

**PREVALENCE OF PULMONARY HYPERTENSION IN
CHILDREN WITH ADENOID OR ADENOTONSILLAR
HYPERTROPHY AT THE KENYATTA NATIONAL
HOSPITAL**

DR. DIANA MARANGU (MB ChB)

**DISSERTATION SUBMITTED IN PART FULFILLMENT OF THE
REQUIREMENTS OF THE UNIVERSITY OF NAIROBI FOR THE
AWARD OF THE DEGREE MASTER IN MEDICINE PAEDIATRICS
AND CHILD HEALTH**

University of NAIROBI Library



0502726 3

**UNIVERSITY OF NAIROBI
MEDICAL LIBRARY**

DECLARATION

This dissertation is my original work and has not been presented for the award of a degree in any other university.

Investigator

Dr. Diana Marangu (MB ChB)

Senior House Officer - Department of Paediatrics and Child Health, University of Nairobi

Tel: (+254 722) 282815

Email: marangud@yahoo.com

Signature : 

Date : 16th November 2012

APPROVAL BY SUPERVISORS

This dissertation has been presented with our full approval as supervisors.

Prof. Ruth Nduati (MB ChB, MMED Paeds, MPH)

Associate Professor of Paediatrics and Child Health

Chairperson - Department of Paediatrics and Child Health, University of Nairobi

Tel: (+254 722) 235323

Email: ruth_nduati2000@yahoo.com

Signature:



Date:

16/11/2012

Dr. Christine Jowi (MB ChB, MMED Paeds, Cardiology)

Senior Lecturer, Consultant Paediatrician & Cardiologist

Department of Paediatrics and Child Health, University of Nairobi

Tel: (+254 722) 293454

Email: jocajowi@africaonline.co.ke

Signature:



Date:

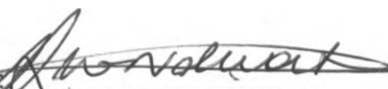
16/11/2012

Dr. Joyce Aswani (MB ChB, MMED ENT Surg)
Lecturer and Consultant Otolaryngologist & Surgeon
Department of ENT Surgery, University of Nairobi
Tel: (+254 722) 814483
Email: ioyceaswani@hotmail.com

Signature : 


Date : 16-11-2012

Dr. Florence Murila (MB ChB, MMED Paeds. Neonatology)
Senior Lecturer and Consultant Neonatologist
Department of Paediatrics and Child Health, University of Nairobi
Tel: (+254 729) 430022
Email: fmurila@gmail.com

Signature : 
for Dr. Murila

Date : 16/11/2012

Dr. Wambani Sidika (MB ChB, MMED Radiology)
Honorary Lecturer (UON), Consultant Paediatric Radiologist & Chief of Radiology
Department of Radiology, Kenyatta National Hospital Nairobi
Tel: (+254 722) 711065
Email: wamsidi@gmail.com

Signature : 

Date : 16TH NOV. 2012

DEDICATION

To my beloved family: my father Mr. Julius Brown Marangu, my mother Mrs. Florence Kagwiria Marangu and my siblings, Brenda Kananu and Vincent Mutuma.

ACKNOWLEDGEMENT

I would like to express my sincere appreciation to:-

- My supervisors: Prof. Ruth Nduati, Dr. Christine Jowi, Dr. Joyce Aswani, Dr. Sidika Wambani and Dr. Florence Murila for guidance and invaluable input throughout the study process.
- The caregivers and their children who participated willingly in the study.
- The Hurlingham Heart Clinic for wholly sponsoring the echocardiography.
- Prof. Isaac Muthure Macharia, Mr. Joseph Irungu, Matron Marianne Irungu, the ENT clinicians especially Mr. Maurice Mudeny, Mr. Abdinasir Haji and the postgraduate students especially Dr. Elaine Yuko, Dr. Gachambi Mwangi and Dr. Meera Patel who enabled me undertake this study at the ENT clinic
- Mrs. Mryanne Kasomo and Mrs. Mary Bwonya who assisted in taking the radiographs and evaluating image quality.
- Philip Ayieko and Kenneth Mutai for assisting me in data analysis.
- All my friends and colleagues who have provided guidance in one way or another especially Dr. Ambrose Agweyu, Dr. Jeanette Dawa and Dr. Gakuo Karuga.
- Kenyatta National Hospital and the University of Nairobi.
- The Marangu family for their invaluable support during the various stages of this manuscript.

TABLE OF CONTENTS

DECLARATION	i
APPROVAL BY SUPERVISORS	ii
DEDICATION.....	iv
ACKNOWLEDGEMENT.....	v
TABLE OF CONTENTS	vi
LIST OF TABLES	viii
LIST OF FIGURES	ix
STUDY DEFINITIONS.....	xi
ABSTRACT	xiii
INTRODUCTION AND LITERATURE REVIEW	1
Diagnosis of adenoid and tonsillar hypertrophy	2
Complications of adenotonsillar hypertrophy	5
Pulmonary hypertension in adenotonsillar hypertrophy	5
Diagnosis of pulmonary hypertension in adenotonsillar hypertrophy	6
Prevalence of pulmonary hypertension in adenotonsillar hypertrophy.....	11
Treatment of Pulmonary Hypertension in Adenotonsillar Hypertrophy.....	15
STUDY JUSTIFICATION AND UTILITY	16
STUDY OBJECTIVES.....	17

Primary Objective	17
Secondary Objective	17
MATERIALS AND METHODS.....	18
Study Area, Design, Sample Size and Population	18
Procedures	21
Ethical Considerations.....	27
DATA MANAGEMENT AND ANALYSIS.....	29
RESULTS.....	31
DISCUSSION.....	46
CONCLUSION.....	54
RECOMMENDATIONS	54
REFERENCES	54
APPENDICES	62

LIST OF TABLES

Table 1: Summary of relevant studies on pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH	14
Table 2: Patient characteristics of children with adenoid or adenotonsillar hypertrophy at KNH	33
Table 3: Patient characteristics associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH.....	34
Table 4: Daily symptoms associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH.....	36
Table 5: Sensitivity, specificity, positive and negative predictive values of clinical parameters for pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH.....	37
Table 6: Combination of best clinical predictors of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH.....	38
Table 7: Physical examination findings associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH.....	42
Table 8: Image quality of the lateral neck radiographs in the children with in children with adenoid or adenotonsillar hypertrophy at KNH.....	43
Table 9: Proportion of children in the study with ANR >0.63 or TPR >0.66.....	43
Table 10: Independent factors associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH.....	42

LIST OF FIGURES

Figure 1: Grading of tonsil hypertrophy as proposed by L. Brodsky.....3

Figure 2: Lateral neck radiograph measurements as proposed by Shintani.....4

Figure 3: Echocardiographic findings in pulmonary hypertension.....8

Figure 4: Flow chart depicting study participant enrollment.....21

Figure 5: Prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH31

Figure 6: Distribution of echo derived mPAP in children with adenoid or adenotonsillar hypertrophy at KNH.....32

Figure 7: Frequency of day and night symptoms in children with adenoid or adenotonsillar hypertrophy at KNH.....35

Figure 8: Receiver operator curve for symptoms in children with pulmonary hypertension and adenoid or adenotonsillar hypertrophy at KNH.....38

Figure 9: Frequency of cardiovascular findings in children with adenoid or adenotonsillar hypertrophy at KNH39

Figure 10: Distribution of oxygen saturations on pulse oximetry in children with adenoid or adenotonsillar hypertrophy at KNH.....40

Figure 11: Tonsil size on ENT examination findings in children with adenoid or adenotonsillar hypertrophy at KNH..... 41

Figure 12: Box plot for lateral neck radiograph findings in children with pulmonary hypertension and adenoid or adenotonsillar hypertrophy at KNH.....44

LIST OF ABBREVIATIONS

ANR – adenoid nasopharyngeal ratio

ENT – Ear, Nose and Throat

KNH – Kenyatta National Hospital

mPAP – Mean pulmonary arterial pressure

mPAPest – Estimated mean pulmonary arterial pressure

sPAP – Systolic pulmonary arterial pressure

TPR – Tonsil pharyngeal ratio

WHO – World Health Organization

WHZ – Weight for height

STUDY DEFINITIONS

1. **Clinician diagnosed adenoid hypertrophy**

Adenoid hypertrophy diagnosed by any clinician at the Kenyatta National Hospital.

2. **Radiologically confirmed adenoid hypertrophy**

Adenoid hypertrophy documented on a lateral neck radiograph by any radiologist at the Kenyatta National Hospital.

3. **Predominant adenoid hypertrophy**

Clinician diagnosed and radiologically confirmed adenoid hypertrophy with tonsil grade 0-2 on Brodsky classification as illustrated in Figure 3.⁴⁵

4. **Predominant adenotonsillar hypertrophy**

Clinician diagnosed and radiologically confirmed adenoid hypertrophy with tonsil grade 3-4 on Brodsky classification as illustrated in Figure 3.⁴⁵

5. **Hypoxia**

Oxygen saturations < 92% on pulse oximetry.⁷²

6. **Tachypnoea**

An elevated respiratory rate above cut-off for age as defined by the World Health Organization (WHO) cut-offs for respiratory tract infections. Respiratory rate >50 breathes per minute in children aged 2 to 11 months; >40 breathes per minute in children aged 12 to 59 months and >30 breathes per minute in children aged 12 to 59 months.⁵⁰

7. **Tachycardia**

An elevated heart rate as measured while the patient is calm above the upper limit for age. Heart rate >135 beats per minute in children aged 6 to 12 months; >115

beats per minute in children aged 13 to 59 months and >100 beats per minute in children aged 12 to 59 months.⁷⁰

8. Adenoid nasopharygeal ratio (ANR)

The adenoid size measured in centimeters divided by the nasopharyngeal size measured in centimeters.

9. Tonsil pharyngeal ratio (TPR)

The tonsil size measured in centimeters divided by the pharyngeal size in measured in centimeters.

10. Good quality image

Lateral neck radiograph taken with the mouth closed, without rotation, or magnification.

11. Poor quality image

Lateral neck radiograph taken with the mouth open, rotated or magnified.

12. Pulmonary hypertension

Mean pulmonary arterial pressure (mPAP) \geq 25mmHg determined after subjecting echocardiographically derived systolic pulmonary arterial pressure (sPAP) to the Chemla equation ($0.61 * sPAP + 2mmHg$).^{12,43,60,61}

ABSTRACT

Background: Adenotonsillar hypertrophy is a common condition in childhood, whose serious complications of pulmonary hypertension and cor-pulmonale are common and devastating but local prevalence is unknown.

Objectives: To determine the prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at Kenyatta National Hospital (KNH) and secondarily determine the clinical-radiological factors associated with pulmonary hypertension in these children.

Methods: This was a cross sectional descriptive study in children aged 0-12 years attending ENT clinic and general pediatric wards at KNH, with clinician diagnosed adenoid hypertrophy confirmed on lateral neck radiography. Eligible patients were consecutively recruited into the study between September and November 2011. The patients were evaluated for symptoms, physical findings, lateral neck radiograph measurements of the adenoids, tonsils and airways; and Doppler echocardiographic assessment of systolic pulmonary artery pressure (sPAP). The paediatric radiologist and the cardiologist were blinded to the patients' severity of symptoms. Pulmonary hypertension was defined as mPAP of $>25\text{mmHg}$ estimated by the Chemla equation ($0.61\text{sPAP}+2\text{minHg}$).

Results: The prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH is 21.1% (95% CI 14.3% to 29.4%). Independent

factors associated with pulmonary hypertension include daily hyperactivity (OR= 0.22 [95% CI 0.06 to 0.87] p=0.03), oxygen saturation (OR= 0.72 [95% CI 0.54 to 0.97] p=0.03) and palpable P2 (OR= 9.84 [95% CI 3.2 to 55.4] p=0.01). Mouth breathing singly or in combination with restless sleep on history showed the highest sensitivity (88.5%) and negative predictive value (86.4%) for pulmonary hypertension in these children.

Conclusion and Recommendations: One in every 5 children with clinician diagnosed and radiologically confirmed adenoid or adenotonsillar hypertrophy at KNH had pulmonary hypertension. Clinical screening and echocardiography evaluation is vital for early identification and prevention of pulmonary hypertension.

INTRODUCTION AND LITERATURE REVIEW

Adenotonsillar hypertrophy – Developmental anatomy and pathophysiology

The Waldeyer's ring consists of the nasopharyngeal tonsil or adenoid, tubal tonsil, palatine tonsils, and lingual tonsil. It is the lymphoid tissue of the upper respiratory tract. The adenoid tissue is located in the roof and posterior wall of the nasopharynx. The paired palatine tonsils lie within the anterior and posterior faucial pillars.¹ In this dissertation, tonsils refer to the palatine tonsils.

Globally, adenotonsillar hypertrophy is a common condition of childhood.¹ From birth until approximately the age of 12 years, the lymphoid tissue within the upper airway increases. It progressively reduces in size during adolescence and adulthood.² A study conducted in normal children showed that the growth of the lymphatic tissue of the upper airway was proportionate to the somatic growth of the surrounding tissues. This explains why normal children have stable airways; even during sleep.³ Any change in this symmetrical pattern of growth would be abnormal. Thus adenoid and tonsillar hypertrophy is considered pathologic only in certain groups of children.⁴ The causes of adenotonsillar hypertrophy are not completely understood. Factors associated with symptomatic enlargement of upper airway lymphatic tissue include microbial stimuli, external irritants, allergy and genetics.⁵⁻¹⁰

In addition to conferring local immunity, the upper airway also plays a role in respiration, swallowing and speech. It mostly comprises soft tissue and is collapsible to serve these functions. Negative intrathoracic pressure is generated following diaphragmatic contraction

during inspiration. However in the wakeful state, upper airway patency is maintained by increased pharyngeal neuromuscular tone. During sleep, this neuromuscular tone is reduced and the upper airway is more collapsible. Resistance is further increased if there is upper airway obstruction due to adenoid or tonsillar hypertrophy, resulting in partial or complete collapse. Hence there is reduced or absent airflow resulting in sleep disturbed breathing.

Sleep disordered breathing is a spectrum comprised of occasional snoring, habitual snoring, upper airway resistance syndrome and the extreme end of the continuum, obstructive sleep apnea hypopnea syndrome. Adenotonsillar hypertrophy is the single most significant anatomical risk factor for sleep disordered breathing in children aged 2 to 8 years. Other less common causes of sleep disordered breathing in children are obesity, craniofacial anomalies and neurologic disorders.^{1,12-14}

Diagnosis of adenoid and tonsillar hypertrophy

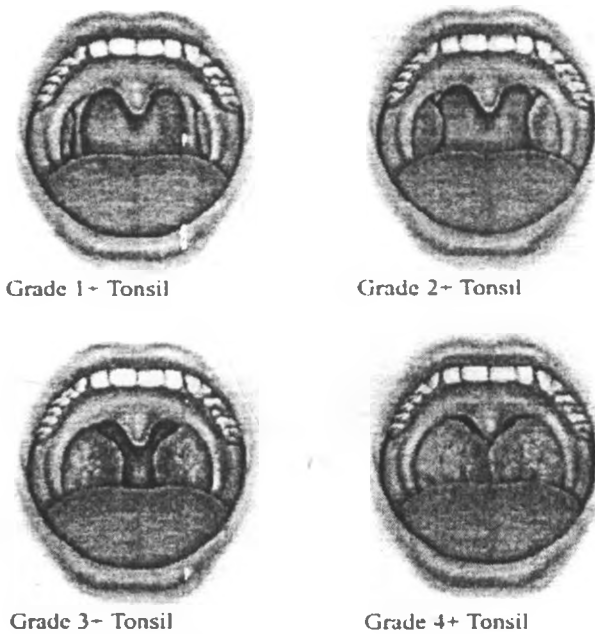
CLINICAL EVALUATION

The commonest presentation of children with significant adenotonsillar enlargement is snoring and difficulty in breathing during sleep. Parents additionally report of night sweats and restless sleep. Day time symptoms commonly include mouth breathing and nasal obstruction.^{15,16,17,18} Apnoeic attacks at night and frequent coughs and colds are additional significant symptoms experienced by children with adenoid and tonsillar hypertrophy.¹⁵

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.^{3,19,20} Grading tonsil hypertrophy on physical examination and measurements of adenoid and tonsil size by lateral neck radiography are diagnostic modalities that are accessible and affordable in our setting. Brodsky classification of tonsil hypertrophy is illustrated in Figure 1 below.⁶⁶

Figure 1: Grading of tonsil hypertrophy as proposed by L. Brodsky.



