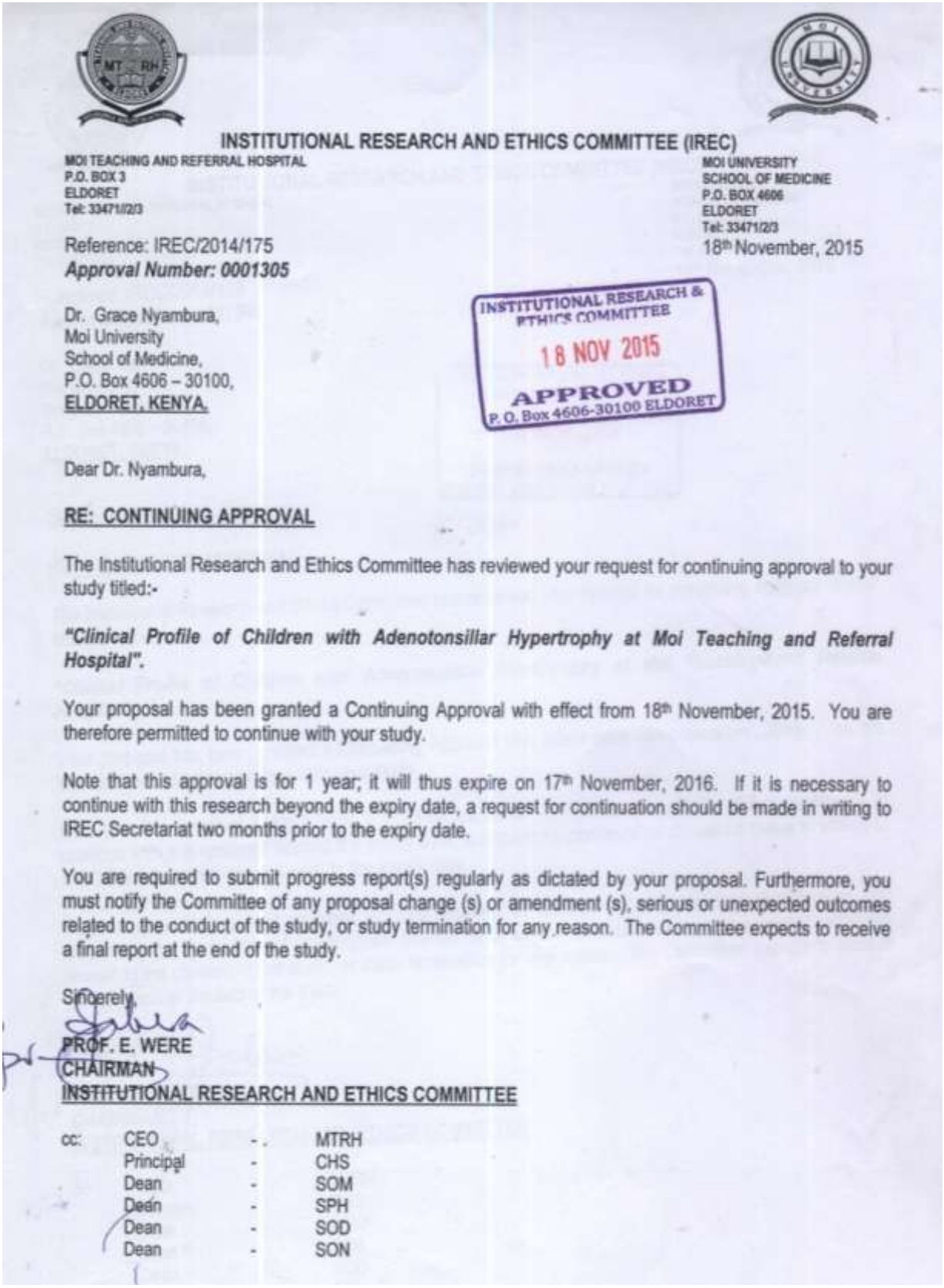
You are required to submit progress report(s) regularly as dictated by your proposal. Furthermore, you must notify the Committee of any proposal change (s) or amendment (s), serious or unexpected outcomes related to the conduct of the study, or study termination for any reason. The Committee expects to receive a final report at the end of the study.

