

**PREVALENCE AND PATTERN OF SENSORINEURAL HEARING IMPAIRMENT
AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS AT THE KENYATTA
NATIONAL HOSPITAL**

PRINCIPAL RESEARCHER: DR. NEVILLE OKWIRI

MMED OTORHINOLARYNGOLOGY, HEAD & NECK SURGERY

H58/68742/11

SUPERVISORS: PROFESSOR ISAAC MACHARIA

PROFESSOR AND CONSULTANT ENT, HEAD & NECK SURGEON

DEPARTMENT OF SURGERY, UNIVERSITY OF NAIROBI

DR. CATHERINE IRUNGU

LECTURER AND CONSULTANT ENT, HEAD & NECK SURGEON

DEPARTMENT OF SURGERY, UNIVERSITY OF NAIROBI

A STUDY SUBMITTED TO THE UNIVERSITY OF NAIROBI SCHOOL OF MEDICINE IN
PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE AWARD OF MASTERS
DEGREE IN OTORHINOLARYNGOLOGY, HEAD & NECK SURGERY

DECLARATION

I hereby declare that this is my original work and has not been presented for the award of any academic credit in any Research institution or University.

No part of this thesis may be reproduced without the permission of the author or university.

Neville Okwiri.

Signature _____ **Date** _____

This thesis has been submitted with our approval as the University Supervisors.

Professor Isaac Macharia

Professor and Consultant ENT- Head and neck surgeon,

Department of surgery,

University of Nairobi

Signature _____ Date _____

Dr. Catherine Irungu

Lecturer and Consultant ENT- Head and neck surgeon,

Department of surgery,

University Of Nairobi

.Signature _____ Date _____

ACKNOWLEDGEMENT

I wish to acknowledge those whose efforts resulted in the completion of this study. I am very grateful to my supervisors Prof. Isaac Macharia and Dr. Catherine Irungu for their guidance throughout the study period together with all other faculty members of the Department of E.N.T, Head and Neck Surgery for their review and positive criticism during the study duration.

This study was made possible by partial financial grants from the Extension and Research committee of the Kenyatta National Hospital and the Deans Committee Research Grant (DCRG) of the University of Nairobi.

ABBREVIATIONS

ABR – Auditory Brainstem Response

ANSI American National Standards institute

BMI – Body Mass Index

DM – Diabetes Mellitus

dbHL – decibel hearing level

ENT – Ear, Nose and Throat

Hz - Hertz

KNH - Kenyatta National Hospital

PTA - Pure Tone Audiometry

CONTENTS

ACKNOWLEDGEMENT	3
ABBREVIATIONS	4
CONTENTS.....	5
ABSTRACT.....	7
BACKGROUND	7
AIM.....	7
METHODS	7
RESULTS	8
<i>CONCLUSION AND RECOMMENDATION</i>	8
CHAPTER 1: INTRODUCTION	9
EPIDEMIOLOGY	10
DIAGNOSIS	11
MANAGEMENT.....	11
CHAPTER 2: LITERATURE REVIEW	12
2.2 STUDY JUSTIFICATION	14
2.3 AIMS AND OBJECTIVES OF THE STUDY	14
GENERAL OBJECTIVE.....	14
SPECIFIC OBJECTIVES	14
CHAPTER 3: STUDY METHODS.....	15
3.1 STUDY SITE AND POPULATION	15
3.2 STUDY DESIGN.....	15
3.3 SAMPLING	16
3.4 STUDY PROCEDURES	16

3.5 DATA MANAGEMENT.....	17
3.6 DATA ANALYSIS.....	17
3.7 ETHICAL CONSIDERATIONS.....	18
CHAPTER 4: RESULTS.....	19
CHAPTER 5: DISCUSSION.....	28
WORK PLAN.....	31
BUDGET	31
REFERENCES	32
APPENDIX.....	38
APPENDIX 1: CONSENT INFORMATION DOCUMENT.....	38
KIAMBATISHO 1: FOMU YA MAELEZO KUHUSU IDHINI YA MGONJWA.....	40
KIAMBATISHO 2: KIBALI CHA UTAFITI.....	41
Appendix 3: study proforma	42
Appendix 4: Ethical approval	45

ABSTRACT

BACKGROUND

Type 2 Diabetes Mellitus is an illness in which insulin secretion and action is impaired. In 2014 the global prevalence of diabetes was estimated to be 9% among adults. In Africa the prevalence of diabetes has been estimated to range from 1% in rural Uganda to 12% in urban Kenya. Patients with diabetes have been shown to have worse hearing as compared to healthy individuals.

AIM

To determine the prevalence and pattern of sensorineural hearing impairment among patients with type 2 diabetes mellitus at the Kenyatta National hospital.

METHODS

Study setting: Kenyatta National Hospital ENT, Head and Neck Surgery outpatient department, Diabetic outpatient clinic and Nairobi Audiology Centre.

Study design: This was a hospital-based cross sectional survey that was carried out for a duration of three months between the months of February and May 2016.

Methodology: A total of 78 patients between 22 – 55 years of age on follow up for type 2 Diabetes Mellitus were recruited into the study. Pure tone audiometry was carried out at 250, 500, 1000, 2000, 4000, 6000 and 8000 Hz. The participants then underwent Auditory Brainstem Response (ABR) testing. Demographic, anthropometric, clinical and laboratory data was collected on a preformatted questionnaire. Data was analyzed using SPSS. Descriptive statistics was used for the population demographic characteristics. Univariate and multivariate analysis was used to determine correlates of risk factors to hearing impairment in diabetes mellitus patients

RESULTS

A total of 78 patients were recruited in to the study and overall 39.7% of patients with type 2 diabetes were found to have hearing loss. Mild sensorineural hearing loss accounted for 90.3% of those with hearing loss with a majority of them having high frequency hearing loss.

The ABR wave I, III & V absolute latencies were found to be significantly shorter as compared to normative data. However, the interpeak latencies were similar to normative data.

CONCLUSION AND RECOMMENDATION

The hearing loss associated with type 2 diabetes was shown to be mild and as such, the evidence we have as of now, doesn't justify the added cost to advocate for routine hearing assessment in patients with type 2 diabetes.

CHAPTER 1: INTRODUCTION

11. BACKGROUND

The dysfunction in type 2 diabetes is characterised by hyperglycaemia that occurs as a result of insulin resistance, inadequate insulin secretion, and inappropriate glucagon secretion. Patients with type 2 diabetes mellitus retain some ability to secrete insulin (1).

The complications that contribute to morbidity and mortality in diabetes mellitus include; hypoglycaemia, increased risk of infections, microvascular complications, neuropathic complications, and macrovascular disease. Hyperglycaemia which is a hallmark of all forms of diabetes mellitus affects the micro vasculature and nerves, that are integral in hearing.

The chronic hyperglycaemia leads to diabetic microvasculopathy in the peripheral nerves, renal glomerulus, and retina. These lead to blindness, end stage kidney disease, and debilitating neuropathies (2). Diabetes also leads to increased incidence of macrovascular atherosclerosis resulting in a higher risk of myocardial infarction, stroke and limb amputation (3). Molecularly the hyperglycaemic vasculopathy seems to stem from over production of superoxide by the mitochondrial electron transport chain (4).

A post mortem examination of 8 temporal bones of diabetics who had been known to have worse hearing as compared to age and sex matched controls revealed microangiopathy (5). Histologically other studies showed arteriosclerotic changes in the cochlea (6).

There is a marked similarity between the cochlear stria vascularis and the renal nephrons (7, 8), hence the vasculopathy that leads to renal failure could also be responsible for hearing loss in diabetic individuals (9).