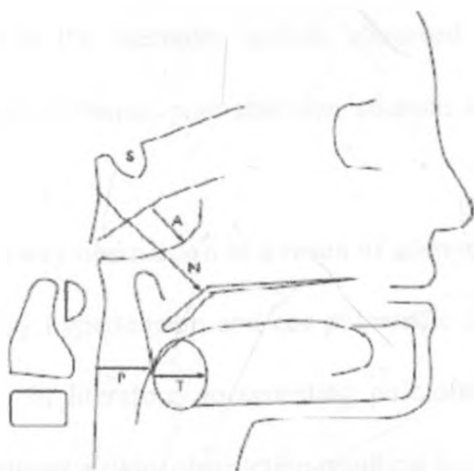
- (0) Tonsils are within the tonsillar fossa.
- (1+) Tonsils occupy <25 % of the lateral dimension of the oropharynx as measured between the anterior tonsillar pillars.
- (2+) Tonsils occupy < 50 % of the lateral dimension of the oropharynx.
- (3+) Tonsils occupy less than 75 % of the lateral dimension of the oropharynx.
- (4+) Tonsils occupy 75% or more of the lateral dimension of the oropharynx.

LATERAL NECK RADIOGRAPHY

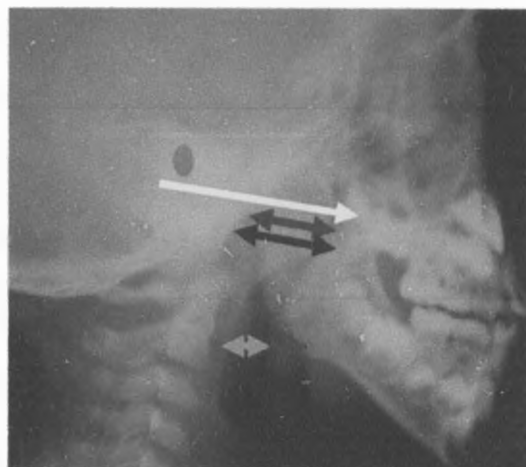
Lateral neck radiographs have been used in the diagnosis of adenoid hypertrophy and upper airway obstruction. Their greatest utility is in screening to determine the patients that require a thorough ENT follow up. Various cephalometry assessment methods have been described in the literature however, there is no consensus as to what are the most useful landmarks.^{67,68,69} Lateral neck radiograph assessment for adenoid size, nasopharyngeal diameter, adenoid nasopharyngeal ratio (ANR), tonsil size, pharyngeal diameter and tonsil

pharyngeal ratio (TPR) with their relevant landmarks as described by Shintani are illustrated in figure 2 below.⁵⁶

Figure 2: Lateral neck radiograph measurements as proposed by Shintani



Lateral neck radiograph measurements as proposed by Shintani



Lateral neck radiograph showing adenoid hypertrophy

- A, adenoid size
- N, nasopharyngeal measurement
- P, pharyngeal measurement
- S, sella turcica
- T, tonsil size

A and T are measured as a perpendicular distance at their maximum convexity
ANR - ratio of the width of the adenoid to the depth of the nasopharyngeal space
TPR - ratio of the width of the tonsil to the depth of the pharyngeal space

POLYSOMNOGRAPHY

Polysomnography is recognized as the gold standard to assess for the presence and severity of obstructive sleep apnea hypopnea syndrome.^{3,19,20} This diagnostic modality is currently not available in our setting.

Complications of adenotonsillar hypertrophy

More than a century ago, clinicians began recognizing the sequelae of sleep related upper airway obstruction. Adenotonsillar hypertrophy with consequent paediatric sleep disordered breathing is associated with a multitude of complications. The main areas reported in the literature include disturbed somatic growth, hyperactivity, aggression, behavioral problems, poor attention, enuresis and the cardiovascular morbidity.^{2,12,13,19}

Upper airway obstruction as a result of adenotonsillar hypertrophy is an important cause of pulmonary hypertension and cor pulmonale (right heart failure) in children.²¹⁻³¹ The first reference in literature documenting pulmonary hypertension occurring in children with chronic upper airway obstruction resulting from adenotonsillar hypertrophy was in 1965 by Menashe and colleagues and since then, there have been numerous publications on pulmonary hypertension due to adenotonsillar hypertrophy.²¹⁻³⁸

Pulmonary hypertension in adenotonsillar hypertrophy

Upper airway obstruction results in hypoxemia, re-oxygenation and respiratory acidosis induced by hypercarbia. This leads to oxidative stress, inflammation, endothelial dysfunction with consequent vasoconstriction of the pulmonary vasculature. The tunica muscularis of the medium and small sized pulmonary arteries hypertrophies.^{28,32-34} In the acute phase, the pulmonary vasoconstriction is reversible by alleviating the chronic airway obstruction. Chronic vasoconstriction in the long term results in structural remodeling of the pulmonary vascular bed.³⁵⁻³⁷ These subsequent chronic structural changes to the pulmonary arteriolar bed may only be partially reversible once the chronic upper airway obstruction is relieved. Irreversible pulmonary hypertension and cor-pulmonale ensue.^{23,25}

Diagnosis of pulmonary hypertension in adenotonsillar hypertrophy

Clinical evaluation, electrocardiography, and imaging modalities such as chest radiography and echocardiography with Doppler may be employed in the diagnosis of pulmonary hypertension.⁴²⁻⁴³ The gold standard diagnostic test for the evaluation of pulmonary hypertension is by direct catheterization of the right ventricle and pulmonary artery, which is highly invasive and expensive.³⁸⁻⁴⁰

CARDIAC CATHETERIZATION

According to the updated classification of pulmonary hypertension of Dana Point 2008 as well as the Pulmonary Vascular Research Institute (PVRI) pediatric taskforce of Panama 2011, pulmonary hypertension is a hemodynamic and pathophysiologic condition defined as an increase in mean pulmonary arterial pressure (mPAP) ≥ 25 mmHg at rest as assessed by right heart catheterization.^{42-43,56}

Studies have shown that the normal mPAP at rest is 14–18 mmHg, with an upper limit of normal of about 20 mmHg. Further evaluation of patients presenting with mPAP between 21 and 24 mmHg has been recommended in epidemiologic studies, as the significance of this range has not been established.⁴²⁻⁴³

CLINICAL EVALUATION

Children with pulmonary hypertension present with non specific symptoms such as fatigue, weakness, angina, syncope or abdominal distension. Examination findings may include a left parasternal heave and a palpable second heart sound (P2). Auscultation may reveal a

pansystolic murmur of tricuspid regurgitation and a diastolic murmur of pulmonary regurgitation. Other findings that may be appreciated include jugular venous distension, hepatomegaly, peripheral edema, ascites and evidence of poor perfusion as features of right heart failure in advanced disease. Clues to the cause (e.g. adenoid hypertrophy on lateral neck radiograph) should be explored.^{42,43}

ELECTROCARDIOGRAPHY

Supportive evidence of pulmonary hypertension may be provided by electrocardiography. Right ventricular hypertrophy, strain and dilatation may be evident. Electrocardiography has insufficient sensitivity (55%) and specificity (70%) to be a screening tool for detecting significant pulmonary hypertension.^{42,43}

CHEST RADIOGRAPHY

Chest radiography reveals central pulmonary arterial dilation and loss of the peripheral blood vessels. In advanced disease, right atrial and right ventricular enlargement may be seen. The degree of pulmonary hypertension does not correlate with the extent of chest radiographic abnormalities.^{42,43}

ECHOCARDIOGRAPHY

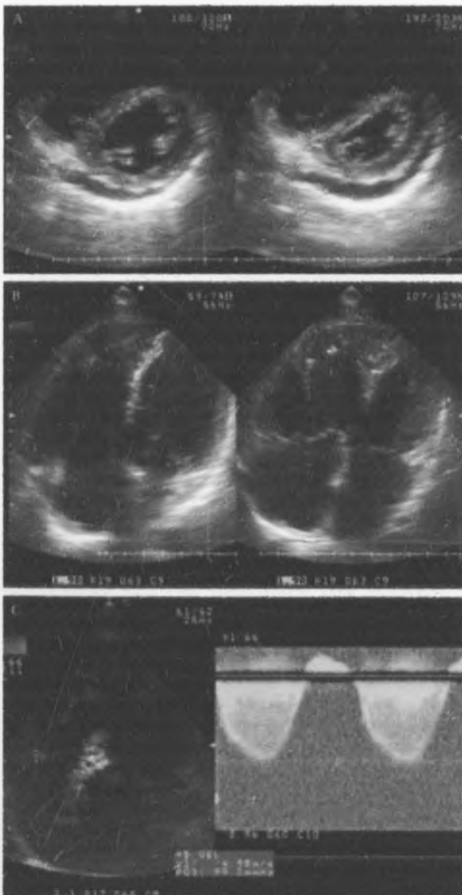
M-Mode

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.

2- Dimensional echocardiography

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. Figure 3 illustrates some of these features.

Figure 3: Echocardiographic findings in pulmonary hypertension



(A) Parasternal short axis view.

Paradoxical septal motion - flattening or D shape of the interventricular septum.

(B) Apical four chamber view.

Right atrial and ventricular enlargement, flattening or D shape of the interventricular septum, and underfilled left heart chambers.

(C) Doppler analysis.

Systolic tricuspid regurgitant velocity 4.98m/s and an estimated sPAP of 99.2mmHg which is severe pulmonary hypertension.

Doppler echocardiography

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 coefficient $r = 0.93$, standard estimate error 8mmHg) demonstrating its utility in the monitoring and screening of pulmonary hypertension.⁴¹

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.³⁸⁻⁴³

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.⁶⁰⁻⁶⁴ A well investigated equation proposed by Aduen et al and Chemla et al to estimate mPAP (mPAPest) is calculated as systolic PAP (sPAP) $\times 0.61 + 2$ mmHg.^{60,61} Other equations documented in the literature that have been used to estimate mPAP include the Syed and the Mahan formulae which are comparable with regard to sensitivity, specificity and predictive values when compared to cardiac catheterization.^{60,63,64} The Chemla equation is the simplest to apply.

Estimated mPAP using the Chemla equation has been shown to have an acceptable accuracy of 77% to 98% within 10mmHg of right heart catheterization measured mPAP and thus suitable for clinical use.^{60,61} An estimated mPAP ≥ 25.5 mmHg using the Chemla equation is useful in the diagnosis of pulmonary hypertension with an excellent sensitivity (98%), specificity (100%), positive (98%) and negative (88%) predictive value.⁶³ Studies done to validate the Chemla equation were all in adults.

Prevalence of pulmonary hypertension in adenotonsillar hypertrophy

A descriptive study of electrocardiograph (ECG) and chest radiograph changes in children with adenoid and tonsillar hypertrophy aged 0-60 months with a peak of 11-20 months, undertaken at KNH revealed that 10% of the children had features of right ventricular hypertrophy on ECG, and 11% of the children had features of cardiomegaly on chest radiograph.¹⁵ Children less than 2 years of age have a normal right axis deviation and these features on ECG may be normal. To our knowledge, no other study on cardiovascular complications in children with adenoid or adenotonsillar hypertrophy has been conducted in Kenya.

Three African studies addressing pulmonary hypertension in children with adenoid and adenotonsillar hypertrophy were identified in the literature. They were all from Egypt. In a study published in 2003 by El-Hoshy and colleagues, pulmonary hypertension was detected in 12 out of 60 children representing a point prevalence of 20%.²⁴ In 2005, Elmofty et al conducted a study to evaluate the effect of adenotonsillectomy on pulmonary arterial pressure in 30 children with upper airway obstruction due to adenotonsillar hypertrophy. Eight out of thirty (26.7%) children had pulmonary hypertension. In this study, pulmonary hypertension was defined as sPAP >30mmHg. Abdel-Aziz in 2011 looked at asymptomatic cardiopulmonary changes caused by adenotonsillar hypertrophy in 95 children, divided into 4 groups. These comprised children with adenoid hypertrophy (40), adenotonsillar hypertrophy (35), tonsillar hypertrophy (6) and normal children (14). Despite defining pulmonary hypertension as mPAP >20mmHg, a point prevalence of pulmonary hypertension was not reported.

In Turkey, Naiboglu et al examined the elevation of pulmonary arterial pressures in 39 children aged 3 to 10 years, with upper airway obstruction caused by adenotonsillar hypertrophy. Every child with adenotonsillar hypertrophy had some probability of having pulmonary hypertension regardless of his or her disease severity. A recommendation of echocardiographic examination in all children with adenotonsillar hypertrophy was made in this study and it was shown to be beneficial for assessing the cardiopulmonary status of the patient and useful at decision making for adenotonsillectomy.⁴⁴

Another study from Turkey conducted by Yilmaz and colleagues evaluated 52 children aged 4 to 11 years with upper airway obstruction due to adenotonsillar hypertrophy. In this study, pulmonary hypertension was defined as mPAP > 20mmHg determined using the Mahan formula. The point prevalence of pulmonary hypertension in this study was 51.9% with 27 children out of 52 children with pulmonary hypertension. Pulmonary hypertension in this study was determined as mPAP > 20mmHg using the Mahan formula. The mPAP in these children significantly decreased after operation showing that chronic upper airway obstruction due to adenotonsillar hypertrophy resulted in higher mean pulmonary arterial pressure, which could be relieved by adenotonsillectomy.

In an Iranian study 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.²⁹ Moghaddam and

colleagues like his Turkish counterparts demonstrated reversibility of the pulmonary arterial pressures after adenotonsillectomy.^{29,37}

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. Tonsil pharyngeal ratio (TPR) on lateral neck radiography was shown to correlate with sPAP ($r = 0.624$; $P < .0001$). Children with TPR >0.66 were shown to be at greater risk for cardiac complications. A recommendation was made for these children to have supplementary studies with echocardiography or be given preference for surgery.⁴⁵

Table 1 summarizes the aforementioned relevant studies on pulmonary hypertension in children with adenoid and or adenotonsillar hypertrophy highlighting the study methods and results with regard to similarities and differences. These studies employed various definitions for pulmonary hypertension with regard to cut-off levels for pulmonary arterial parameters on echocardiography. In addition where mPAP was used in defining pulmonary hypertension, it was estimated using various equations such as the Mahan formula or the Chemla formula. These differences pose difficulties in comparing data head to head.