Sincerely,

PROF. E. WERE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc Director - MTRH Dean - SOP Dean - SOM
Principal - CHS Dean - SON Dean - SOD

APPENDIX 15: CONTINUING APPROVAL



INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 3
ELDORET
Tel: 33471/2/3

MOI UNIVERSITY
SCHOOL OF MEDICINE
P.O. BOX 4606
ELDORET
Tel: 33471/2/3

Reference: IREC/2014/175
Approval Number: 0001305

18th November, 2015

INSTITUTIONAL RESEARCH & ETHICS COMMITTEE
18 NOV 2015
APPROVED
P. O. Box 4606-30100 ELDORET

Dr. Grace Nyambura,
Moi University
School of Medicine,
P.O. Box 4606 – 30100,
ELDORET, KENYA.

Dear Dr. Nyambura,

RE: CONTINUING APPROVAL

The Institutional Research and Ethics Committee has reviewed your request for continuing approval to your study titled:-

"Clinical Profile of Children with Adenotonsillar Hypertrophy at Moi Teaching and Referral Hospital".

Your proposal has been granted a Continuing Approval with effect from 18th November, 2015. You are therefore permitted to continue with your study.

Note that this approval is for 1 year; it will thus expire on 17th November, 2016. If it is necessary to continue with this research beyond the expiry date, a request for continuation should be made in writing to IREC Secretariat two months prior to the expiry date.

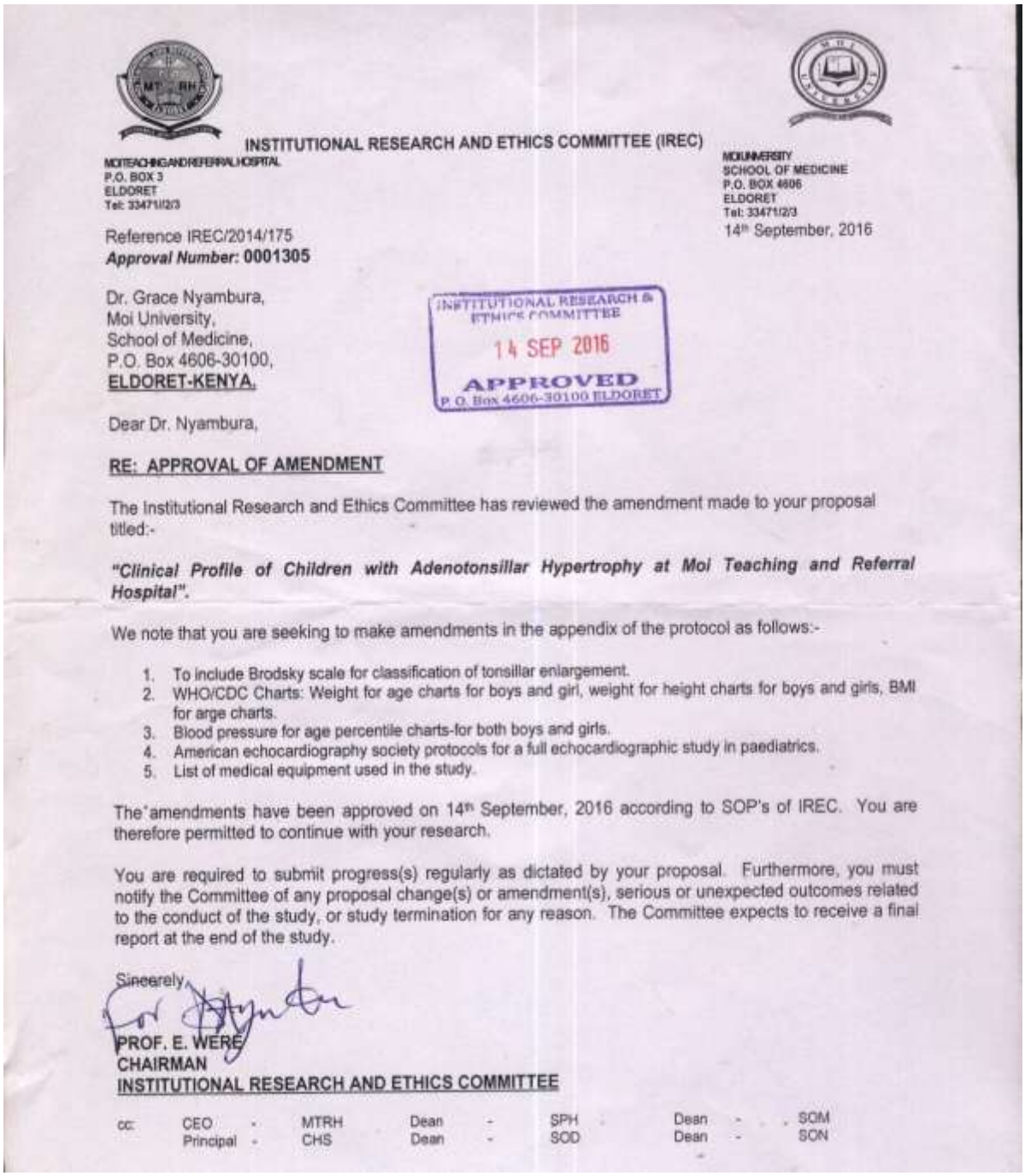
You are required to submit progress report(s) regularly as dictated by your proposal. Furthermore, you must notify the Committee of any proposal change (s) or amendment (s), serious or unexpected outcomes related to the conduct of the study, or study termination for any reason. The Committee expects to receive a final report at the end of the study.