The displacement of the cochlea is highly frequency specific (10). Maximal displacement for higher frequencies is at the basal end while lower frequencies are at the apex. A number of studies have shown that hearing loss among diabetics predominantly involves the high frequencies (8,11,12). This is similar to presbycusis, that affects higher frequencies first (13,14). Sensory hair cell loss and cochlea neuron loss at the basal turns are thought to be responsible for presbycusis (15,16). Nakae et al (17) made similar observations in an animal model of diabetes mellitus. High frequency hearing impairment is associated with difficulties understanding speech and thus impacting negatively on ones life (18).

The Central auditory system begins at the cochlea nuclei and in addition to transmission of acoustic information to higher centres is responsible for critical functions such as sound localization (19). The brainstem auditory evoked response (ABR) was first described by Sohmer and Feinmesser in 1967 (20). The ABR has seven waves. Activity in the cochlear nerve is represented by waves I & II. Waves III & IV represent activity in the cochlea nucleus and superior olivary complex respectively. Wave V is the most robust and represents activity generated primarily from neurons located within the lateral Lemniscal tracks (21).

ABR is commonly used to estimate peripheral hearing sensitivity of patients who cannot (eg infants & young children) or will not (eg malingerers) cooperate with behavioural testing. ABR is also useful in otoneurological assessment. Generally this is accomplished by examining the morphology and latencies of the click evoked ABR measured using high level stimuli. There are several different ways in which the click evoked ABR is altered by the presence of retrocochlear pathology. For example, an ABR that is absent in an ear with audiometric thresholds in the normal to moderate range, an ABR that is characterised only by early peaks, or an ABR with a wave V amplitude that is much smaller than the amplitude of wave I have all been interpreted as evidence of potential abnormality (22,23). Such grossly abnormal ABR morphologies are thought to reflect partial conduction block or significant loss of cross fiber synchrony along the neural pathways between the cochlear and the inferior colliculus. A second measure that is commonly used to diagnose potential retrocochlear pathology is prolonged interpeak or absolute latencies. In most persons, a high level click stimulus will generate an ABR with a wave V latency of approximately 5.5 msec and with a I-V interpeak latency of approximately 4.0 msec (24). The presence of retrocochlear pathology may slow neural conduction velocities. The presence of changes in central auditory and cognitive processing have also been documented in subjects with diabetes (25).

EPIDEMIOLOGY

In 2009 the prevalence of DM in Kenya was estimated to be 4.2% (26). However a marked difference was noted between the rural and urban populations. The prevalence ranged from a high of 12.2% in Nairobi to 2.2% in rural areas. These findings were similar to a study by Mathenge et al in 2010 (27). A study by Ayah R & Otieno CF (28) in Kibera, found the age

adjusted prevalence of diabetes to be 5.3%. Noteworthy in this study was the presence of very high concomitant, major, cardiovascular disease risk factors of cigarette smoking and alcohol abuse. The overall prevalence of diabetes is expected to rise due to increased rural to urban migration and the adaptation of a much more sedentary urban lifestyle (28).

DIAGNOSIS

The current WHO diagnostic criteria for diabetes is fasting plasma glucose $>7.0\text{mmol/l}$ or a 2 hour post prandial plasma glucose $> 11.1\text{ mmol/l}$ (1).

Hearing loss in adults defined as the pure tone average of the frequencies 500, 1000, 2000 and 4000 Hz greater than 25 db hearing level in the worse ear (29).

MANAGEMENT

Glycaemic control is achieved via diet and lifestyle modification, oral hypoglycaemics and exogenous insulin administration. Control of lipids and hypertension helps reduce macrovascular complication risk.

Management of patients with hearing impairment depends on the degree. Hearing aids are the main option for patients with mild, moderate or severe impairment. Cochlea implants are indicated for patients with profound hearing loss.

CHAPTER 2: LITERATURE REVIEW

In a survey of 5,742 participants patients with diabetes mellitus were found to have greater hearing loss as compared to non-diabetics (30). Cheng YJ et al (31) showed that the prevalence of hearing impairment among adults without diabetes aged 25 to 69 years from 1971 to 2004 in the United States decreased from 27.9% to 19.1% but among adults with diabetes there was no significant change (46.4% to 48.5%). In a systematic review by Akinpelu et al (32) in 2014 found the prevalence of hearing loss ranging from 44% to 69.7% for diabetic subjects and from 20% to 48.6% for non-diabetic controls. A study by Mozaffari et al among diabetic patients less than 60 years found a 45% prevalence of sensorineural hearing loss in the diabetics versus 20% in controls (33)

Hearing thresholds in patients with diabetes have been reported to correlate with diabetic complications, poor glycaemic control and diabetes duration (7, 34) however other studies didn't have similar findings (8, 35, 36). In the general population cardiovascular disease and its risk factors have been associated with hearing impairment (37, 38). A study of diabetics (39) among a population based sample showed that nephropathy was associated with hearing impairment.

Sensorineural hearing loss has been attributed to diabetic neuropathy (40). Duck et al (41) reported that co-existing diabetes may intensify hypertensive end organ disease of the cochlea.

Kakralapudi et al (42) in a chart review of diabetics found that creatinine levels were associated with severity of hearing loss. Other reports have found that severity of hearing impairment is positively associated with urine albumin excretion rate in patients with type 2 diabetes (43). The association between chronic hyperglycaemia and increased risk of microvascular complications in patients with type 1 diabetes mellitus was demonstrated in the Diabetes Control and Complications Trial (DCCT) (44). This has been corroborated by the epidemiology of diabetes intervention and complication study that has demonstrated continued benefit from intensive treatment (45,46). Studies have shown an association between glycaemic control, as assessed by HbA_{1c} levels, and hearing loss (47).

A study by Durmus et al (48) assessed neural conductance along the auditory pathway in diabetic patients and controls with normal hearing. ABR recordings revealed that absolute latencies of waves I, III & V were prolonged in the diabetic group as compared to controls. Toth in 2003 (49) revealed a difference in the interpeak latencies I – III & I – V between diabetic

patients and healthy controls. Gupta et al in 2010 (50) found the latency of wave I to be equal in diabetics and controls. The latency of waves III & V were delayed. The interpeak latencies I – III, III – V, and I – V were delayed in the diabetic group. Longer disease duration and severity were associated with abnormalities in the ABR.

Author	Type of study	N (DM/Controls)	Remarks	Method of Hearing assessment
Bainbridge et al (2004)	Cross sectional survey NHANES	758/1501	Greater hearing loss among diabetics	PTA
Cheng YJ (2009)	Cross sectional	NHANES	No change in prevalence of HL among diabetics	PTA
Akinpelu (2014)	Systematic review	29 articles reviewed	➤ High frequency HL	N/A
Mozaffari (2010)	Case control	80/80	Prevalence 45% Diabetics vs 20% controls	PTA
Dalton DS (1998)	longitudinal	344/3227	Nephropathy associated with hearing loss	PTA
Panchu (2008)	Case control	41/41	HbA1c > 8% associated with	PTA

			higher thresholds	
Gupta (2010)	Case control	25/25	Increased latency of wave V	ABR
Toth (2001)	Case control	15/15	Lower amplitude of waves I,III,V	ABR
Durmus C (2004)	Case Control	43/43	Increased absolute latency	PTA/ABR

2.2 STUDY JUSTIFICATION

Most studies of diabetes and hearing have limited themselves to the peripheral auditory system. We undertook a cross sectional survey, investigating both the peripheral and central auditory pathways.

2.3 AIMS AND OBJECTIVES OF THE STUDY

GENERAL OBJECTIVE

To determine the prevalence of sensorineural hearing loss among patients with type 2 Diabetes Mellitus at the KNH.