The prevalence of pulmonary hypertension in the above studies ranges from 7.3% to 51.9% in children with adenoid and adenotonsillar hypertrophy, meaning one in two to fifteen children with adenoid or adenotonsillar hypertrophy has pulmonary hypertension.^{29,44,45}

Table 1: Summary of relevant studies on pulmonary hypertension in children with adenoid and or tonsillar hypertrophy

STUDY	PATIENT CHARACTERISTICS	ECHO MACHINE	METHODS	RESULTS
Yilmaz³⁷ (2004) Turkey	52 children 36male (69%) Age:4-11 years UAO due to ATH	HP Sonos 5500System Andover Echocardiography	PH defined as:- mPAP >20mmHg mPAP: Mahan formula	PH prevalence 51.9% mPAP mean 23.13mmHg SD ± 7.68
Elmofly⁶⁵ (2005) Egypt	30 children 19male (63%) Age:4-10years UAO due to ATH	HP Sonos 2000System Echocardiography	PH defined as:- sPAP >30mmHg sPAP: $4(TRV)^2$	PH prevalence 26.7% sPAP mean 34.21 ± 6.7mmHg
Moghaddam²⁹ (2010) Iran	55 children 35 male (64%) Age: 4-14years ATH with OSA symptoms	Vivid 3 Npro/Expert transducer 2-5MHz (Norway)	PH defined as:- mPAP > 25mmHg mPAP: Mahan formula sPAP: $4(TRV)^2 + 1.23$	PH prevalence 7.3% mPAP mean 11mmHg sPAP mean 15.56mmHg,
Granzotto⁴⁵ (2010) Brazil	45 children Male 30 (66.7%) Age: 72.0 ± 32.3months Adenotonsillectomy indicated for SDB	Vivid 7 General Electric Healthcare (UK)	PH defined as :- sPAP>30mmHg sPAP: $4(TRV)^2$ mPAP: Chemla formula	PH prevalence 13% sPAP mean 25.89mmHg SD ± 3.36 mPAP mean 17.79mmHg SD ± 2.05
Abdel-Aziz³⁰ (2011) Egypt	80 children with SDB Male 46 (57.5%) Age: 2.5 to 7 years	Toshiba Sonolayer SSA- 270A cardiac ultrasound (Japan).	PH not defined PAP not determined	PH prevalence not quoted PAP mean 22.7mmHg SD ± 3.8
Tatlipinar⁵¹ (2012) Turkey	95 children AH: Male 50% Age 6.96years ±2.11 ATH: Male 50% Age 6.69years±1.68	Vivid 7 General Electric Healthcare (UK)	PH defined as:- mPAP>20mmHg mPAP: Mahan formula	PH prevalence not quoted <u>Adenoid hypertrophy:</u> mPAP mean 23.55mmHg SD ± 4.93 <u>Adenotonsillar hypertrophy:</u> mPAP mean 25.60mmHg SD ± 4.82

AH-adenoid hypertrophy, ATH-adenotonsillar hypertrophy, OSA-obstructive sleep apnea, PAP-pulmonary arterial pressure, PH-pulmonary hypertension, SDB-sleep disturbed breathing, TRV-tricuspid regurgitant velocity, UAO-upper airway obstruction.

Treatment of Pulmonary Hypertension in Adenotonsillar Hypertrophy

Prevention of pulmonary hypertension as a result of obstruction of the upper airways due to adenoid and or tonsillar hypertrophy relies on early identification and treatment of adenotonsillar hypertrophy.²⁹

In less severe cases, non-surgical interventions may be considered. Intranasal corticosteroids may reduce adenoid size and significantly improve nasal obstruction symptoms in children with mild to moderate adenoidal hypertrophy.^{28,47} In addition, leukotriene receptor antagonists such as montelukast have been shown to result in improvement of mild obstructive symptoms in children with adenoid hypertrophy.^{12,28,47}

Adenoidectomy with or without tonsillectomy is the treatment of choice for the prevention and treatment of pulmonary hypertension due to upper airway obstruction as a result of adenoid or adenotonsillar hypertrophy. This is the most common otorhinolaryngology surgery performed in our setting with minimal reported complications.^{16,48} Monset-Couchard and colleagues in 1975 noted that when adenotonsillar hypertrophy was the cause of pulmonary hypertension, adenotonsillectomy led to improvement in the clinical state, and reversible ECG changes.⁴⁹ Various studies have already attested to the success of adenotonsillectomy for the alleviation of symptoms and echocardiographic signs of adenotonsillar hypertrophy.³⁵⁻³⁷ Recurrence may occur after surgery in up to 20%.^{1,35} Resolution of residual obstructive symptoms after adenotonsillectomy may be promoted by a combination of intranasal steroids and oral montelukast.^{12,28,47} Once pulmonary hypertension is severe and irreversible, the only alternative for the patient is heart lung transplant. This intervention is not available in our resource limited setting.^{42,43,46}

STUDY JUSTIFICATION AND UTILITY

Adenotonsillar hypertrophy is a common condition in childhood, whose serious complications of pulmonary hypertension and cor pulmonale are common and lethal. The local prevalence of pulmonary hypertension among children with upper airway obstruction is unknown.

This study aims to determine the burden of pulmonary hypertension among children with radiologically confirmed upper airway obstruction, attending KNH ENT clinic or paediatric wards. The findings of this study will lay down the foundation for further research.

RESEARCH QUESTION

What is the prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at the Kenyatta National Hospital (KNH)?

STUDY OBJECTIVES

Primary Objective

- To determine the prevalence of pulmonary hypertension of children with adenoid or adenotonsillar hypertrophy at KNH.

Secondary Objective

- To determine the clinical-radiological factors associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH.

MATERIALS AND METHODS

Study Area, Design, Sample Size and Population

STUDY AREA

This study was carried out within the ENT department and the pediatric wards of KNH. KNH serves as a National referral tertiary facility and teaching hospital for the college of health sciences, University of Nairobi.

The clinical areas of the ENT department comprise a filter clinic, consultant clinic and ward. Patients with ENT problems are referred to the ENT filter clinic first where they are screened and if appropriate referred to the ENT consultant clinic. The ENT filter clinic draws its clientele from various clinics and wards in KNH as well as from the district, provincial or other private hospitals or clinics within the country. Annually there are over 6000 clinical contacts made with children with adenoid and or tonsillar hypertrophy at the ENT filter clinic making this an optimum catchment area with regard to diversity.

Of the 2000 pediatric admissions 25% were attributed to chronic conditions of tonsils and adenoids. It is in this regard that children were also sampled from the pediatric wards.

STUDY DESIGN

Hospital based cross sectional descriptive study

SAMPLE SIZE

The sample size (N) was calculated using Fischer's formula

$$N = \frac{z_{\alpha}^2 p(1 - P)}{d^2}$$

Z_{α} was the standardized normal deviate corresponding to a significant level α . the level significance α was taken as 0.05 giving $Z = 1.96$

P was the expected proportion of echo abnormalities in children with adenotonsillar hypertrophy.

d was the precision of the estimated values in the study: d was estimated at ± 0.06 .

The expected proportion of echo abnormalities was estimated at 13 % as per Granzotto et al's study carried out on tonsil size as a predictor of cardiac complications in children with sleep disordered breathing.⁴⁵

$$N = 121$$

INCLUSION CRITERIA

Children aged 0-12 years with clinician diagnosed adenoid hypertrophy with or without tonsillar hypertrophy as the only cause of upper airway obstruction, confirmed on lateral neck radiography.

EXCLUSION CRITERIA

All children with:-

1. Neurological abnormalities. (E.g. Cerebral palsy)
2. Genetic syndromes with craniofacial abnormalities. (E.g. Down syndrome)
3. Other causes of airway obstruction (deviated septum, nasal polyposis, gross turbinate hypertrophy)
4. Known cardiac disease or cardiac disease discovered on echocardiography/ chronic lung disease/ human immunodeficiency virus (HIV)/ sickle cell anemia..
5. Body mass index (BMI) > 95th percentile for the age.
6. Refusal to grant written informed consent.
7. Echocardiography not performed.

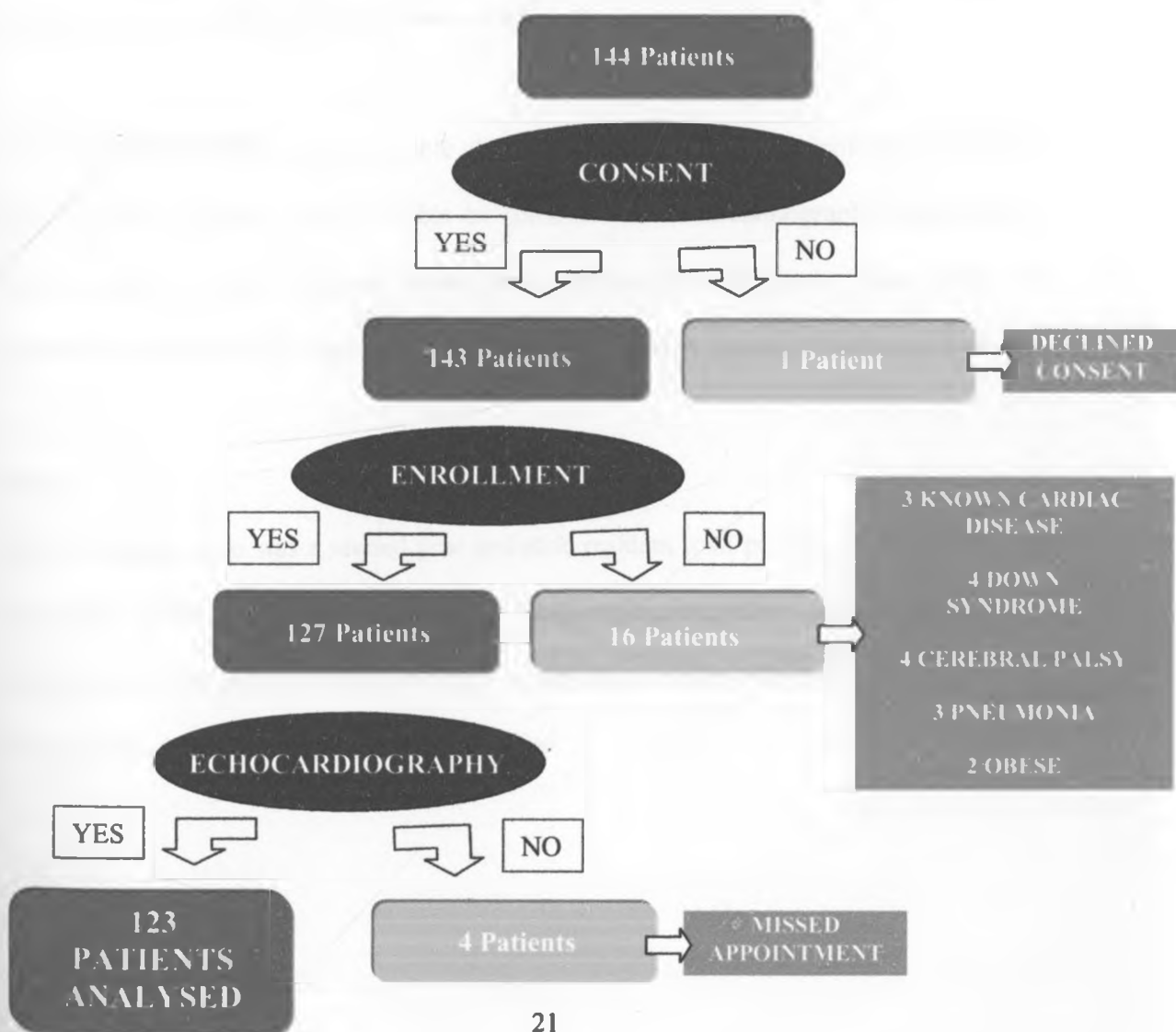
Sampling Method

All children who satisfied the inclusion criteria and had no exclusion criteria were enrolled into the study through consecutive sampling.

Procedures

During the study period of September to December 2011, a total of 144 caregivers of children aged 0-12 years with clinician diagnosed adenoid and or tonsillar hypertrophy confirmed on lateral neck radiography, were approached and requested to participate in the study. Ethical approval was granted by the Kenyatta National Hospital Ethics and Research Committee. (Appendix I)

Figure 4: Flow chart depicting study participant enrollment



A written informed consent was obtained. (Appendix II & III) Exclusion criteria were validated during history taking and physical exam using a checklist. (Appendix IV) The clinical data was recorded on the questionnaire and proforma tool which was adapted from Goldstein.¹⁸ (Appendix V)

Figure 4 depicts a flow chart of the study participant enrollment process. One caregiver declined consent, 3 children had known cardiac disease, 4 had cerebral palsy, 4 had Down syndrome, 3 had active pneumonia, and 2 were obese, hence were excluded. No child was known to have HIV or sickle cell anemia. On ENT exam, no child had a deviated septum, nasal polyposis or gross turbinate hypertrophy.

One hundred and twenty seven eligible children were consecutively enrolled in the study. Four of these children were not able to come for the echocardiography appointment, reasons given by their caregivers being prior scheduled commitments. There were 123 children who underwent Doppler echocardiography and were included into the analysis.

History

The investigator who was a second year pediatric resident took pertinent history from the caregivers of the children recruited in the study on an individual basis. This included demographic data, history of day and night symptoms duration of symptoms, use of nasal steroids and whether surgery had been scheduled.

Physical Examination Findings

The physical examination entailed a general exam, an ENT and cardiovascular evaluation. This was conducted by the principal investigator for each child recruited in the study on an individual basis.

On physical examination, weight was taken using a calibrated digital salter scale model 9010 and measured to one decimal point in kilograms. Height was taken using a stadiometer assisted by the caregiver.(Appendix VI) Body mass index was calculated using the sex specific Centre for Disease Control (CDC) charts.⁷¹

The ENT examination involved assessment of presence of mouth breathing and grading of tonsil size using the Brodsky classification as is shown in figure 1.

The cardiovascular exam included taking oxygen saturation through pulse oximetry, evaluating the respiratory rate and heart rate, assessment for a palpable p2, murmur, jugular venous distension, edema and hepatomegaly. Pulse oximetry and pulse rate was taken using a digital hand held pulse oximeter model MD300C41 by Beijing Choice Electronic Tech. Ltd. Tachypnea was defined as a respiratory rate above cut-off for age as derived from WHO cut-offs for respiratory tract infections.⁵⁰ Tachycardia was defined as an elevated heart rate as measured while the patient is calm above the upper limit of normal range.⁷⁰

Radiologic findings

During the study period, all children who required a lateral neck radiograph as part of their routine workup had it done at the patients cost. Special stickers were availed to the various participating departments to be put on their lateral neck radiograph request forms for standardization. Lateral neck radiography was performed in the KNH radiology department using a standardized technique. The lateral post nasal soft tissue radiograph was taken by the same qualified pediatric technologist with the patient in supine position using a horizontal beam. The focus film distance was 100 centimetres. Direct exposure was employed with 57 - 60 peak kilovoltage (kVp) and 4.5 - 5.0 milliampere second (mAs), at 100 cm. using fine focus. The pediatric x-ray room-one equipment was exclusively used for the study for standardization of the exposure factors and technique. The images were recorded on a radiographic film.