Sincerely,

PROF. E. WERE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc: CEO - MTRH
Principal - CHS
Dean - SOM
Dean - SPH
Dean - SOD
Dean - SON

APPENDIX 16: APPROVAL OF AMENDMENT



INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE (IREC)

MOI TEACHING AND REFERRAL HOSPITAL
P.O. BOX 3
ELDORET
Tel: 33471/2/3

MOI UNIVERSITY
SCHOOL OF MEDICINE
P.O. BOX 4606
ELDORET
Tel: 33471/2/3

14th September, 2016

Reference IREC/2014/175
***Approval Number:* 0001305**

Dr. Grace Nyambura,
Moi University,
School of Medicine,
P.O. Box 4606-30100,
ELDORET-KENYA.

INSTITUTIONAL RESEARCH & ETHICS COMMITTEE
14 SEP 2016
APPROVED
P. O. Box 4606-30100 ELDORET

Dear Dr. Nyambura,

RE: APPROVAL OF AMENDMENT

The Institutional Research and Ethics Committee has reviewed the amendment made to your proposal titled:-

"Clinical Profile of Children with Adenotonsillar Hypertrophy at Moi Teaching and Referral Hospital".

We note that you are seeking to make amendments in the appendix of the protocol as follows:-

1. To include Brodsky scale for classification of tonsillar enlargement.
2. WHO/CDC Charts: Weight for age charts for boys and girl, weight for height charts for boys and girls, BMI for arge charts.
3. Blood pressure for age percentile charts-for both boys and girls.
4. American echocardiography society protocols for a full echocardiographic study in paediatrics.
5. List of medical equipment used in the study.

The amendments have been approved on 14th September, 2016 according to SOP's of IREC. You are therefore permitted to continue with your research.

You are required to submit progress(s) regularly as dictated by your proposal. Furthermore, you must notify the Committee of any proposal change(s) or amendment(s), serious or unexpected outcomes related to the conduct of the study, or study termination for any reason. The Committee expects to receive a final report at the end of the study.

Sincerely,

PROF. E. WERE
CHAIRMAN
INSTITUTIONAL RESEARCH AND ETHICS COMMITTEE

cc: CEO - MTRH, Dean - SPH, Dean - SOM
Principal - CHS, Dean - SOD, Dean - SON