SPECIFIC OBJECTIVES

1. To determine demographic and clinical characteristics of study subjects.
2. To determine pattern & degree of sensorineural hearing loss among diabetics.
3. To assess neural conductance along the auditory pathway

CHAPTER 3: STUDY METHODS

3.1 STUDY SITE AND POPULATION

The study site was the KNH Diabetic outpatient clinic located at clinic number 17 within the outpatient complex. The clinic is open on every weekday and in addition a consultant diabetic clinic is held on Friday. The screening hearing test was carried out at the KNH ENT outpatient clinic located at clinic number 34 within the outpatient complex. The ABR testing was carried out at the Nairobi audiology centre 1st floor Landmark plaza on Argwings Kodhek road approximately one kilometer from KNH. Currently KNH does not have a functional ABR machine. The Nairobi Audiology centre was chosen due its proximity to KNH and the availability of qualified audiologists.

The inclusion criteria were:

1. Patients diagnosed with type 2 diabetes mellitus at the Kenyatta National Hospital.
2. Patients between the ages of 18 and 55 years.
3. Patients who consent to participate in the study.

The exclusion criteria were:

1. Age greater than 55 years whose hearing loss may be complicated by presbycusis or less than 18 years as the vast majority of diabetic patients in this age group suffer from type 1 diabetes.
2. Diagnosed with congenital hearing loss
3. History of occupational noise exposure, middle ear disease, prior treatment with ototoxic medication and temporal bone trauma
4. Patients declining to participate in the study

3.2 STUDY DESIGN

This was a hospital based cross sectional survey.

3.3 SAMPLING

Simple random sampling was utilized until the desired sample size was reached.

The desired sample size as calculated using the formula⁵¹;

$$\text{Where: } n = \frac{\{u\sqrt{\pi(1-\pi)} + v\sqrt{\pi_0(1-\pi_0)}\}}{(\pi - \pi_0)^2}$$

n = sample to be selected

u = One-sided percentage point of the normal distribution corresponding to power of 80%, therefore 0.84

v = Two-sided percentage point of the normal distribution corresponding to 95% level of significance, therefore 1.96

π_0 = The proportion of hearing loss cases in the general population. Estimated to be 5.3% according to the World Health Organization⁵²

π = The proportion hypothesized to be detected, given by 15%⁵²

The sample size obtained thus becomes 78 persons. Including a 5% increase in the sample to account for drop outs, we get a sample to be selected of 83 persons.

3.4 STUDY PROCEDURES

The principal researcher took participants bio data and a comprehensive history including duration of diabetes, co-morbidities, and medication use. The principal researcher also carried out a comprehensive otologic examination that included tuning fork tests and otoscopy to rule out middle ear disease.

Pure tone audiometry was done for each ear using a clinical audiometer AC 33 with supra aural earphones in a sound proof booth at the ENT department at the following frequencies: 250, 500, 1000, 2000, 4000, 6000 & 8000HZ. Pure tone audiometry was conducted by one trained audiologist to avoid inter-personal bias.

Participants were classified as having low/mid frequency hearing impairment if the average of the pure tone thresholds measured at 250, 500, 1000 & 2000 HZ in either ear exceeded 25dbHL. Participants were classified as having high frequency hearing impairment if the pure tone average measured at 4000, 6000 & 8000 HZ in either ear exceeds 25 dbHL.

ABR was recorded in a sound proof booth at the Nairobi Audiology center by a single trained audiologist. The Nairobi Audiology center is a private institution to which patients from KNH requiring ABR services are currently referred to. The Nairobi Audiology center is located on the 1st floor of Landmark Plaza along Argwings Kodhek road approximately one kilometer from KNH. Study participants were transported by the principal investigator to the site at no extra cost. ABR was recorded by placing active electrodes positioned at vertex and reference electrodes at each mastoid. A stimulus at a supra-threshold level of 80 dB was generated by using a 100 microsecond pulse. The equipment used for ABR was IHS – BERA. Two recordings were utilized.

Weight was measured using a SECA 799 electronic column scale that was calibrated daily, the scale has an attached measuring rod for measuring height, and this allowed both height and weight to be determined in one step.

Venous blood samples was collected by a trained phlebotomist and analysed at the KNH clinical chemistry laboratory number 16. Approximately 5 millilitres of blood was collected for analysis of glycosylated haemoglobin (HbA1c). Fasting blood glucose was analysed from a pin prick blood sample and measured in mmol/l using an Accu – Chek Aviva glucometer

3.5 DATA MANAGEMENT

Data was collected on questionnaires and entered into Microsoft Excel worksheets which were then be transferred to SPSS for analysis. All data was cleaned (including checks for completeness and consistency) before commencing analysis. All questionnaires and informed consent forms were stored securely in a lockable drawer in the University of Nairobi ENT department. Soft copy versions of the data were stored in a password protected laptop. The data was accessible only to the principal investigator

3.6 DATA ANALYSIS

The variables that were analysed were audiometry measures including right ear low frequency, right ear high frequency, left ear low frequency and left ear high frequency; gender; age; other

factors including anthropometric measures such as body mass index, waist circumference, glycosylated haemoglobin, fasting blood sugar and wave amplitude and inter-wave latencies. The outcome measures were the audiometry measures. Gender was the only binary variable. The continuous variables were converted to categorical variables for simplicity during univariate analysis. The dataset had no missing variables. All the data was used to conduct the analysis.

3.7 ETHICAL CONSIDERATIONS

The study was carried out after obtaining ethical approval from KNH – UON ethics and research committee (P704/11/2015 – appendix 4)

Results of the study will be published and made available to members of the medical fraternity

CHAPTER 4: RESULTS

a) Gender and Age characteristics

The sample had 44.9% (n=35) male participants and 55.1% (n=43) female participants. The study sample had a mean age of 43.9 years with a standard deviation of 8.3 and ranging from 22 to 55 years.