Image quality was assessed with regard to rotation, magnification and open mouth and this was documented in the data collection proforma. Lateral neck radiograph findings (cephalometry) were interpreted by an independent pediatric radiologist assisted by the principal investigator. The pediatric radiologist was blinded to the severity of the patients' disease with regard to history and physical examination findings. Adenoid nasopharyngeal ratio (ANR) and tonsil pharyngeal ratio (TPR) and were measured in centimeters to the nearest decimal point with a ruler using a standardized technique proposed by Shintani as shown in Figure 2.⁵⁶

Echocardiography

Echocardiography was performed by the pediatric cardiologist assisted by the principal investigator. The cardiologist was blinded to the severity of the patients' disease with regard to history and physical examination findings.

Transthoracic echocardiography was performed on all patients with the use of a portable VIVID I Echo Color Ultrasound System echocardiography machine. Cardiac measurements were performed according to the guidelines of the American Society of Echocardiography.⁶⁰ M-mode, 2D echo and Doppler echocardiography were employed as shown in Figure 3. Systolic pulmonary artery pressure (sPAP) was measured using the modified Bernoulli equation applied using the tricuspid regurgitation jet.^{42,43} Mean pulmonary artery pressure (mPAP) was then derived using the Chemla equation = $(0.61 * sPAP) + 2\text{mmHg}$.⁶¹ Pulmonary hypertension was defined as an estimated mPAP of $>25\text{mmHg}$.^{42,43,60,61}

Any other additional findings were also recorded. Digital images and videos were stored for future validation. Majority of the young infants were pacified with breastfeeding by the mother or distracted with toys. Sedation with chloral hydrate at a dose of 25mg/kg/dose to 75mg/kg/dose was administered when needed.

Study Outcomes

The study outcomes assessed included:

- Outcome variable
 - Pulmonary hypertension – sPAP, mPAP: presence or absence of pulmonary hypertension (mPAP >25mmHg)
- Associated factors
 - Demographics – age, sex
 - History – night time symptoms and day time symptoms, duration of symptoms, use of nasal steroids, whether surgery has been scheduled
 - Physical exam – weight for height z score, mouth breathing, tonsil size, oxygen saturation, respiratory rate, tachypnea, heart rate, tachycardia, palpable P2, murmur, jugular venous pressure, oedema, hepatomegaly
 - Radiological parameters – adenoid size, adenoid nasopharyngeal ratio, tonsil size, tonsil pharyngeal ratio

The ENT team independently carried out appropriate patient management.

Ethical Considerations

1. Permission: Permission to undertake this study was sought from Kenyatta National Hospital Scientific and Ethics Committee. A letter of protocol approval was obtained prior to the commencement of the study, as well as approval for other study documents subject to review by the Ethics, Research and Standards Committee.

2. Risks: No experimental investigations or treatments were employed in this study.

3. Benefits: The study participants had echocardiographic evaluation by the investigator and significant findings were communicated to the ENT & pediatric team for the appropriate adjustment of the patient's management. Prioritization for adenoidectomy and or tonsillectomy was given for children found to have pulmonary hypertension.

4. Confidentiality: Subject confidentiality was strictly held in trust by the investigator. The study protocol, documentation, data and all other information generated were held in strict confidence. No information concerning the study or the data was released to any unauthorized third party. All evaluation forms, reports and other records that left the site were identified only by the Subject Identification Number (SIN) to maintain subject confidentiality. Clinical information was not be released without written permission of the subject, except as necessary for monitoring by Ethics, Research and Standards Committee.

5. Informed consent: Informed consent was obtained from the caregivers after explaining to them the objective of the study. The consent form described the purpose of the study and the procedure to be followed. The investigator conducted the consent discussion and checked that the parent/caregiver comprehended the information provided and answered any question about the study. Consent was voluntary and free from coercion. A copy of the consent form was given to the parent or caregiver and that she/he had consented to the study was documented in the record.

DATA MANAGEMENT AND ANALYSIS

Data collection was confidential using a structured questionnaire and proforma tool. Filled questionnaires were solely utilized for this study and subsequently stored safely at the end of the study after entering the data in a Microsoft Access 2007 database. Data analysis was performed using Statistical Package for Social Sciences (SPSS) version 17.0.

Continuous variables e.g. age, height, weight were summarized using means and standard deviations for normally distributed data e.g. age, weight; or medians and associated interquartile ranges for skewed data e.g. duration of symptoms, tonsil size. Categorical variables e.g. sex, presence or absence of pulmonary hypertension were summarized using proportions with associated 95% confidence intervals.

Chi square test of association was used to analyze most of the data in table 2 as these was a comparison of categorical variables e.g. pulmonary hypertension and age. Odds ratio was reported to give an estimate of risk. Student's t-test was employed when means were compared in children with and without pulmonary hypertension e.g. comparing the mean age in children with pulmonary hypertension versus the mean age in children without pulmonary hypertension. Mann Whitney U test was used to compare medians between the children with pulmonary hypertension and those without pulmonary hypertension, as this was skewed data e.g. the median duration of symptoms in children with and without pulmonary hypertension. All statistical tests were at 5% significance level.

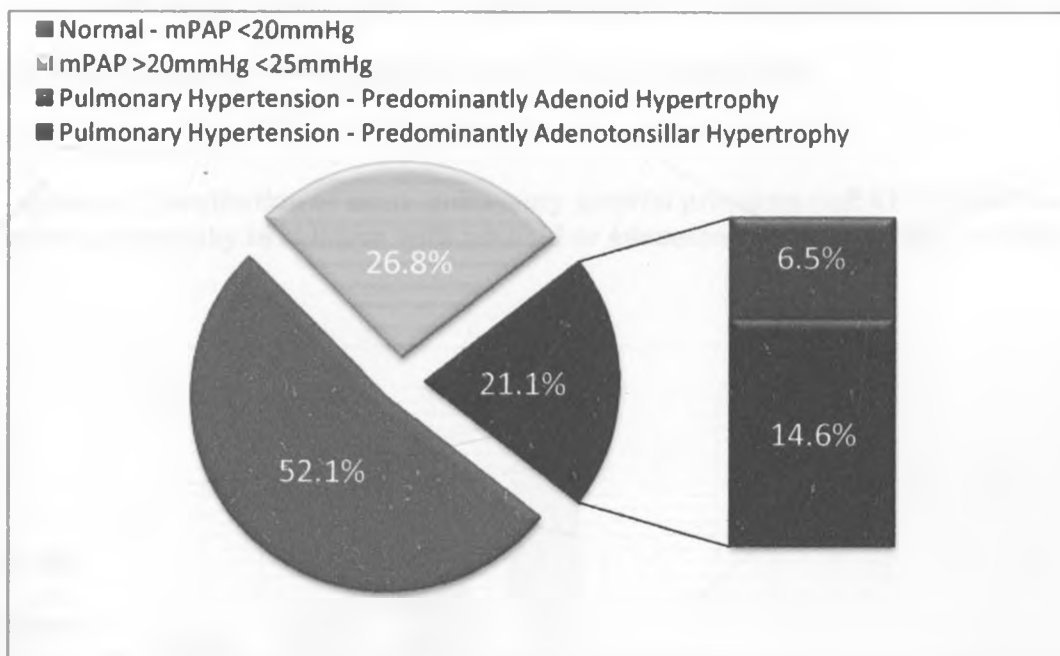
A logistic regression analysis was conducted with the main outcome included as a binary variable generated by categorizing the mean pulmonary arterial pressures taken during the study into children with pulmonary hypertension and children without pulmonary hypertension. The outcome was regressed on age and sex which were selected before the analysis as potential confounders of the relationship between the prevalence of pulmonary hypertension and patient factors. The factors that were significantly associated with pulmonary hypertension in the unadjusted bivariate analyses were then included in the logistic regression model, based on a cutoff level of 0.05. These variables included use of nasal steroids, nasal obstruction, hyperactivity, presence of palpable P2 and hepatomegaly. ANR and TPR were also included. The model was used to obtain adjusted odds ratio along with 95% confidence intervals. P-values calculated in the model were based on the Wald test. With the addition of each new variable to the model, likelihood ratio tests were used to compare the different models to determine the contribution of each factor to the model fit.

RESULTS

Prevalence of pulmonary hypertension

Out of all the 123 participants, a total of 26 children with adenoid or adenotonsillar hypertrophy had pulmonary hypertension giving an overall prevalence of 21.1% (95% CI 14.3-29.4) as depicted in Figure 5.

Figure 7: Prevalence of pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH



Eight out of the 26 (30.8%) children with pulmonary hypertension had predominantly adenoid hypertrophy translating to a prevalence of 6.5% (95%CI 2.9–12.4) in this group of children. Eighteen of the 26 (69.2%) children with pulmonary hypertension had

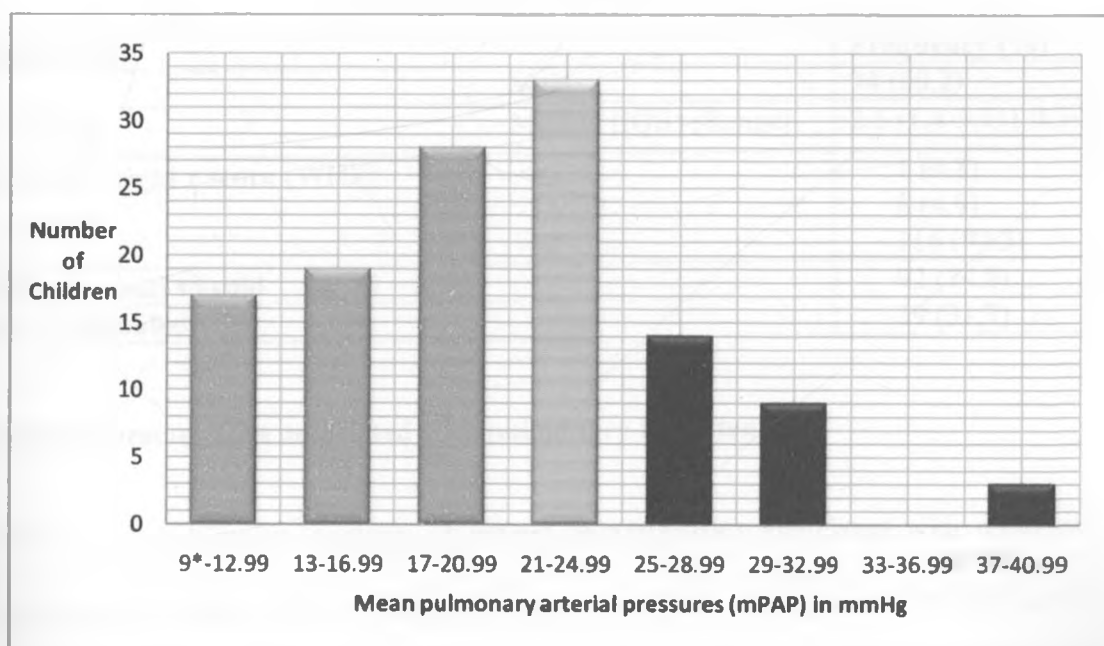
predominantly adenotonsillar hypertrophy giving a prevalence of 14.6% (95%CI 8.9 - 22.1) as illustrated in Figure 5.

As shown in Figure 5, thirty three children out of 123(26.8%) had mPAP >21mmHg but < 25mmHg. Sixty four children out of 123 (52.1%) had normal mPAP < 20mmHg.

Distribution of mean pulmonary arterial pressures

Figure 6 depicts a bar graph of the distribution of mean pulmonary arterial pressures for all the one hundred and twenty three children analysed. The distribution of children with pulmonary hypertension with various pressure ranges is highlighted.

Figure 6: Distribution of mean pulmonary arterial pressures (mPAP) derived on echocardiography in children with adenoid or adenotonsillar hypertrophy at KNH



PATIENT CHARACTERISTICS

We had a total of 123 children presenting with adenoid or adenotonsillar hypertrophy, 74 (60%) males and 59 (26%) females, giving a male to female ratio of 1.5: 1. The median age of the study population was 2.5 years, the youngest patient in the study was 3.6 months and the oldest child was 8.5 years old. Majority of the participants (94.4%) were well nourished with weight for height Z-scores > -2 . Six (4.8%) children were moderately malnourished and one (0.8%) was severely malnourished.

Ninety two children out of 123 (74.8%) were on intranasal steroids, sixty seven (72.8%) of whom were using intranasal steroids intermittently as opposed to continuously. Thirty nine out of 123 (31.7%) were scheduled for surgery. Most of the patients 115 out of 123 (93.4%) were sampled from the ENT filter clinic.

Table 2: Patient characteristics in children with adenoid or adenotonsillar hypertrophy at KNH (N = 123)

Characteristic		Frequency (%)
Sex	Male	74 (60.2)
Age in years	Median (IQR) [Range]	2.5 (1.4-3.5) [0.3-8.5]
Weight for height z score (WHZ)	<-3	1 (0.8)
	-3 to -2	6 (4.9)
	>-2	116 (94.3)
Use of intranasal steroid		92 (74.8)
Surgery scheduled		39 (31.7)

Patient characteristics associated with pulmonary hypertension

Table 4 shows bivariate analysis of patient characteristics associated with pulmonary hypertension. Children with pulmonary hypertension were younger (2.30 ± 1.71 yrs) than those without pulmonary hypertension (2.91 ± 1.73 yrs) but the difference was not

significant ($p = 0.11$). The odds of pulmonary hypertension was approximately 2.7-fold greater among male children compared to female children, $OR=2.7(0.92-8.74)$, $p=0.049$. Children whose WHZ score was $<-2SD$ had a 3-fold increased risk of having pulmonary hypertension compared to those whose WHZ score was $\geq -2SD$, however this was of no statistical significance $OR=3.0(0.41-19.1)$, $p=0.16$. Children who were not on intranasal steroids had a significant 3-fold increased risk of having pulmonary hypertension than those children who were on intranasal steroids $OR=2.93(1.1-8.2)$, $p=0.02$.