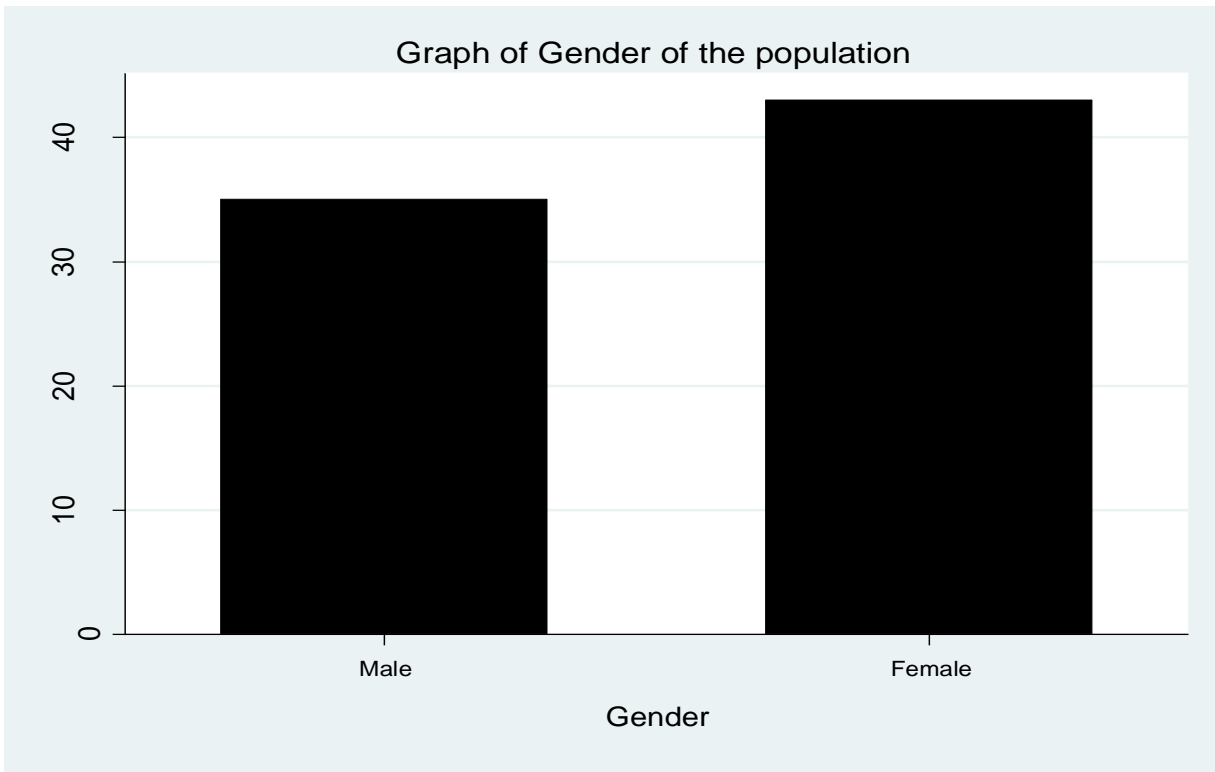


Figure 1: Graph of Gender distribution in the study population

Prevalence Of Sensorineural Hearing impairment in the Study Population

The crude prevalence rate of hearing loss was found to be 39.7%. The WHO audiometric descriptor was used to assess the hearing level. Patients with slight sensorineural hearing loss accounted for most of the patients with hearing loss (90.3%). No patient had severe or profound hearing loss. Amongst the patients with hearing impairment, high frequency hearing loss was a lot more prevalent.

Table 1:

Audiometry Outcome Measure	Audiometry Categories	Audiometry status frequency (Percentage)	Mean (Standard Deviation) in dbHL	Range
Right low frequency	Normal Hearing	72 (92.31%)	15.47 (5.79)	5 – 30
	Slight Hearing impairment	6 (7.69%)		
Right high frequency	Normal Hearing	55 (70.51%)	19.95 (9.31)	5 – 50
	Slight Hearing impairment	21 (26.92%)		
	Moderate hearing impairment	2 (2.56%)		
Left low frequency	Normal Hearing	73 (93.59%)	16.12 (6.41)	5 – 38
	Slight Hearing impairment	5 (6.41%)		
Left high frequency	Normal Hearing	51 (65.38%)	20.29 (10.69)	5 – 56
	Slight Hearing impairment	24 (30.77%)		
	Moderate hearing impairment	3 (3.85%)		

Symmetry of Hearing Loss

A t-test was used to compare the means of audiometry outcome measures and thus assess the asymmetry of hearing loss in this sample. The Right high frequency mean was compared to the left high frequency mean while the right low frequency mean was compared to the left low frequency mean.

The t test results for the comparison between the right high frequency and left high frequency had a p-value of 0.6467, a mean difference of -0.35, and a 95% confidence interval of -2.17 to 1.48. The data showed that in the population, there was very little evidence against the

hypothesis that the right high frequency and left high frequency have different means. This therefore implies that the two means are equal thus symmetrical.

The t test results for the comparison between the right low frequency and left low frequency had a p-value of 0.8508, a mean difference of -0.64, and a 95% confidence interval of -1.86 to 0.58. The data showed that in the population, there was very little evidence against the hypothesis that the right low frequency and left low frequency have different means. This therefore implies that the two means are equal thus symmetrical.

ABR WAVE LATENCIES

The ABR latency values for the left and right ears are shown in the tables below. A t-test was further used to compare this values against the normative results. All the latency values yielded a p-value of 0.001 except interpeak latency values I-III and I-V. Therefore, there is very strong evidence against the hypothesis that the values in this sample are equal to the normative values. This therefore means that the results from this sample are not similar to normative results. However, in the case of interpeak latency I-III, there is very little evidence (p=0.865) against the hypothesis that the values in this sample are equal to the normative values while in the case of I-V (p=0.123) there is weak evidence against the hypothesis that the values in this sample are equal to the normative values.

Table 2: Left and Right ear latency measures

Latency (msec)	I.	III.	V.	I-III	III-V	I-V
Left Ear						
Mean(SD)	.67(.26)	2.87(.43)	4.64(.60)	2.20(.40)	1.77(.36)	3.97(.54)
Range	.20 – 1.73	1.67 – 3.73	2.8 – 5.47	1.13 – 2.8	.87 – 2.63	2.2 – 4.73
Right Ear						
Mean(SD)	.67(.21)	2.88(.40)	4.61(.61)	2.21(.40)	1.73(.32)	3.93(.61)
Range	.27 – 1.27	1.60 – 3.47	2.67 – 5.47	1.0 – 2.87	.87 – 2.27	2.0 – 5.00
Comparison against the normative values*	1.54	3.70	5.60	2.20	1.84	4.04

p-values	0.001	0.001	0.001	0.865	0.003	0.123
95% CI	0.62 – 0.72	2.79 – 2.97	4.47 – 4.75	2.12 – 2.30	1.66 – 1.80	3.80 – 4.07

*reported by Schwartz, Pratt, and Schwartz (1990). Subjects: 20 subjects; 10 male, 10 female; age range of 19 to 36 years (mean age of 26 years); hearing threshold criteria not specified. Intensity level 80dB nHL; stimulus rate was not specified; repetitions, 2000 – 4000Hz. Acquisition-band pass filters.

RISK FACTORS

The anthropometric risk factor measures were body mass index, waist circumference, glycosylated haemoglobin, fasting blood sugar. The fasting blood sugar had a mean of 6.98 (standard deviation 2.02) and ranging from 2.2 to 14 mmol/l. The waist circumference ranged from 70 cm to 118 cm with a mean of 90.13 cm and standard deviation of 10.08 cm. The BMI and glycosylated haemoglobin are shown in table 3 below.

Table 3: The risk factors

Risk factor	Categories	Frequency (Percentage)
Body Mass Index	Underweight (< 18.0)	3 (3.85%)
	Normal weight (18.1 – 24.9)	28 (35.90%)
	Overweight (25.0 – 29.9)	30 (38.46%)
	Obese (>30)	17 (21.79%)
Waist Circumference	High risk men (>120cm)	0 (0%)
	Low risk men (<120cm)	35 (100%)
	High risk women (>88cm)	32 (71%)
	Low risk women (<88cm)	11 (25.58%)
Glycosylated haemoglobin	Good glycaemic control (<7%)	43 (55.13%)
	Poor glycaemic control (>7%)	35 (44.87%)
Fasting blood sugar	Low (<4 mmol/l)	1 (1.28%)

	Normal (4 – 7 mmol/l)	45 (57.69%)
	High (>7 mmol/l)	32 (41.03%)

ASSOCIATIONS

Unadjusted odds ratios and chi-square tests were used to assess the association between hearing loss (outcome) and other independent variables (gender; comorbidities and treatments; other factors including anthropometric measures such as body mass index, glycosylated haemoglobin, and fasting blood sugar.)

Of the 78 patients in the study, 31 (39.7%) had hearing loss. There was strong evidence ($p=0.002$) of association between hearing loss and body mass index (OR presented in table 4 is stratum specific, pooled OR=0.33, 95% CI 0.02 – 4.95). There was weak evidence of association between hearing loss and hypertension (OR=2.45, 95% CI 0.94 – 6.44, $p= 0.058$). There was very little evidence of association between hearing loss and type of medication oral hypoglycaemics and insulin (OR=1.12, 95% CI 0.41 – 3.01, $p= 0.736$), sex (OR=1.22, 95% CI 0.28 – 3.06, $p= 0.672$), oral hypoglycaemics only (OR=1.17, 95% CI 0.46 – 2.98, $p= 0.736$), Glycosylated haemoglobin (OR=1.27, 95% CI 0.51 – 3.17, $p= 0.612$) and fasting blood sugar (OR= 0.63, 95% CI 0.25 – 1.58, $p=0.434$). **(Table 4)**

Table 4: Association of hearing loss with members' characteristics

	<u>Categories</u>	<u>No, did not have hearing loss</u> <u>N=47</u>	<u>Yes, Had hearing loss</u> <u>N=31</u>	<u>Odds ratio of hearing loss</u> <u>(95% CI)</u>	<u>P-values</u>
<u>Comorbidities hypertension</u>	<u>No</u>	<u>27 (71.05%)</u>	<u>11 (28.95%)</u>	<u>1.00(Reference)</u>	<u>0.058</u>
	<u>Yes</u>	<u>20 (50.00%)</u>	<u>20 (50.00%)</u>	<u>2.45(0.94, 6.44)</u>	
<u>Sex</u>	<u>Male</u>	<u>22 (62.86%)</u>	<u>13 (37.14%)</u>	<u>1.00(Reference)</u>	<u>0.672</u>
	<u>Female</u>	<u>25 (58.14%)</u>	<u>18 (41.86%)</u>	<u>1.22(0.48, 3.06)</u>	
<u>Oral hypoglycaemics and Insulin</u>	<u>No</u>	<u>20 (62.50%)</u>	<u>12 (37.50%)</u>	<u>1.00(Reference)</u>	<u>0.736</u>
	<u>Yes</u>	<u>27 (58.70%)</u>	<u>19 (41.30%)</u>	<u>1.12 (0.41, 3.01)</u>	
<u>Oral Hypoglaecaemics only</u>	<u>No</u>	<u>20 (62.50%)</u>	<u>12 (37.50%)</u>	<u>1.00(Reference)</u>	<u>0.736</u>
	<u>Yes</u>	<u>27 (58.70%)</u>	<u>19 (41.30%)</u>	<u>1.17 (0.46, 2.98)</u>	
<u>Body Mass Index</u>	<u>Underweight (< 18.0)</u>	<u>2 (66.67%)</u>	<u>1 (33.33%)</u>	<u>1.00(Reference)</u>	<u>0.002</u>
	<u>Normal weight (18.1 – 24.9)</u>	<u>24 (85.71%)</u>	<u>4 (14.29%)</u>	<u>0.33(0.02, 4.59)</u>	
	<u>Overweight (25.0 – 29.9)</u>	<u>11 (36.67%)</u>	<u>19 (63.33%)</u>	<u>3.45 (0.28, 42.62)</u>	
	<u>Obese (>30)</u>	<u>10 (58.82%)</u>	<u>7 (41.18%)</u>	<u>1.4 (0.11, 18.61)</u>	
<u>Glycosylated haemoglobin</u>	<u>Good glycaemic control (<7%)</u>	<u>27 (62.79%)</u>	<u>16 (37.21%)</u>	<u>1.00(Reference)</u>	<u>0.612</u>
	<u>Poor glycaemic control</u>	<u>20 (57.14%)</u>	<u>15 (42.86%)</u>	<u>1.27 (0.51, 3.17)</u>	

	(<u>>7%</u>)				
<u>Fasting blood sugar</u>	<u>Low (<4 mmol/l)</u>	<u>1 (100.00%)</u>	<u>0 (0.00%)</u>	<u>1.00(Reference)</u>	<u>0.434</u>
	<u>Normal (4 – 7 mmol/l)</u>	<u>29 (64.44%)</u>	<u>16 (35.56%)</u>	<u>0.63 (0.25, 1.58)</u>	
	<u>High (>7 mmol/l)</u>	<u>17 (53.13%)</u>	<u>15 (46.88%)</u>	<u>1.00 (Omitted because of collinearity)</u>	

CHAPTER 5: DISCUSSION

The prevalence rate of sensorineural hearing impairment among type 2 diabetics was found to be 39.7% which was slightly lower than what Akinpelu found in a systematic review in 2014 showing the prevalence ranging from 44% to 69.7% (32). However, the rate we found was higher than the prevalence of 21.6% found in a recent study in a tertiary health institution in Ogbomoso, Nigeria (53). A study by Mozzafari et al among diabetics less than 60 years found a 45% prevalence of sensorineural hearing loss (33).

It is interesting to note that the vast majority of patients with hearing loss (90.3%) had a mild hearing loss. The higher frequencies of 4 – 8 kHz were the most affected and this was similar to the findings by Cullen and Cinnamond (8). This is important because most cases of mild hearing impairment may not produce sufficient clinical symptoms to warrant aggressive treatment. This implies that the possibility of having hearing loss impact the quality of life of type 2 diabetics is slight. However, these mild degrees of hearing loss may be easily worsened when superimposed upon by other conditions that affect the hearing organ.

The bilaterally symmetrical high frequency sensorineural hearing impairment is in keeping with histological findings in the inner ear of type 2 diabetics. A post mortem examination of 8 temporal bones of diabetics who had been known to have worse hearing as compared to age and sex matched controls revealed microangiopathy. Makashima and Tanaka found atrophy of spiral ganglia in the basal to middle turn of the cochlear (54)

We could not corroborate reports (7, 34) indicating that audiometric thresholds are correlated with poor glycaemic control. There was a strong association between hearing loss and BMI (OR 0.33, 95% CI 0.02 – 4.95) and a slight association with co-morbid hypertension (OR=2.45, 95%

CI 0.94 – 6.44, P=0.058), cardiovascular disease and its risk factors have been linked to hearing impairment in the general population (37, 38).

ABR WAVE LATENCIES

The absolute wave latencies I, III, V were significantly shorter as compared to normative values as reported by Schwartz, Pratt and Schwartz (55) P value = 0.001. This is different from the reports of Durmus who found prolonged absolute latencies (48), and Gupta who found the latency of waves III & V were prolonged and wave I was equal in diabetics and control (50).

The interpeak latencies I – III, III – V, and I – V were similar when compared to normative values P values 0.865, 0.003, 0.123 respectively.

The shortened absolute latencies could be accounted for by the normative data set used from a Caucasian population and differing laboratory conditions. A study by Zakaria MN et al in Malaysia showed differences in the ABR wave latencies and amplitudes of ethnic Malay and Chinese when compared to Caucasian normative data sets (56).

CONCLUSION

The overall prevalence of sensorineural hearing impairment in type 2 diabetics was found to be 39.74%. Majority of the patients had mild hearing loss (90.3%) and was more prevalent in the higher frequencies (4 – 8 kHz). These results are comparable to most studies.

The absolute wave latencies I, III & V were significantly shorter when compared to adult normative ABR data (P value = 0.001) and this differed from most other studies that found a prolongation of the latencies.

STUDY LIMITATIONS

It was not possible to completely rule out hearing loss from causes such as noise exposure and ototoxic medication especially as the feasibility of getting records of prior ototoxic medication usage was impossible.

The cost and added complexity of establishing a local normative data set for ABR latencies was a huddle this study could not surmount.

RECOMMENDATIONS

As the hearing loss associated with type 2 diabetes mellitus was shown to be mild, in a country such as Kenya where audiometry resources and qualified audiologists are scarce, the available evidence we have as of now doesn't justify the added cost to advocate for routine hearing assessment in patients with type 2 diabetes mellitus.