Table 3: Patient characteristics associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH (N = 123)

Patient Characteristics	Pulmonary hypertension N=26 (%)	Normal pulmonary pressure N= 97 (%)	OR (95% CI)	P value
Mean age in years* (SD)	2.30(1.71)	2.91(1.73)	-	0.11
Sex (male)	20(76.9)	54(55.7)	2.70(0.92-8.74)	0.049
WHZ score ($<-2SD$)	3(42.9)	4(57.1)	3.0(0.41-19.1)	0.16
Intranasal steroid use (no)	17(65.4)	38(39.2)	2.93(1.1-8.2)	0.02
Scheduled surgery	6(23.8)	33(34)	0.58(0.278)	0.278

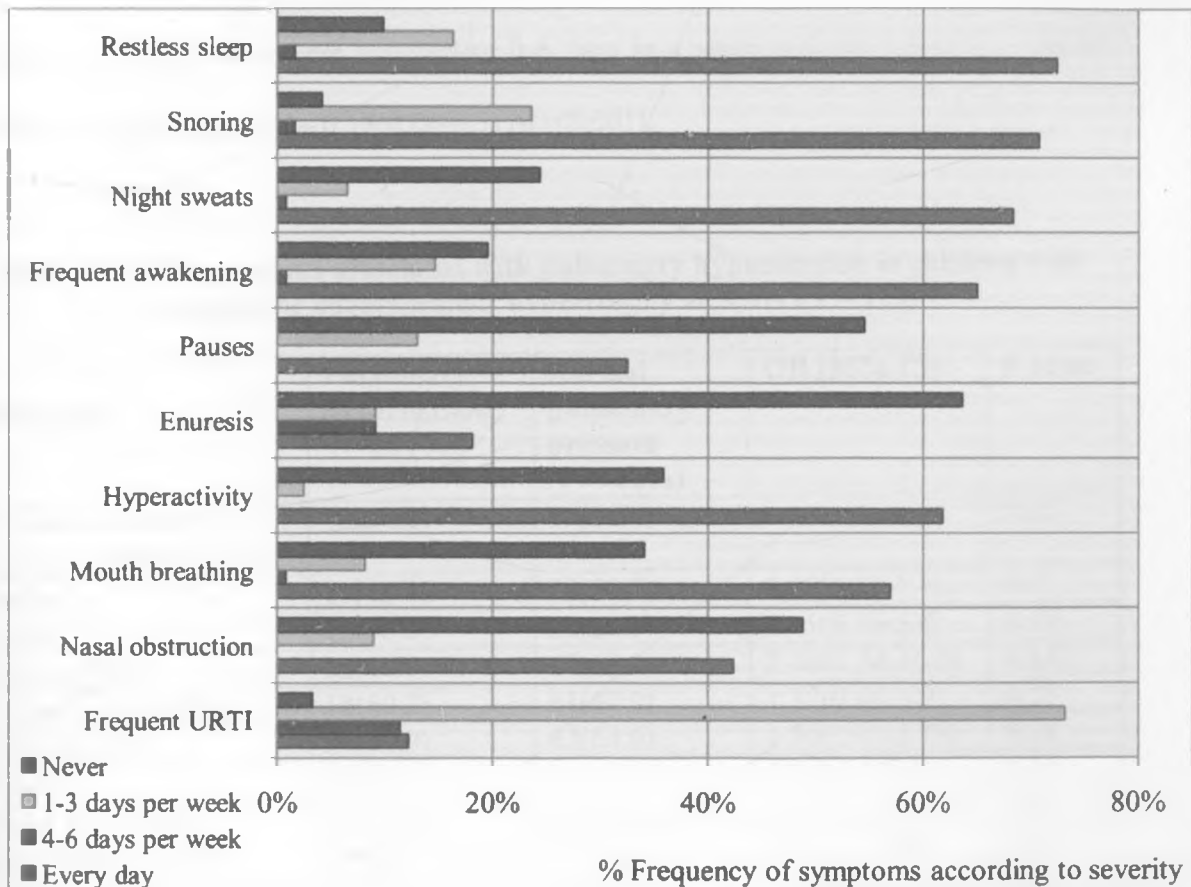
* Student t-test

SYMPTOMS

The children were classified according to the severity of the symptoms never, 1-3 times per week, 4-6 days per week and every day. Ten symptoms were evaluated. Day symptoms included nasal obstruction, mouth breathing, frequent upper respiratory tract infections (URTI) and hyperactivity. Night symptoms included night sweats, frequent awakening, restless sleep, pauses, snoring and enuresis. Enuresis was only evaluated in children above the age of 4 years. Only 22 children of the 123 analysed were above the age of 4 years. The

majority of children reported daily occurrence of 6 of the 10 symptoms that were evaluated. Eighty nine (72.3%) reported daily restless sleep, 87 (71.3%) snoring, 84 (68.9%) night sweats, 80 (65.6%) frequent awakening, 76 (61.8%) hyperactivity and 70 (56.9%) mouth breathing. Figure 7 depicts the frequency of symptoms according to severity experienced in the children in our study.

Figure 7: Frequency of day and night symptoms in children with adenoid or adenotonsillar hypertrophy at KNH



Daily symptoms associated with pulmonary hypertension

In bivariate analysis, children with pulmonary hypertension had a shorter duration of symptoms than the children without, however, this difference was not statistically significant ($p = 0.117$). Children with a history of nasal obstruction every day had a significant almost 3-fold increased risk of pulmonary hypertension compared to the children who experienced nasal obstruction 0-6 days/week OR= 2.7(1.0-7.4) [$p=0.025$]. There was no significant difference observed with regard to history of snoring, pauses, mouth breathing, restless sleep and frequent awakening when children with and without PH were compared. Children who were hyperactive daily had a 22% reduced risk of PH compared to those who were hyperactive 0-6 days in a week and this difference was of statistical significance OR= 0.18(0.06-0.5) [$p<0.001$].

Table 4: Daily symptoms associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH (N = 123)

Symptom	Pulmonary hypertension N=26 (%)	Normal pulmonary pressure N= 97 (%)	OR (95% CI)	P value
Symptom duration in months** Median (IQR)	10(5-20)	15(8-24)	-	0.117
Snoring	11(42.3)	58(59.8)	0.49(0.18-1.3)	0.11
Pauses	12(46.2)	28(28.9)	2.11(0.78-5.6)	0.10
Restless Sleep	22(84.6)	67(69.1)	2.46(0.74-10.6)	0.143
Frequent Awakening	18(69.2)	61(62.9)	1.33(0.49-3.9)	0.55
Night Sweats	20(76.9)	63(64.6)	1.80(0.61-5.98)	0.28
Enuresis	2(7.7)	3(3.1)	4.0(0.02-0.5)	0.19
Mouth Breathing	19(73.1)	51(52.6)	2.45(0.88-7.45)	0.061
Nasal Obstruction	16(61.5)	36(37.1)	2.7(1.0-7.40)	0.025
Frequent URTI	4(15.4)	11(11.3)	1.42(0.3-5.4)	0.58
Hyperactivity	8(30.8)	69(71.1)	0.18(0.06-0.5)	<0.001

** Mann Whitney U test

As shown in Table 5, clinical findings with the highest sensitivity for pulmonary hypertension were history of frequent URTIs (96%), restless sleep (92%), snoring (92.3%), mouth breathing (88.5%) or night sweats (84.6%). A history of enuresis (94%), clinically present edema (99%) or a palpable P2 (91%) had the highest specificity. Clinical findings with the highest negative predictive value (NPV) included mouth breathing (90%), nasal obstruction (88%), night sweats (88%), pauses (87%) and restless sleep (83%). All the clinical factors analysed had a poor positive predictive value (PPV).

Table 5: Sensitivity, specificity, PPV and NPV of clinical symptoms and signs for pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy

	Sensitivity	Specificity	PPV	NPV	Area under ROC
Snoring	92.3	3.1	20.3	60	.48
Pauses	65.4	59.8	30.4	86.6	.63
Restless Sleep	92.3	10.3	21.6	83.3	.51
Frequent Awakening	76.9	19.6	20.4	76	.48
Night Sweats	84.6	27.8	23.9	87.1	.56
Mouth Breathing	88.5	28.1	25	90	.58
Nasal Obstruction	73.1	54.6	30.2	88.3	.64
Frequent URTI	96.2	3.1	21	75	.50
Hyperactivity	34.6	26.8	11.3	60.5	.31
Enuresis	15.4	93.8	40	80.4	.55
Edema	11.5	99	75	80.7	.55
Palpable P2	34.6	90.7	50	83.8	.63

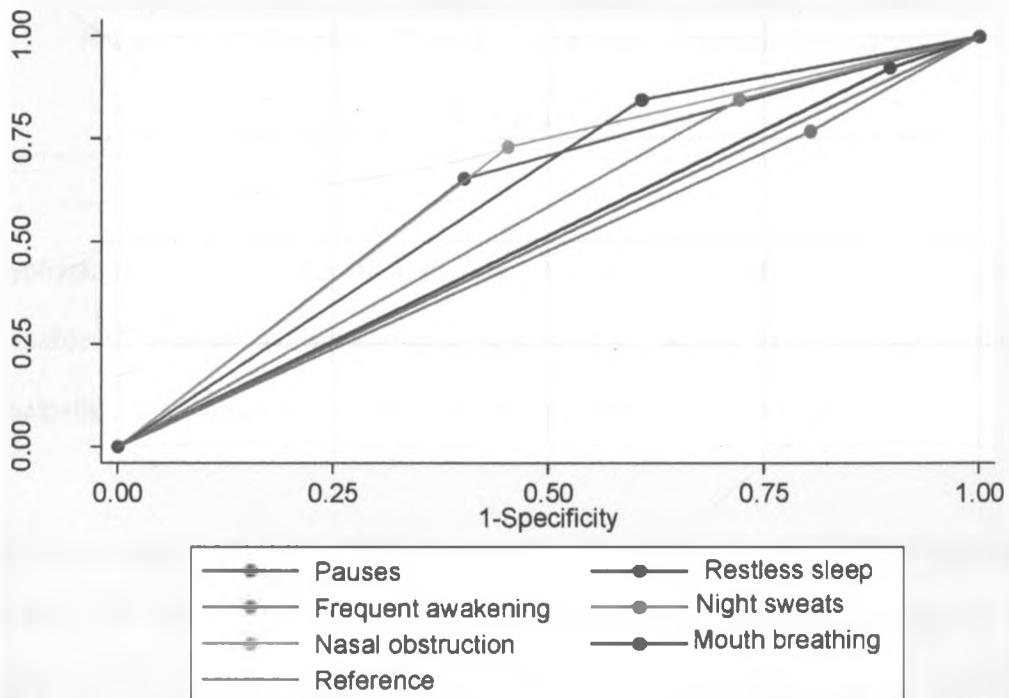
Table 6 shows the clinical symptoms which when combined serve as predictors with the highest sensitivity, specificity, positive and negative predictive value. Presence of mouth breathing or restless sleep on history had the highest sensitivity (88.5%) and negative predictive value (86.4%) when predictors were combined. No combination gave a suitable specificity and positive predictive value.

Table 6: Combination of predictors with the highest sensitivity, specificity, positive and negative predictive value

	Sensitivity	Specificity	PPV	NPV	Area ROC
Mouth breathing or restless sleep	88.5	19.6	22.8	86.4	.54
Mouth breathing or frequent awakening	84.6	25.2	18.6	20	.43
Snoring or frequent awakening	80.8	30.8	17.9	16.7	.41

Figure 8 depicts the receiver operating curves for these clinical features. Mouth breathing and restless sleep were the most sensitive symptoms with the AUC of 0.58 and 0.51 respectively.

Figure 8: Receiver operating curves for different daily symptoms experienced in children with adenoid or adenotonsillar hypertrophy at KNH

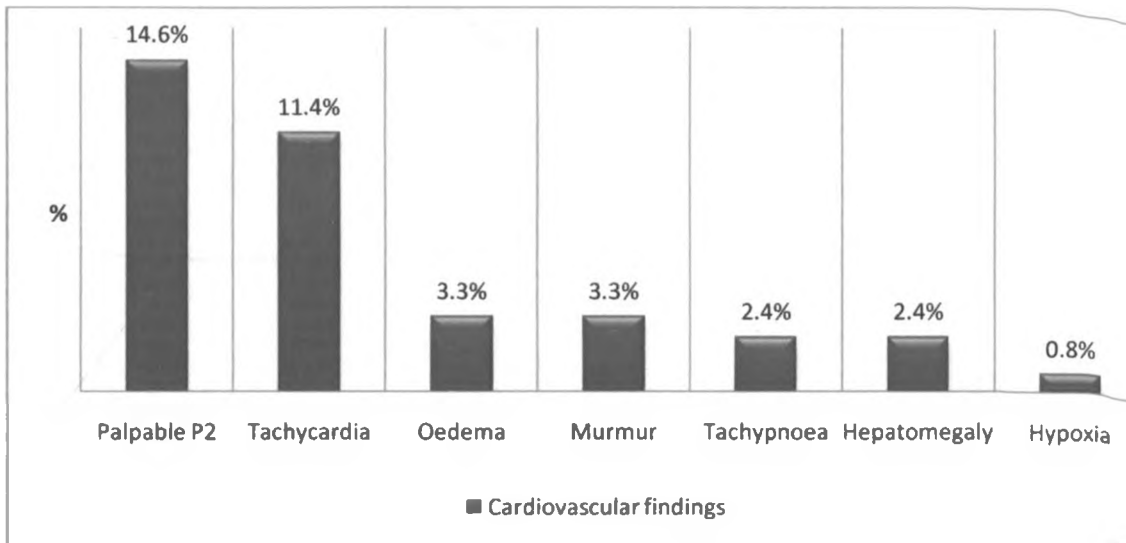


PHYSICAL EXAMINATION

CARDIOVASCULAR EVALUATION

The frequency of the cardiovascular findings among the children presenting with adenoid or adenotonsillar hypertrophy are shown in Figure 9.

Figure 9: Frequency of cardiovascular findings in children with adenoid or adenotonsillar hypertrophy at KNH (N = 123)



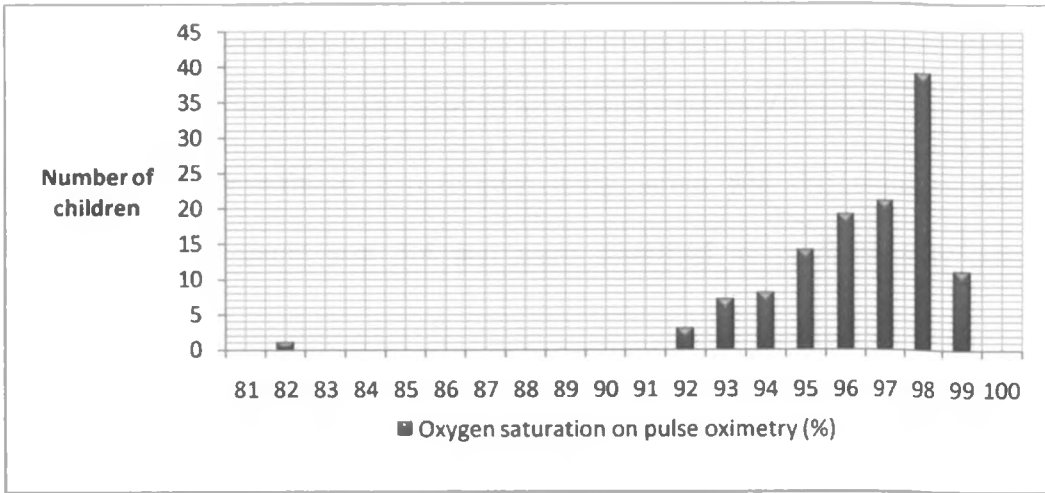
Hypoxia, tachypnoea, tachycardia, presence of oedema, elevated jugular venous pressure; palpable P2, murmur and hepatomegaly were sought. The two most common findings were a palpable P2 in 18 (14.6%) children and tachycardia in 14 out of 123 (11.4%).

Oedema was reported in 4 (3.3%), murmur 4 (3.3%), tachypnoea 3 (2.4%), hepatomegaly 3 (2.4%), and hypoxia 1 (0.8%). Two of the four murmurs heard were loudest in the left upper and lower sternal border, while the other two were loudest only in the lower left

sternal border. The murmurs were functional when evaluated on echocardiography later on. No patient had jugular venous distension.

Figure 10 illustrates the distribution of oxygen saturations among the children analysed. Only one child had hypoxia of 82% on room air giving a skewed distribution.