A study should be done to establish normative ABR data amongst Kenyans for both adults and infants to be used by the various audiology facilities within the country.

WORK PLAN

PERIOD	ACTIVITY
January 2015 – June 2015	Proposal writing
July 2015	Proposal presentation
January 2016	Ethical approval
February 2016 – April 2016	Data collection and analysis
May 2016	Report writing and submission

BUDGET

ITEM	AMOUNT (KSHS)
ABR @ 5,000	400,000
PTA @ 700	56,000
STATIONARY	5,000
PRINTING & BINDING	5,000
TRANSPORT	15,000
STATISTICIAN	30,000
DISSEMINATION OF RESULTS	15,000
TOTAL	526,000

REFERENCES

1. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of diabetes mellitus. Geneva, World Health Organization, 1999 (WHO/NCD/NCS/99.2)
2. UK prospective diabetes study (UKPDS) Group. Intensive blood glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet* 352, 837-853 (1998)
3. Wei, M., Gaskill, S. P., Haffner, et al. Effects of diabetes and level of glycaemia on all-cause and cardiovascular mortality. The San Antonio Heart Study. *Diabetes care* 7, 1167-1172 (1998)
4. Brown Lee M. Biochemistry and molecular cell biology of diabetic complications. *Nature* 2001; 414: 813-820
5. Wackym P, Linthicum FJ. Diabetes mellitus and hearing loss: clinical and histopathological relationships. *Am J Otol* 1986; 7: 176-182.
6. Costa OA. Inner ear pathology in experimental diabetes. *Laryngoscope* 1967; 77: 68-75
7. Kurien M , Thomas K, Bhanu T. hearing thresholds in patients with diabetes mellitus. *J laryngol otol* 1989; 103: 164 – 168.
8. Cullen J, Cinnamon M. hearing loss in diabetics. *J laryngol otol* 1993; 107: 179 – 182.
9. Fukushima J, Cureolglu S, Schapern PA, et al. Effects of type 2 diabetes on cochlear structure in humans. *Arch Otolaryngol Head Neck Surg* 2006; 132:934-938
10. Pickles JO. An introduction to the physiology of hearing, 2nd ed. New York: Academic press, 1988

11. Ishii EK, Talbott EO, Findlay RC, D'Antonio JA, Kuller LH. Is NIDDM a risk factor for noise induced hearing loss in an occupationally noise exposed cohort? *Sci Total Environ* 1992; 127:155-165
12. Ciorba A, Benatti A, Bianchini C, et al. High frequency hearing loss in the elderly: effect of age and noise exposure in an Italian group. *J Laryngol Otol* 2011;125:776-780
13. Gordon-Salant S. Hearing loss and aging: new research findings and clinical implications. *J Rehabil Res Dev* 2005;42:9-24.
14. Sha SH, Kanicki A, Dootz G, et al. Age-related auditory pathology in the CBA/J mouse. *Hear Res* 2008;243: 87-94.
15. Gratton MA, Schmiedt RA, Schulte BA. Age related decreases in endocochlear potential are associated with vascular abnormalities in the stria vascularis. *Hear Res* 1996;102: 181-190
16. [Huang](#) Q, Tang J. Age-related hearing loss or presbycusis. *Eur Arch Otorhinolaryngol* 2010;267:1179-1191
17. Nakae S, Tachibana M. The cochlea of the spontaneously diabetic mouse. II. Electron microscopic observations of non-obese diabetic mice. *Arch Otorhinolaryngol* 1986;243: 313-316.
18. Pauler M , Schuknecht HF, Thornton A. Correlative studies of cochlear neuronal loss with speech discrimination and pure tone thresholds, *Arch Otolaryngol* 1986; 243:200-206
19. [Helfert](#) RH, Snead CR, Altschuler RA. The ascending auditory pathways in: Altschuler RA, Bobbin RP, Clopton BM, Hoffman D, eds. *Neurobiology of hearing: the central auditory system*. New York: Raven press,1991: 1-25 .
20. Sohmer H, Feinmesser M: Cochlear action potentials recorded from the external ear in man. *Ann Otol Rhinol Laryngol* 1967; 76:427-436.

21. Moller AR, Jannetta PJ: *Neural generators of the auditory brainstem response*. In: Jacobson JT, ed. *The auditory brain stem response*, San Diego, California: College-Hill Press Inc; 1985.
22. Starr A, Achor LJ: Auditory brain stem responses in neurological disease. *Arch Neurol* 1975; 32:761-768.
23. Hyde ML: *The effect of cochlear lesions on the ABR*. In: Jacobson JT, ed. *The auditory brain stem response*, San Diego, California: College-Hill Press Inc; 1985:133-146.
24. Stockard JE, Westmoreland BF: Technical considerations in the recording and interpretation of the brain stem auditory evoked potential for neonatal neurologic diagnosis. *Am J EEG Tech* 1981; 21:31-54.
25. Lisowska G, Namyslowski G, Morawski K, Strojek K. Cochlear dysfunction and diabetic microangiopathy. *Scand Audiol* 2001; 30(suppl 52): 199-203.
26. Christensen DL et al.: prevalence of glucose intolerance and associated risk factors in rural and urban populations of different ethnic groups in Kenya. *Diabetes Res clin Pract* 2009, 84(3): 303-10.
27. Mathenge W, Foster A, Kuper H. Urbanization, ethnicity and cardiovascular risk in a population in transition in Nakuru, Kenya: a population based survey *BMC public health*.2010;10:569
28. Ayah R, Joshi MD, Otieno CF, et al. A population based survey of diabetes and correlates in an urban slum community in Nairobi, Kenya. *BMC public health* 2013;13:371
29. WHO report of the informal working group on prevention of deafness and Hearing impairment programme planning. Geneva 1991
30. Bainbridge KE, Hoffman HJ, Cowle CC. Diabetes and hearing impairment in the United States: audiometric evidence from the National Health and Nutrition Examination survey, 1999 to 2004. *Ann intern Med* 2008; 149: 1-10.

31. Cheng YJ, Gregg EW, Saaddine JB, et al. Three decade change in the prevalence of hearing impairment and its association with diabetes in the United States. *Prev Med* 2009;49: 360-364
32. Olubunmi V. Akinpelu, Mario Mujica-Mota. Is type 2 diabetes mellitus associated with alterations in hearing? A systematic review and Meta-analysis. *Laryngoscope*, 124:767-776,2014
33. Mozaffari M, Tajik A, Ariaei N, Ali Ehyaii F, Benham H. Diabetes Mellitus and sensorineural hearing loss among non-elderly people. *East Mediterr Health J* 2010;16:947-952
34. Celik O, Yalcin S, Celebi H, Ozturk A. Hearing loss in insulin dependent diabetes mellitus. *Auris Nasus Larynx* 1996; 23; 127-132
35. Parving A, Elberling C, Balle V, Parbo J, Dejgaard A, Parving HH. Hearing disorders in patients with insulin dependent diabetes mellitus. *Audiology* 1990; 29: 113-121
36. Ferrer JP, Biurrun O, Lorente J, et al. Auditory function in young patients with type 1 diabetes mellitus. *Diabetes Res Clin Pract* 1991; 11: 17-22
37. Gates GA, Cobb JL, D'Agostino RB, Wolf PA. The relation of hearing in the elderly to the presence of cardiovascular disease and cardiovascular risk factors. *Arch Otolaryngol Head Neck Surg* 1993;193: 156 -161
38. Cruickshanks Kj, Klein R, Klein BE, Wiley TL, Nondahl DM, Tweed TS. Cigarette smoking and hearing loss: the epidemiology of hearing loss study. *JAMA* 1998; 279:1715-1719
39. Dalton DS, Cruickshanks KJ, Klein R, Klein BE, Wiley TL. Association of NIDDM and hearing loss. *Diabetes care* 1998; 103: 164-168
40. Friedman S, Schulman R, Weiss S. hearing and diabetic neuropathy. *Arch intern med* 1975; 135: 573-576
41. Duck SW, Prazma J, Bennett PS, Pillsbury HC. Interaction between hypertension and diabetes mellitus in the pathogenesis of sensorineural hearing loss. *Laryngoscope* 1997; 107(12 pt 1): 1596 -1605
42. Kakarlapudi V, Sawyer R, Staecker H. the effect of diabetes on sensorineural hearing loss. *Otol neurotol* 2003;24:382-386

43. Feng Chin Shen, Ching-Jung Hsieh. Severity of hearing impairment is positively associated with urine albumin excretion rate in patients with type 2 diabetes. *J Diabetes invest* 2014; 5: 743-747
44. The effect of intensive treatment of diabetes on the development and progression of long term complications in insulin dependent diabetes mellitus. The Diabetes Control and Complications Trial Research group. *N Engl J Med.* 1993 Sep 30. 329(14): 977-986
45. Nathan DM, Cleary PA, Backlund JY, Genuth SM, Lackin JM, Orchard TJ, et al. intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. *N Engl J Med.* 2005 Dec 22. 353(25): 2643-53
46. Genuth S. insight from the diabetes control and complications trial/ epidemiology of diabetes interventions and complications study on the use of intensive glycaemic treatment to reduce the risk of complications of type 1 diabetes. *Endocr pract.* 2006 Jan – Feb. 12 suppl 1:34-41
47. Panchu P. Auditory acuity in type 2 diabetes mellitus. *Int J Diabetes Dev Ctries* 2008;28:114-120.
48. Durmus C, Yetiser S, Durmus O. Auditory brainstem evoked responses in Insulin dependent and non-insulin-dependent diabetic subjects with normal hearing. *International J of Audiology* 2004;43(1);29-33.
49. Toth et al, Brainstem auditory evoked potential examinations in diabetic patients, *Scandinavian audiology*2001;30:156-159.
50. Gupta R, Aslam H, Hasan S, Siddiqi S. Type 2 diabetes mellitus and auditory brainstem responses – a hospital based study. *Indian J Endocrinol Metab* 2010;14:9-11.
51. Kirkwood BR, Sterne JA. *Essentials of Medical Statistics.* 2nd Edn. London: Blackwell scientific Publications, pp 131 – 137, 413 – 428.
52. World Health Organization. *The Global Burden of Disease: 2004 Update.* Geneva: World Health Organization; 2008.
53. Adebola SO, Olamoyegun MA, Sogebi OA et al. Otologic and audiological characteristics of type 2 diabetics in a tertiary health institution in Nigeria. *Braz J otorhinolaryngol.* 2016 Feb 10. Pii:51808-8694[Epub ahead of print]

54. Makishima K, Tanaka K. Pathological changes of the inner ear and central auditory pathways in diabetics. *Ann Otol Rhinol Laryngol.* 1971; 80: 218-88
55. Schwartz DM, Morris MD & Schwartz JA. (1990). Influence of click polarity on the brainstem auditory evoked response (BAER) revisited. *Electroencephalography and clinical neurophysiology*, 77, 445-457
56. Zakaria MN, Jalaei B, Aw CL et al. Are speech evoked auditory brainstem response outcomes influenced by ethnicity? *Neurol sci.* 2016 Feb 26 [Epub ahead of print]

APPENDIX

APPENDIX 1: CONSENT INFORMATION DOCUMENT

TITLE: Prevalence and pattern of sensorineural hearing impairment among patients with type 2 diabetes mellitus at the Kenyatta National Hospital.

INTRODUCTION: My name is Dr. Neville Okwiri; I am pursuing a degree of Masters of Medicine in Ear, Nose, Throat, Head & Neck surgery at the University of Nairobi.

I would like to seek your permission to participate in the study as titled above. Kindly read the information provided. You are free to discuss this with family and friends and I am willing to answer any questions raised.

OBJECTIVES OF THE STUDY: TO determine the prevalence and pattern of sensorineural hearing impairment among patients with type 2 diabetes mellitus at the Kenyatta National hospital.

HOW YOU WILL PARTICIPATE: Your role in this study, should you agree to participate will be as follows. We will ask you questions and review your medical records seeking to know when the condition was diagnosed and current treatment. A comprehensive otologic examination will be carried out. A blood and urine sample will be obtained, as part of the comprehensive care of diabetics these are routine tests that are done at KNH even if one wasn't participating in the study. Minimal pain and discomfort will be experienced at the time of drawing the sample.

You will undergo a screening hearing test at the KNH ENT audiology unit and a specialized auditory brainstem response testing at Nairobi audiology centre located one kilometer from KNH. We will bear the cost of testing and transportation. Similar findings from all participants will be used for analysis.

BENEFITS: You may not accrue direct benefit by participating in this study, but your participation will greatly contribute to our better understanding of the effects that diabetes has on hearing.

RISKS: Participation in this study is expected to have no risk. Your choosing or declining to participate in this study will NOT have any impact on the quality of care that you will receive.

VOLUNTARISM: Participation in this study is voluntary.

RIGHTS OF STUDY PARTICIPANTS: You are at liberty to decline participation or withdraw from the study at any point.

COMPENSATION: No monetary or material compensation will be offered to study participants.

CONFIDENTIALITY: Strict confidentiality will be maintained at all times. Your name and test results will not be linked and no single test result will be reported on its own but as a summation of all the results.

If you understand everything said and are willing to participate kindly sign the consent form provided.

If you have any questions or need further clarifications about the study contact the principal investigator, *Dr. Neville Okwiri* on phone number *0733-954411*.

If you have any questions on your rights as a participant contact the *Kenyatta National Hospital Ethics and Research Committee (KNH-ERC)* by calling *2726300 Ext. 44355*.

KIAMBATISHO 1: FOMU YA MAELEZO KUHUSU IDHINI YA MGONJWA

KICHWA: Idadi na ruwaza ya upungufu wa kusikia miongoni mwa wagonjwa na kisukari aina 2 katika hospitali kuu ya Kenyatta.

KIINGILIO: Jina langu ni Daktari Neville Okwiri mwanafunzi wa Shahada ya Uzamili wa upasuaji wa masikio, mapua na koo.

Ningependa kuchukua idhini au ruhusa kwako kushiriki katika utafiti. Tafadhali soma maelezo yafuatayo, ukihitaji kushauriana na jamaa na familia unauhuru wa kufanya hivyo na niko tayari kujibu maswali yoyote.

LENGO LA UTAFITI: Kutathmini idadi na sababu zinazo changia kwa upungufu wa kusikia miongoni mwa wagonjwa na kisukari aina 2 katika hospitali kuu ya Kenyatta.

JINSI UTASHIRIKI: Utapimwa kusikia kwako. Kipimo cha auditory brainstem response kitafanywa katika maabara ya kibinafsi ya Nairobi audiology centre. Gharama ya usafirishaji pamoja na vipimo zita dhaminiwa na mtafiti mkuu. Tutakuuliza maswali pamoja na kuchambua rekodi zako za hospitali kuhusu malalamiko, hali ya afya, na matibabu.

FAIDA: Habari itakayotokea na utafiti huu peingine haitakufaidi binafsi lakini itatupa maarifa ambayo itaboresha matibabu wa ugonjwa hii siku zijazo.