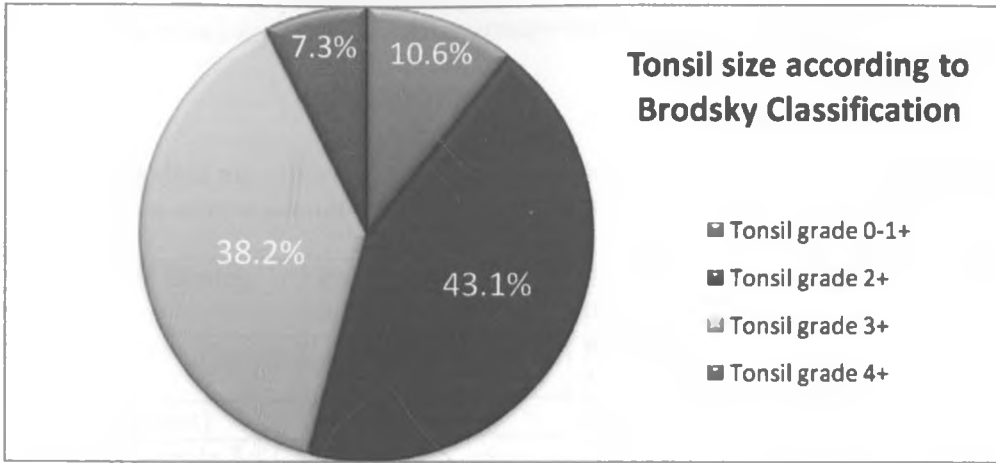
Figure 10: Distribution of oxygen saturations on pulse oximetry in children with adenoid or adenotonsillar hypertrophy at KNH (N = 123)



ENT EVALUATION

On ENT evaluation, mouth breathing was reported in 92 (74.8%) of the children. Tonsil size was graded using the Brodsky scale. Majority of the children 100 (81.3%) had tonsil size grade 2 or 3 on the Brodsky scale. Grade 2 tonsils were reported in 53 (43.1%), grade 3 tonsils in 47 (38.2%) and grade 0-1 tonsils in 13 (10.6%). Only nine (7.3%) children had tonsils assessed to be grade 4 as shown in Figure 11.

Figure 11: Tonsil size on ENT examination findings in children with adenoid or adenotonsillar hypertrophy at KNH (N=123)



Sixty six (53.7%) children with adenoid hypertrophy had tonsil size 0-2 and were classified as predominantly adenoid hypertrophy. Fifty seven (46.3%) children with adenoid hypertrophy had tonsil size 3-4 and were classified as predominantly adenotonsillar hypertrophy.

Physical examination findings associated with pulmonary hypertension

Table 7 shows the physical examination findings associated with pulmonary hypertension in these children. In bivariate analysis, a palpable P2 on physical exam had a significant – fold increased risk of having pulmonary hypertension OR = 5.18(1.54-17.0) [p=0.003]. Hepatomegaly on examination was significantly associated with pulmonary hypertension, with all children manifesting with hepatomegaly having pulmonary hypertension (p=0.009). Mouth breathing and Brodsky scale tonsil grade 3-4 on physical exam were not significantly associated with pulmonary hypertension. Oxygen saturations were lower in children with pulmonary hypertension at a mean of 95.2% (SD ±3.3). Children with

pulmonary hypertension were more likely to have tachypnoea and less likely to have murmurs compared to children without pulmonary hypertension; however, the differences were not of statistical significance. All murmurs heard were functional.

Table 7: Physical examination findings associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH (N = 123)

Physical examination finding	Pulmonary hypertension N=26 (%)	Normal pulmonary pressure N= 97 (%)	OR (95% CI)	P value
Mouth Breathing [Present]	23(88.5)	69(71.1)	0.33(0.22-52.4)	0.081
Tonsil Size [Brodsky grade 3-4]	18(69.2)	48(49.5)	2.25(0.83-6.54)	0.12
Oxygen saturation %** Median (IQR)	96.0(94-98)	97(96-98)	-	0.02
Tachypnea	2(7.7)	1(1.0)	8.0(0.39-476.7)	0.11
Tachycardia	2(7.7)	12(12.4)	0.59(0.06-2.95)	0.73
Murmur	1(3.8)	3(3.1)	1.24(0.02-16.2)	0.99
Palpable P2	9(34.6)	9(9.3)	5.18(1.54-17.0)	0.003
Hepatomegaly	3(11.5)	0	-	0.009
Edema	3(11.5)	1(1.0)	12.5(0.93-663.8)	0.03

** Mann Whitney U test

LATERAL NECK RADIOGRAPH FINDINGS

Data on image quality is presented in Table 8. Thirty eight out of 123 (31%) were poor quality radiographs and only 19 out of 123 (15%) were good quality images. No child with pulmonary hypertension had a good quality lateral neck radiograph and 15 out of 26 (57.7%) children with pulmonary hypertension had poor images. Most of the lateral radiographs analysed, 87 out of 123 (70.7%) were rotated.

Table 8: Image quality of the lateral neck radiographs of children with adenoid or adenotonsillar hypertrophy (N=123)

Image quality parameters	Frequency [n=123] (%)	Pulmonary Hypertension [n= 26] (%)
Open mouth	61 (49.6)	18 (69.2)
Rotation	87 (70.7)	20 (76.9)
Magnification	59 (48.0)	19 (73)
Poor quality	38 (31.0)	15 (57.7)
Good quality	19 (14.6)	0

Poor quality – open mouth, rotation and magnification

Good quality – closed mouth, no rotation and no magnification

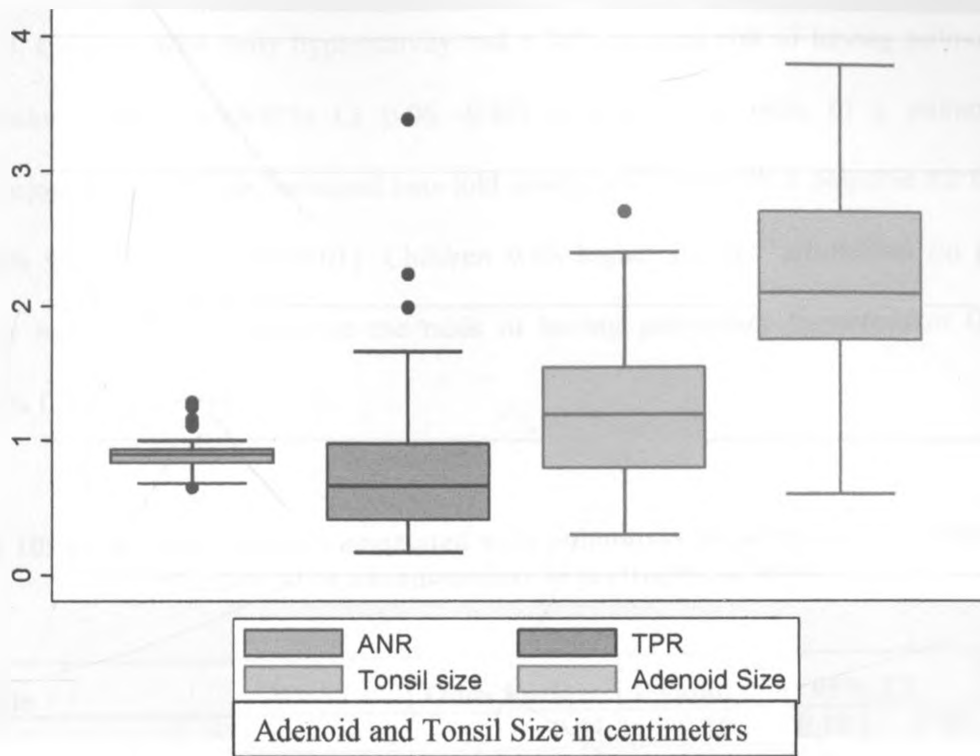
Linear and ratio measurements performed on the lateral neck radiographs included adenoid size, tonsil size, ANR and TPR. All the children had an ANR > 0.63. Thirty six out of 123 (29.3%) had TPR > 0.66. Of these 36 children, 7 (19.4%) with TPR >0.66 had pulmonary hypertension as shown in Table 9.

Table 8: Proportion of the children in the study with ANR>0.63 or TPR > 0.66

ANR and TPR	N	Percent
The proportion of children in the study with ANR >0.63	123	100
The proportion of children in the study with TPR >0.66	36	29.3
Proportion of children with TPR > 0.66 with pulmonary hypertension	7/36	19.4

The adenoid size ranged from 0.6cm to 3.8cm with a mean of 2.2cm (SD ±0.6). The adenoid nasopharyngeal ratio (ANR) ranged from 0.7 to 1.3 with a mean of 0.9 (SD ±0.1). The tonsil size ranged from 0.0cm to 2.7cm with a median of 0.5cm and an interquartile range of 0.0cm to 1.4cm. The tonsil pharyngeal ratio (TPR) ranged from 0 to 3.4 with a median of 0.4 and an interquartile range of 0 to 0.8 as illustrated in Figure 12.

Figure 12: Box plot depicting lateral neck radiography findings in children with adenoid or adenotonsillar hypertrophy at KNH (N=123)



Lateral neck radiograph findings associated with pulmonary hypertension

In bivariate analysis, adenoid size and the adenoid nasopharyngeal ratio were greater in the children with pulmonary hypertension than in those without pulmonary hypertension. Tonsil size and the tonsil pharyngeal ratio were significantly smaller in the children with pulmonary hypertension than among those without pulmonary hypertension. These differences were however of no statistical significance.

Independent clinical-radiological factors associated with pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy at KNH

The independent risk factors identified using logistic regression analysis, are shown in Table 10. Children with daily hyperactivity had a 78% reduced risk of having pulmonary hypertension. OR = 0.22(95% CI 0.06 -0.87) [p=0.03]. The odds of a pulmonary hypertension diagnosis was increased two-fold among children with a palpable P2 OR = 2.01(95% CI 0.02 -0.58) [p=0.01]. Children with higher oxygen saturations on pulse oximetry had a 28% reduction in the odds of having pulmonary hypertension OR = 0.72(95% CI 0.54 -0.97) [p=0.03].

Table 10: Independent factors associated with pulmonary hypertension of children with adenoid or adenotonsillar hypertrophy at KNH

Variable		Odds Ratio	P value	95% CI	
Female		0.44	0.29	0.10	1.97
Age in years		1.43	0.16	0.87	2.37
Intranasal steroids		2.29	0.20	0.64	8.22
Tonsil size (Brotsky scale)	2	0.29	0.38	0.02	4.44
	3	0.78	0.86	0.05	13.38
	4	1.49	0.81	0.06	36.18
Pauses	1-3 nights/week	2.93	0.17	0.63	13.62
	Every night	2.01	0.31	0.52	7.85
Disturbed sleep		1.21	0.76	0.35	4.26
Hyperactivity	1-3 days/week	0.21	0.39	0.01	7.39
	Every day	0.22	0.03	0.06	0.87
Respiratory rate		1.05	0.34	0.95	1.18
Oxygen saturation		0.72	0.03	0.54	0.97
Palpable P2		2.01	0.01	0.02	0.58
Adenoid size (cm)		1.31	0.57	0.51	3.36

DISCUSSION

The prevalence of pulmonary hypertension among children aged 0.3 to 8.5 years with adenoid hypertrophy diagnosed radiologically at the KNH ENT clinic from September 2011 to December 2011 was 21.1% (95% CI 14.3 – 29.4%). This study was carried out at high altitude (~1680m above sea level). The researcher did not find any other studies conducted in Sub Saharan Africa to determine the magnitude of this problem.

There are three African studies conducted among Egyptian children available in the literature. In a study by El-Hoshy and colleagues in 2003, pulmonary hypertension was detected in 12 (20%) out of 60 children with adenotonsillar hypertrophy and cor pulmonale, similar to our study findings.²⁴ In a study of 30 children aged 4-10 years, Elmofty and colleagues found a point prevalence of 26.7%.⁶⁵ Pulmonary hypertension in this latter study was defined as sPAP >30mmHg. Articles on pulmonary hypertension prior to the year 2008 cite various cut-offs for pulmonary hypertension. However after the consensus meeting for the updated classification of pulmonary hypertension at Dana Point, California, a cut-off mPAP value of ≥ 25 mmHg was agreed and is the current definition of pulmonary hypertension.⁴² To be able to compare the prevalence of 26.7% by the Egyptian colleagues with our data then an sPAP of >30mmHg converted to mPAP using the Chemla formula would translate to an mPAP >20mmHg as their definition of pulmonary hypertension. In our study, 79 children out of 123 (47.8%) had an mPAP > 20mmHg. Our prevalence of pulmonary hypertension therefore would almost be twice their prevalence of pulmonary hypertension.

The mean of the pulmonary arterial pressures reported was $22.7\text{mmHg} \pm 3.8$ among 80 Egyptian children aged 2.5 to 7 years who had presented with snoring, adenoid hypertrophy and variable presence of tonsillar enlargement. Pulmonary hypertension was not defined and a point prevalence of pulmonary hypertension was not reported.³⁰ The mean mPAP in our study was $18.87\text{mmHg} \pm 6.19$. There was not enough data to show whether our study population was similar to these Egyptian children. Inclusion of more ill children would result in a high mPAP. In addition, the type of pulmonary arterial pressure, either sPAP or mPAP used was not indicated therefore making it difficult to make a comparison.

The majority of studies on children with adenoid hypertrophy in relation to pulmonary hypertension have been carried out in the Middle East.^{29,37,51} In a study of 52 children aged 4-11 years with upper airway obstruction due to adenotonsillar hypertrophy, Yilmaz and colleagues found a point prevalence of 51.9%. Pulmonary hypertension was defined as mPAP $>20\text{mmHg}$ using the Mahan formula. This is very similar to our study findings despite the different formulas used in deriving mPAP. In our study, 79 children out of 123 (47.8%) had an mPAP $> 20\text{mmHg}$

Using an mPAI cut-off of $\geq 25\text{mmHg}$ and the Mahan formula, Moghaddam et al found a point prevalence of pulmonary hypertension reported of 7.3% among 55 Iranian children aged 4-14yrs with adenotonsillar hypertrophy and obstructive symptoms.²⁹ This was the only published study that used the currently accepted cut-off for the definition of pulmonary hypertension, mPAP $\geq 25\text{mmHg}$. The prevalence of pulmonary hypertension in

our study is three times higher (21.1%). This may be due to survivor bias or a different pattern of disease in our population.

Granzotto and colleagues evaluated 45 children aged 4.5 to 8.5 years scheduled for adenotonsillectomy indicated for sleep disturbed breathing. In this study from Brazil, which defined pulmonary hypertension as mPAP >20mmHg using the Chemla formula, a point prevalence of 13% was reported.^{45,61} The Chemla formula used is similar to our study. In our study, 79 children out of 123 (47.8%) had an mPAP > 20mmHg, nearly four times the prevalence of pulmonary hypertension in Brazil.

Globally, there is a lack of standardization of methods used in these studies with regard to differences in echocardiography machines, cut-off points defined for pulmonary hypertension, equations used to derive mPAP as well as patient selection especially in studies in children.^{29,30,37,45,51} Notwithstanding those differences, our study seems to have a higher prevalence of pulmonary hypertension, only similar to the Turkish study of 2004 by Yilmaz and colleagues. One may question whether our racial and genetic differences may play a role such that disease severity is inherently greater in our population. Variations in age, body mass index and altitude may also contribute to these differences.

Thirty three children out of 123(26.8%) had mPAP >21mmHg but < 25mmHg. In the literature, the clinical significance of this group is not known. Prior work has suggested that epidemiological work would perhaps give a clue.⁴³ Long term follow-up of such

patients would help determine the significance of pulmonary arterial pressures in this group.