ATHARI: Hakuna hatari yoyote itakayo jiri kwa kushiriki au kutoshiriki.

HIARI: kushiriki katika utafiti huu ni kwa hiari yako.

HAKI: Uko uhuru kutoshiriki au kujiondoa kwa utafiti huu wakati wowote ule.

FIDIA: Hakuna pesa au chochote kile kitapewa kwa washiriki wa utafiti huu

USIRI: usiri utahakikishwa wakati wote.

Kama umeridhika na maelezo, na uko tayari kushiriki, tafadhali weka sahihi yako kwenye fomu ya idhini.

Ikiwa una swali ama ungetaka kupata maelezo zaidi kuhusu utafiti huu, wasiliana na mtafiti mkuu *Daktari Neville Okwiri* kupitia nambari ya simu 0733-954411 au KNH-ERC kupitia nambari ya simu 2726300 Ext. 44355.

APPENDIX 2: CONSENT FORM

STUDY NUMBER _____

I Mr. / Mrs. / Miss _____ hereby agree to enroll myself into this study as explained to me by Dr. Neville Okwiri

My signature is confirmation that I have understood the nature of the study and that whatever information I give will remain confidential.

I also confirm that no monetary or material gains have been promised or given to me for participating in the study.

Signed _____ Date _____

Signature of principal investigator _____

Date _____

KIAMBATISHO 2: KIBALI CHA UTAFITI

NAMBARI YA UTAFITI _____

Mimi Bi / Bwana _____ nimekubali kushiriki katika utafiti huu baada ya kuelezwa na daktari Neville OKwiri

Sahihi yangu ni thibitisho ya kwamba nimeelewa umuhimu wa utafiti huu na kwamba habari yoyote nitakayotoa itawekwa siri.

Pia nathibitisha ya kwamba sijapewa au kuahadiwa pesa au chochote kile, kushiriki kwenye utafiti huu. Sahihi _____ tarehe _____

Appendix 3: study proforma

STUDY NUMBER _____

1. BIODATA

Age _____

Sex M _____ F _____

2. CLINICAL

- Duration of illness _____
- Type of medication

Diet modification ONLY	
Oral hypoglycaemics ONLY	
Insulin ONLY	
Oral hypoglycaemics & Insulin	

- Co – morbidities

	YES	NO
Hypertension		
Chronic kidney disease		
Stroke		
Heart disease		
Foot ulcer/ amputation		

3. CLINICAL EXAMINATION

- Tuning fork tests: Rinne's left _____ Rinne's right _____ Weber _____
- PTA findings : Degree of Hearing loss (WHO audiometric descriptor²⁷)

Normal (<25dbHL)		
Slight (25 – 40dbHL)		
Moderate (41 -60 dbHL)		
Severe (61 – 80 dbHL)		
Profound (>81 dbHL)		

- Anthropometric measures

Weight	
Height	
BMI	
Waist circumference	

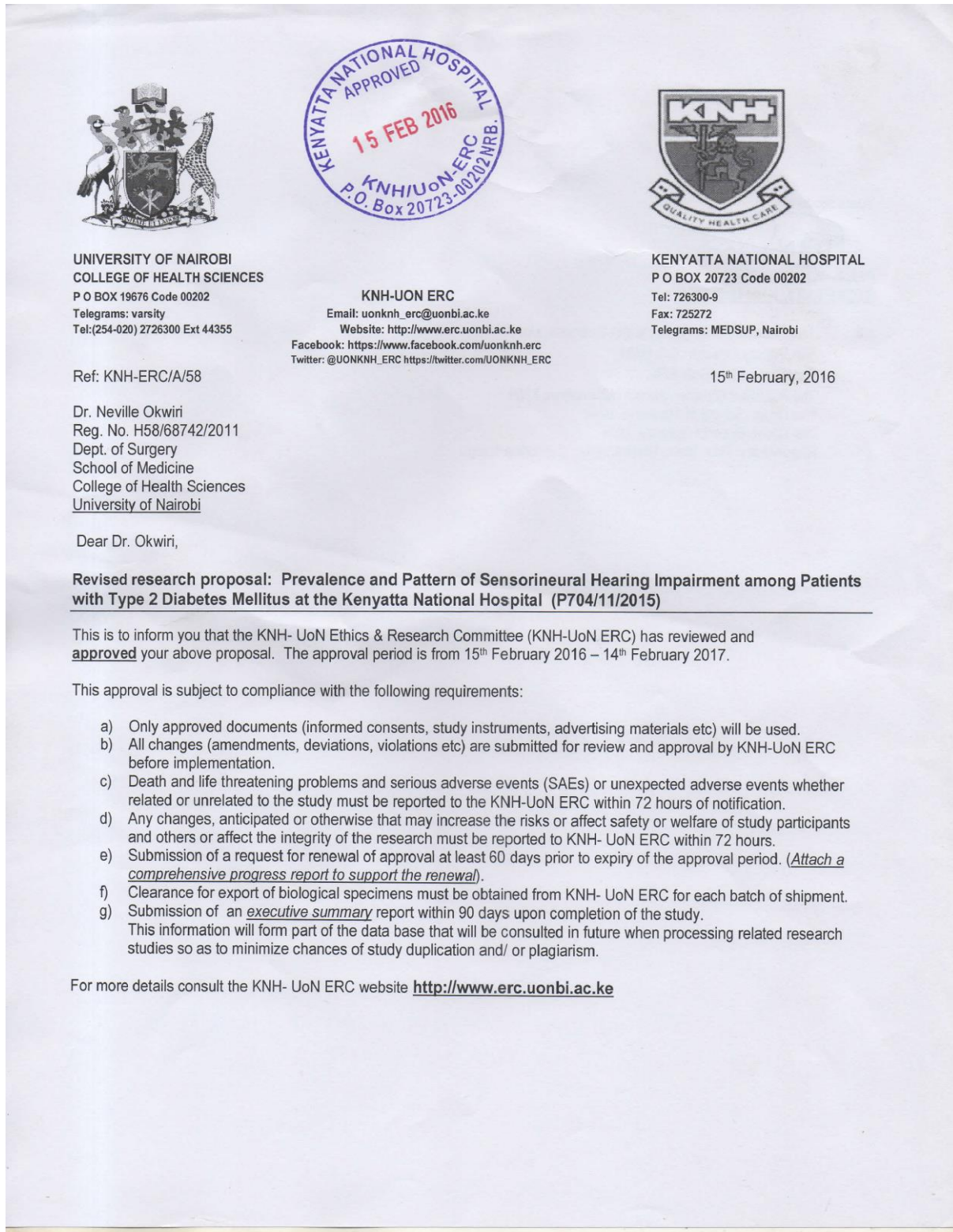
4. LABORATORY MEASURES

Fasting blood sugar	
HbA1c	
Urinalysis	
Serum creatinine	
EGFR	

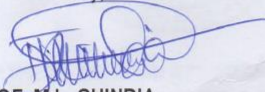
5. AUDITORY BRAINSTEM RESPONSE

Wave	Intensity (in dB)	Latency (msec)	morphology
I			
III			
V			
I - III			
III - V			
I - V			

Appendix 4: Ethical approval



Yours sincerely,



PROF. M.L. CHINDIA
SECRETARY, KNH-UoN ERC

- c.c. The Principal, College of Health Sciences, UoN
 The Deputy Director, CS, KNH
 The Chair, KNH-UoN ERC
 The Assistant Director, Health Information, KNH
 The Dean, School of Medicine, UoN
 The Chair, Dept. of Surgery, UoN
 Supervisors: Prof. Isaac Macharia, Dr. Catherine Irungu