The children in our study were much younger than those in published studies, the median age being 2.5 years with a range of 3.6 months to 8.5 years compared to a range of 4 to 14 years in the majority of the studies. Age was not significantly associated with pulmonary hypertension in our study and this is comparable to other published studies.^{29,30,37,45,51} The slight male preponderance was universal in the studies compared herein. In our study, males had an almost 3-fold increased chance of having pulmonary hypertension on bivariate analysis, gender was eliminated as an independent factor associated with pulmonary hypertension on multivariate analysis and this is comparable to other studies by Moghaddam et al, Abdel-Aziz et al, Yilmaz et al, Granzotto et al and Tatlipinar et al.^{29,30,37,45,51}

Most of the children in our study were using intranasal steroids (74.8%) with a good number using the intranasal steroids intermittently (72.8%) as opposed to continuously. Children who did not use intranasal steroids had a 3-fold increased odds of having pulmonary hypertension, implying that intranasal steroids confer protection. This is in keeping with the literature.⁴⁷ The jury is still out on the optimum duration of continuous intranasal steroid use.⁵² In multivariate analysis, non-use of intranasal steroid none-use was not associated with pulmonary hypertension.

Children had all the symptoms and signs of adenoid or adenotonsillar hypertrophy described in the literature.^{2, 13-16} The study children had fairly advanced disease with 6 of 10 symptoms reported to occur every day. This may be due to the fact that the children sampled were from a tertiary facility and may have been quite sick.

In our literature review we found many studies that correlated symptoms and signs of upper airway obstruction to obstructive sleep apnoea hypopnoea syndrome.^{51,57,58} Few studies have looked at clinical evaluation in predicting pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy.^{45,65} To our knowledge, this may be the first study to associate symptoms of upper airway obstruction, ENT and cardiovascular systemic findings to objectively measured pulmonary arterial pressures via echocardiography. In our study, a history of mouth breathing was found to have a 89% sensitivity and a 91% negative predictive value for pulmonary hypertension, whereas it had a 100% specificity and positive predictive value for obstructive sleep apnoea hypopnoea syndrome in Japanese children.⁵⁷

The presence of frequent upper respiratory tract infections, restless sleep, snoring, mouth breathing and night sweats on history were the most sensitive predictors for pulmonary hypertension and offer an opportunity for us to use these variables objectively as a screening tool for pulmonary hypertension in children with adenoid or adenotonsillar hypertrophy in resource-constrained settings. Daily mouth breathing singly or in combination with restless sleep showed the highest sensitivity (88.5%) and negative

predictive value (86.4%). This is probably the most useful finding in this study. This simple screening can be used to prioritize children for life saving surgery.

The prominent cardiovascular findings noted is the observation of a palpable P2 in one out of seven children and tachycardia in one out of ten children. The presence of palpable P2, a known sign of pulmonary hypertension was significant on multivariate analysis.^{42,43}

Published studies show that daytime oxygen saturations have a poor correlation with severity of obstructive sleep apnea hypopnea syndrome, with hypoxia occurring mainly at night or intermittently.⁵⁸ The hospital does not have a sleep laboratory, therefore night-time oxygen saturations were not measured. In this study, both bivariate and multivariate analysis of daytime oxygen saturations were significantly lower in the children with pulmonary hypertension compared to those with without.

Our study population had smaller tonsil size compared to other published studies.^{29, 30} This was probably because the majority of our children were younger. Before the age of 4 years, adenoid hypertrophy is predominant to tonsillar hypertrophy.^{3,4} In our study, <50% of the children had tonsils size grade ≥ 3 on the Brodsky scale compare to about 50-100% of children in published studies evaluating pulmonary arterial pressures in children with adenotonsillar hypertrophy. However, children with predominantly adenotonsillar hypertrophy had a higher prevalence of pulmonary hypertension, 18 out of 26 children (14.6%) compared to the children with predominantly adenoid hypertrophy, 8 out of 26 children (6.5%). Tonsil size in our study was not an independent risk factor for pulmonary

hypertension. Tatlipinar and colleagues unlike their counterparts from Brazil also showed that tonsillar hypertrophy (Brotsky grade 3 and 4) does not statistically correlate with pulmonary arterial pressures.^{45,51}

In published studies, ANR >0.63 has been used as a criteria for enlarged adenoids.⁵¹ Although not used as a criteria for inclusion into our study, all children in our study had adenoid hypertrophy defined as an ANR > 0.63cm. Similar to published studies, there was no statistically significant difference in mean ANR of children with and without pulmonary hypertension. One study found tonsil pharyngeal ratio (TPR) >0.66 to correlate well with pulmonary hypertension in children with adenotonsillar hypertrophy.⁴⁵ In our study TPR was not associated with pulmonary hypertension.

Study strengths

The strength of this study is the ability to use agreed upon cut-off points for pulmonary hypertension unlike previous articles in the literature which have different cut-offs for this parameter. In this study, pulmonary hypertension is defined as mPAP > 25mmHg as per the most recent expert consensus that was published in the year 2008 as the updated classification for pulmonary hypertension.^{42,43} The Chemla equation used to determine mPAP in our study is simple and has excellent correlation with cardiac catheterization. We also had the opportunity to use a non-invasive method to measure pulmonary arterial pressures and evaluate pulmonary hypertension.

The number of children with adenoid or adenotonsillar hypertrophy was much larger than in the studies we have compared giving us power and allowing us to make valid conclusions on our findings. The large study population recruited from a busy tertiary hospital ENT clinic and paediatric wards. The factors associated with pulmonary hypertension in children with adenoid hypertrophy or adenotonsillar hypertrophy in our study have not been investigated in previous studies. This is new information, biologically plausible, and can be easily used as a screening tool in our setting.

Immediate echocardiography results and feedback was given to the caregivers, radiology team and ENT team. This information expedited treatment and surgery for these children. Interestingly, the study team became a source of quick consultation which opened up a need that was quickly appreciated. This fostered multidisciplinary collaboration within four departments – paediatrics and child health, cardiology, ENT and radiology.

Study limitations and actions taken to reduce bias

Selection bias may have occurred as patients without lateral neck radiographs were excluded. Nonetheless, the children attending the ENT filter clinic during the study period were comparable with the children in our study with regard to age and sex making our results generalizable.¹¹ Despite standardization of the radiography technique, image quality was poor. There may have been an aspect of recall bias as the caregivers gave history of clinical symptoms which may have started a long time prior to seeking health care. There is no data of normal pulmonary pressures in children for age, sex, body mass index and geographical variation in our setting to truly cater for differences if any.

CONCLUSION

The point prevalence of pulmonary hypertension is 21.1% (95% CI 14.3 – 29.4%) in children with radiographically confirmed adenoid or adenotonsillar at KNH.

The presence of daily mouth breathing and or restless sleep is a sensitive screening tool for pulmonary hypertension (88.5%) with a high negative predictive value (86.4%)

RECOMMENDATIONS

1. Children with features of upper airway obstruction should be routinely screened for pulmonary hypertension using echocardiography.
2. A study to validate the proposed screening tool in a more general population of children with clinical symptoms of upper airway obstruction.

REFERENCES

1. Greenfeld M, Tauman R, DeRowe A, Sivan Y. Obstructive sleep apnea syndrome due to adenotonsillar hypertrophy in infants. *Int J Pediatr Otorhinolaryngol.* 2003; 67(10):1055-60.
2. Chang S, Chae K. Obstructive sleep apnea syndrome in children: Epidemiology, pathophysiology, diagnosis and sequelae. *Korean J Pediatr.* 2010; 53(10):863-71.
3. Arens R, McDonough J, Corbin A, Hernandez M, Maislin G, Schwab R, Pack A. Linear dimensions of the upper airway structure during development: assessment by magnetic resonance imaging. *American Journal of Respiratory and Critical Care Medicine.* 2002; 165(1):117-122.
4. Jeans W, Fernando D, Maw A, Leighton B. A longitudinal study of the growth of the nasopharynx and its contents in normal children. *The British journal of radiology.* 1981; 54(638):117-121.
5. Zautner AE. Adenotonsillar disease. *Recent Pat Inflamm Allergy Drug Discov.* Adenotonsillar disease. 2012; 6(2):121-9.
6. Huang SW, Giannoni C. The risk of adenoid hypertrophy in children with allergic rhinitis. *Ann Allergy Asthma Immunol.* 2001; 87:350-5.
7. Sadeghi Shabestari M, Moghaddam Y, Ghaharri H. Is there any correlation between allergy and adenoid hypertrophy? *Int J Pediatr Otorhinolaryngol.* 2011; 75:589-91.
8. Modrzynski M, Zawisza E. An analysis of the incidence of adenoid hypertrophy in allergic children. *Int J Pediatr Otorhinolaryngol.* 2007; 71:713-9.
9. Scadding G. Non-surgical treatment of adenoidal hypertrophy: The role of treating IgE-mediated inflammation. *Pediatr Allergy Immunol.* 2010; 21:1095-1106.
10. Friberg D, Sundquist J, Li X, Hemminki K, Sundquist K. Sibling risk of pediatric obstructive sleep apnea syndrome and adenotonsillar hypertrophy. *Sleep.* 2009; 32(8):1077-83.
11. Kenyatta national hospital statistics. (2011).
12. Sinha D, Guilleminault C. Sleep disordered breathing in children. *The Indian journal of medical research.* 2010; 131(3):311-20.

13. Marcus C, Emerson R. Sleep disordered breathing in children. *Sleep Rochester*. 2001; 164(11):16-30.
14. Woodson B, Franco R. Physiology of sleep disordered breathing. *Otolaryngologic Clinics of North America*. 2007; 40(4). 691-711.
15. Gatherer, S. A descriptive study of ECG and chest X-ray changes in children with adenoid and tonsillar hypertrophy. MMed ENT Surgery Dissertation; 2002.
16. Masinde, P. Indications and complications of adenoidectomy and tonsillectomy at KNH from 1987 to 1992 – A six year study. MMed ENT Surgery Dissertation. 1995.
17. Haapaniemi J. Adenoids in school-aged children. *J Laryngol Otol*. 1995; 109:196–2.
18. Thakkar K, Yao M. Diagnostic studies in obstructive sleep apnea. *Otolaryngologic Clinics Of North America*. 2007; 40(4):785-805.
19. Goldstein N, Pugazhendi V, Rao S, Weedon J, Campbell F, Goldman J, Post C, Rao M. Clinical assessment of pediatric obstructive sleep apnea. *Pediatrics*. 2004; 114:33-43.
20. Saedi B, Sadeghi M, Mojtahed M, Mahboubi H. Diagnostic efficacy of different methods in the assessment of adenoid hypertrophy. *American Journal of Otolaryngology - Head and Neck Medicine and Surgery*. 2010; 4-8.
21. Menashe V, Farrehi C, Miller M. Hypoventilation and cor pulmonale due to chronic upper airway obstruction. *J Pediatr*. 1965; 67:198–205.
22. Wilkinson A, McCormick M, Freeland A et al. Electrocardiographic signs of pulmonary hypertension in children who snore. *Br Med J (Clin Res Ed)*. 1981; 282: 1579–81.
23. Perkin R, Anas N. Pulmonary hypertension in pediatric patients. *J Pediatr*. 1984; 105:511–22.
24. El-Hoshy Z, Dewidar H, El-Bosraty H, et al. Adenotonsillar hypertrophy and cor-pulmonale. *Med J Cairo Univ*. 2003;71:579-84.
25. Blum R, McGowan F. Chronic upper airway obstruction and cardiac dysfunction: anatomy, pathophysiology and anaesthetic implications. *Pediatric Anaesthesia*. 2004; 14:75-83.

26. Anna S, Weber T, Montovani J, Matsubara B, Fioretto J. Echocardiographic abnormalities in children with obstructive breathing disorders during sleep. *Jornal de Pediatria*. 2007; 83(6):518-22.
27. Carolina M, Norte B, Cibelle S. Adenotonsillar Hypertrophy as a Cause of Pulmonary Hypertension. *Thorax*. 2008:133-136.
28. Bhattacharjee R, Kheirandish-Gozal L, Pillar G, & Gozal D. Cardiovascular complications of obstructive sleep apnea syndrome: evidence from children. *Progress in Cardiovascular Diseases*. 2009; 51(5):416-33.
29. Moghaddam Y, Babil S, Abavisani K, Melli R. Subclinical Pulmonary Hypertension in Children with Obstructive Adenotonsillar Hypertrophy. *Archive of SID. Cardiovascular Research*. 2010; 17-21.
30. Abdel-Aziz M. Asymptomatic cardiopulmonary changes caused by adenoid hypertrophy. *J Craniofac Surg*. 2011;140-1403.
31. Tatlipinar A, Duman D, Uslu C, Egeli E. The effects of obstructive sleep apnea syndrome due to adenotonsillar hypertrophy on the cardiovascular system in children. *The Turkish Journal of Pediatrics*. 2011; 53:359-63.
32. Durmowicz A, Stenmark K. Mechanisms of structural remodeling in chronic pulmonary hypertension. *Pediatrics in review*. 1999; 20(11):91-102.
33. Biltagi M, Maguid M, Ghafar M, Farid E. Correlation of 8-isoprostane, interleukin-6 and cardiac functions with clinical score in childhood obstructive sleep apnoea. *Acta paediatrica*. 2008; 97(10):1397-1405.
34. Heresi G, Dwei, R. Biomarkers in Pulmonary Hypertension. *Review Literature And Arts Of The Americas*. 2010; 2(1):12-16.
35. Guillemineault C, Huang Y, Glamann C, Li K, Chan A. Adenotonsillectomy and obstructive sleep apnea in children: A prospective survey. *The Laryngoscope*. 2007:169-175.
36. Abd El-Moneim E, Badawy B, Atya M. The effect of adenoidectomy on right ventricular performance in children. *International Journal of Pediatric Otorhinolaryngology*. 2009; 73(11):1584-88.

37. Yilmaz M, Onrat E, Altuntaş A, Kaya D, Kahveci O, Ozel O, Dereköy S. The effects of tonsillectomy and adenoidectomy on pulmonary arterial pressure in children. *American Journal of Otolaryngology*. 2005; 26(1):18-21.
38. Miman M, Kirazli T, Ozyurek R. Doppler echocardiography in adenotonsillar hypertrophy. *International Journal of Pediatric Otorhinolaryngology*. 2000; 54:21-6.
39. Milan A, Magnino C, Veglio F. Echocardiographic indexes for the non-invasive evaluation of pulmonary hemodynamics. *Journal of the American Society of Echocardiography*. 2010; 23(3):225-39.
40. Friedberg M, Feinstein J, Rosenthal D. Noninvasive assessment of pulmonary arterial capacitance by echocardiography. *Journal of the American Society of Echocardiography*. 2007; 20(2):186-90.
41. Yock P, Popp R. Non invasive estimation of right ventricular pressure by Doppler ultrasound in patients with tricuspid regurgitation. *Circulation*. 1984; 70:657-62.
42. Simonneau G, Robbins I, Beghetti M, Channick R, Delcroix M, Denton C, Elliott C. Updated clinical classification of pulmonary hypertension. *Journal of the American College of Cardiology*. 2009; 54(1 Suppl):S43-S54.
43. Galie N, Hoeper M, Humbert M, Torbicki A, Vachiery J, Barbera J et al. Guidelines for the diagnosis and treatment of pulmonary hypertension. The taskforce for the diagnosis and treatment of pulmonary hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society(ERS) endorsed by the International Society of Heart and Lung Transplant (ISHLT). *European Heart Journal*. 2009; 30:2493-2537.
44. Naiboglu B, Deveci S, Duman D, Kaya K, Toros S, Kinis V, Sürmeli M. Effect of upper airway obstruction on pulmonary arterial pressure in children. *International Journal of Pediatric Otorhinolaryngology*. 2008; 72(9):1425-29.
45. Granzotto E, Aquino F, Flores J, Lubianca N. Tonsil size as a predictor of cardiac complications in children with sleep-disordered breathing. *The Laryngoscope*. 2010; 120(6):1246-51.
46. Schermuly R, Dony E, Ghofrani H. Reversal of experimental pulmonary hypertension by PDGF inhibition. *J Clin Invest*. 2005; 115:2811-21.

47. Zhang L, Mendoza-Sassi RA, Cesar JA, Chadha NK. Intranasal corticosteroids for nasal airway obstruction in children with moderate to severe adenoidal hypertrophy. Cochrane review. 2008. Issue 3.
48. Oburra HO, Idenya M. Frequency of adenotonsillectomy in some Nairobi hospitals. East African Medical Journal. 2001; 78:338-42.
49. Monset-Couchard M, Mason CV, Moss AJ. Cor pulmonale in children. Curr Probl Pediatr. 1975; 5(4):3-31.
50. WHO guidelines. www.who.int/publications/guidelines
51. Tatlipinar A, Biteker M, Meric K, Bayraktar G, Tekkesin A, Goceer T. Adenotonsillar Hypertrophy: Correlation Between Obstruction Types and Cardiopulmonary Complications. The Laryngoscope 2012; 122:676-80.
52. Kuhle S, Urshitz M. Anti-inflammatory medications for obstructive sleep apnea in children. Cochrane review. 2011. Issue 1.
53. Gozal L. Effect of montelukast therapy in obstructive sleep apnea (OSA) children (32543). <http://clinicaltrials.gov/ct2/show/NCT01027806> 2009.
54. Bernardi F. Efficacy of oral leukotriene in long term therapy of mild and moderate obstructive sleep apnea syndrome in children (SB-OSAS). <http://clinicaltrials.gov/ct2/show/NCT00912171> 2009.
55. Stroker A, Shapiro N. Parental understanding and attitudes of pediatric obstructive sleep apnea and adenotonsillectomy. Int J Pediatr Otorhinolaryngol. 2007; 71:1709-15.
56. Shintani T, Asakura K, Kataura A. Adenotonsillar hypertrophy and skeletal morphology of children with obstructive sleep apnea syndrome. Acta Otolaryngol 1996; 523(suppl):222-4.
57. Xu Z, Cheuk D, Lee S. Clinical evaluation in predicting childhood OSA. Chest 2006; 130:1765-71.
58. Brouillette R, Morieli A, Leimanis A, Waters K, Luciano R, Darchime F. Nocturnal pulse oximetry as an abbreviated testing modality for pediatric obstructive sleep apnea. Pediatrics. 2000; 105(2):405-412.
59. Cerro M, Abman S, Diaz G, Freudenthal F, Harikrishnan F, Haworth S et al. A consensus approach to the classification of pediatric pulmonary hypertensive

- vascular disease: Report from the PVRI pediatric taskforce, Panama 2011. *Pulmonary Circulation*. 2011; 1(2):286-298.
60. Aduen J, Castello R, Daniels J, Diaz J, Safford R, Heckman M et al. Accuracy and precision of three echocardiographic methods for estimating mean pulmonary artery pressure. *Chest* 2011; 139:347–52.
61. Chemla D, Castelain V, Provencher S, Humbert M, Simonneau G, Herve P. Evaluation of various empirical formulas for estimating mean pulmonary artery pressure by using systolic pulmonary artery pressure in adults. *Chest* 2009; 135:760–8.
62. Arcasoy S, Christie J, Ferrari V et al. Echocardiographic assessment of pulmonary hypertension in patients with advanced lung disease. *Am J Respir Crit Care Med*. 2003; 167(5):735-40.
63. Er F, Ederer S, Nia A, Caglayan E, Dahlem K, Semmo N, Gassanov N. Accuracy of Doppler echocardiographic mean pulmonary artery pressure for diagnosis of pulmonary hypertension.
64. Dabestani A, Mahan G, Gardin J, et al. Evaluation of pulmonary artery pressure and resistance by pulsed Doppler echocardiography. *Am J Cardiol* 1987; 59:662-8.
65. Elmofty I, Abdel-Razek M, Abdel-Rahman H, Rashid H, El-Refaii A. Effect of adenotonsillectomy on pulmonary arterial blood pressure in children with adenotonsillar hypertrophy. Benha University, Egypt. 2005.
http://bu.edu.eg/portal/uploads/Medicine/CARDIOLOGY/Hesham%20Khalid%20Rashid%20Mosa/Hesham%20Khalid%20Rashid%20Mosa_effect%20of%20adenotonsillectomy.doc
66. Brodsky L, Moore L, Stanievich JF. A comparison of tonsillar size and oropharyngeal dimensions in children with obstructive adenotonsillar hypertrophy. *Int J Pediatr Otorhinolaryngol* 1987; 13:149-56.
67. Major M, Flore-Mir C, Major P. Assessment of lateral cephalometric diagnosis of adenoid hypertrophy and posterior upper airway obstruction: a systematic review. *Am J Orthod Dentofacial Orthop*. 2006; 130(6):700-8.

68. Feres M, Hermann J, Cappellette M, Pignataris S. Lateral x-ray view of the skull for the diagnosis of adenoid hypertrophy: a systematic review. *Int J Pediatr Otorhinolaryngol*. 2011; 75(1):1-11.
69. Souki M, Souki B, Franco L, Becker H, Araújo E. Reliability of subjective, linear, ratio, and area cephalometric measurements in assessing adenoid hypertrophy among different age groups. *Angle Orthodontist*. 2012; In- Press.
<http://dx.doi.org/10.2319/010612-13.1>
70. Song M, Baek J, Kwon B, Kim G, Bae E, Noh C, et al. Clinical spectrum and prognostic factors of pediatric ventricular tachycardia. *Circ J*. Sep 2010; 74(9):1951-8.
71. Sex-specific BMI Charts. <http://www.cdc.gov/growthcharts>
72. Jubran A. Pulse oximetry. *Crit Care*. 1999; 3(2):11-17
73. Henry V' et al. Report of the American Society of Echocardiography Committee on Nomenclature and standards in Two-Dimensional Echocardiography. *J Am Coll Cardiol* 1983; 6:1471-78

APPENDICES

Appendix I – Ethical Approval

Recd 11/7/2011

**PREVALENCE OF PULMONARY HYPERTENSION
IN CHILDREN WITH ADENOID & OR TONSILLAR
HYPERTROPHY AT THE KENYATTA NATIONAL
HOSPITAL**



**DR. DIANA MARANGU
MBCbB (UON)**



**RESEARCH PROPOSAL FOR DISSERTATION SUBMITTED IN PART FULFILLMENT
OF THE REQUIREMENTS OF THE UNIVERSITY OF NAIROBI FOR AWARD OF THE
DEGREE OF MASTER OF MEDICINE IN PAEDIATRICS**

Appendix II - Informed Consent

STUDY IDENTIFICATION No. _____ **HOSPITAL No.** _____

The purpose of this study is to estimate the prevalence of pulmonary hypertension seen in children with adenoid and or tonsillar hypertrophy seen at KNH. The information gathered will be used to improve the management of children with adenotonsillar hypertrophy.

There is no harm or risk anticipated for participating in this study. The only additional test that will be carried out is echocardiography which will be at no extra cost to you. Echocardiography is a safe non invasive investigation. Benefits of the study include early identification of any heart problem, which will mean early intervention or treatment.

Participation in this study is out of your own free will. Medical care will not be denied in case you decline to participate in the study. You may terminate participation at any time with no consequences whatsoever. All information will be treated with confidentiality.

I, the undersigned have been explained to, understand the above, and voluntarily accept to participate in the study.

Signature/Thumb print (Parent/Guardian)

Telephone No. (Parent/Guardian)

Date

ENQUIRIES

For any enquiries or further clarification, please contact:-

DR. DIANA MARANGU – PRINCIPAL INVESTIGATOR - Tel 0721-282815

Appendix III- Fomu ya Idhini

Nambari ya Utafiti _____ Nambari ya Hospitali _____

Lengo la utafiti huu ni kuchunguza ukubwa wa shida ya pulmonary hypertension kwa watoto ambao wamengojeka na ukubwa wa adenoids na tonsils katika Hospitali ya Taifa ya Kenyatta. Matokeo ya utafiti itakuwa muhimu katika kuboresha kufuatiliwa kwa watoto wenye ukubwa wa adenoids na tonsils

Hakuna madhara au hatari inayotarajiwa kwa kushiriki katika utafiti huu. Kipimo cha ziada nje ya yale kawaida kwa matibabu itaayofanywa ni ya echo. Hakuna gharama yeyote ya ziada utatokana kwa ajili ya kushiriki katika utafiti. Kipimo cha echo ni mbinu ya uchunguzi ambayo haina madhara. Moja yao ya manufaa ya hii utafiti ni kuwa kukiwa na shida ya roho inagunduliwa mapema kukimaanisha matibabu itapewa mapema. Kushiriki kwa utafiti huu ni kwa hiari yako mwenyewe na hauwezi kushurutishwa. Utahudumiwa ata kama utakataa kuhusika kwa huu utafiti. Una uhuru kutamatisha kuhusika wakati wowote bila madhara yoyote yale.

Habari zozote utakazotoa zitawekwa kwa siri na jina lako halitachapishwa popote.

Mimi, niliyetia sahihi, nilielezewa, nimeelewa, na kwa hiari nakubali kushiriki katika utafiti.

Saini/Alama ya kidole (Mzazi/Mlinzi)

Nambari ya Simu (Mzazi/Mlinzi)

Tarehe

MAELEZO YA ZIADA

Kwa maelezo ya ziada au ufafanuzi, tafadhali wasiliana na:-

DKT. DIANA MARANGU – MTAFITI MKUU - Tel 0721 282815

Appendix IV – Eligibility Criteria Checklist

INCLUSION CRITERIA

All children with

1. Clinician diagnosed adenoid or adenotonsillar disease
AND
2. Radiologically confirmed adenoid hypertrophy on lateral neck radiograph.

EXCLUSION CRITERIA

Approach

1. Refusal to grant consent

History

2. Known cardiac disease/ sickle cell anemia / HIV

General Examination

3. Neurological abnormalities. E.g. Cerebral palsy
4. Genetic syndromes with craniofacial abnormalities. E.g. Down syndrome
5. BMI > 95th percentile for age

ENT Examination

6. Other causes of airway obstruction
(deviated septum, nasal polyposis, gross turbinate hypertrophy)

Echocardiography

7. Echocardiography not performed
8. Other structural cardiac abnormality.

Appendix V – Questionnaire & Proforma Tool

STUDY IDENTIFICATION No. _____ HOSPITAL No. _____

PATIENT CHARACTERISTICS

- | | |
|--------------------------------------|---------------------------------------|
| 1) Date of Birth | a. Snoring [] |
| 2) Sex (M=1, F=2)[] | b. Difficulty in breathing [] |
| 3) Weight (kg) | c. Disturbed sleep [] |
| 4) Height (cm) | 9) Nasal steroids (1=Yes,2=No)[] |
| 5) W/H | a. Continuous [] |
| 6) BMI | b. Intermittent [] |
| 7) Duration of symptoms (mths)..... | 10) Scheduled surgery (1=Yes,2=No)[] |
| 8) Caregiver concern (1=Yes,2=No)[] | Date _____ |

HISTORY & PHYSICAL EXAM (circle appropriately) – Adapted from Goldstein AAP

CLINICAL FEATURE	FREQUENCY OF SYMPTOM OR PHYSICAL SIGN			
	Every Night	Night per/week	Other	Never
<u>Night Symptoms</u>				
Snoring				
Pauses				
Restless sleep				
Frequent awakening				
Night sweats				
<u>Day Symptoms</u>	Every Day	Days per week	Other	Never
Nasal obstruction				
Mouth breathing				
Frequent URTIs				
Hyperactivity				
Enuresis (>4years)				
<u>Physical Exam</u>				
Mouth breathing	Present	Absent		
Tonsil size(Brodzky)	4+	3+	2+	0-1+

CLINICAL FEATURES OF PULMONARY HYPERTENSION & COR PULMONALE

- 1) Oxygen saturation on pulse oximetry (%)
- 2) Respiratory rate (breathes/min)
 - Tachypnea (1= Yes, 2=No) []
- 3) Heart rate (beats/min)
 - Tachycardia(1= Yes, 2=No) []
- 4) Edema (1= Yes, 2=No) []
- 5) Jugular venous distension (1= Yes, 2=No) []
- 6) Hepatomegaly (1= Yes, 2=No) []
- 7) Palpable P2 (1= Yes, 2=No) []
- 8) Murmur (1= Yes, 2=No) []
 - Location
 - Type (1=Systolic, 2=Diastolic) []

LATERAL NECK RADIOGRAPH EVALUATION

X-Ray number:

Closed mouth Open Mouth Rotation Magnification

Adenoid size (cm):

Tonsil size (cm):

Tonsil-pharyngeal ratio (TPR) =

Adenoidal-nasopharyngeal ratio (ANR) =

ECHOCARDIOGRAPHIC EXAMINATION

Echo number:

Tricuspid regurgitation velocity (m/s^2) =

Systolic PAP (mmHg) =

Mean PAP (mmHg) =

Other significant findings:-

.....
.....
.....

Appendix VI – Measuring techniques

Weight measurement (WHO)⁵⁶

- A bathroom weighing scale was used.
- Every morning before the scale was used; it was checked against a known weight of 10 Kg.
- The clothes of the child were removed.
- The child stood on the scale and the measurement taken.
- The measurer reads the reading perpendicular to the pointer to the nearest 0.1 kg and announced loudly.
- The guardian was weighed holding the child that was unable to stand and his/her weight subtracted from the total weight to get the child's true weight.
- Two readings were made and average taken

Height measurement (WHO)⁵⁶

A measuring board was used for measuring height in children.

For children who are less than two years of age or those who are more than two years and cannot stand:

- The child was gently placed on the board with the soles of the feet flat against the fixed vertical part.
- The head was put near the moving part (cursor)
- The child was made to lie straight in the middle of the board, looking directly up.
- The assistant held the feet firmly against the feet board and places one hand and the knees of the child.
- The measurer gently held the child's head places the cursor against the crown of the head and reads out the length to the nearest 0.1 cm.
- The assistant repeated the reading and records it in the recording form.

For children 2 years of age and above:

- The child was made to stand on a horizontal surface against a vertical measuring device.
- The assistant makes sure that the child stands straight with the heels, knees against the wall.
- The cursor was then lowered onto the child's crown of the head.
- The length was read to the nearest 0.1 cm.

The measurer read out loud; the assistant repeated it and recorded it on the recording form.

Lateral neck radiograph measurements (Shintani)⁵⁶

Linear measurements were taken using a ruler measured in centimeters to the nearest 0.1cm including adenoid size, tonsil size, nasopharyngeal size and pharyngeal size, using the landmarks as proposed by Shintani depicted in Figure 4.

UNIVERSITY OF NAIROBI
MEDICAL LIBRARY