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Identifying Diabetic Parameters in Cochlear Mechanics and Models

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Abstract

Hearing impairment is one of the most prevalent chronic disability identified with the elderly people over the age of 65.The peripheral portion of the cochlea and auditory nerve are known to deteriorate with age. The prevalence of hearing loss in diabetes has been shown in many studies to be moderately high, progressive and bilateral. The predominant mechanism of hearing loss in diabetes appears to be related to microangiopathy of inner ear. Mathematical modelling of cochlear dynamics had been attempted over the years with various parameters. This paper, attempts to study the correlation between diabetes and hearing loss and try to identify key parameters in cochlear model which leads to hearing loss.

Keywords

Cochlea, Auditory filters, Sensorineural hearing loss, basilar membrane, motility factor, WKBJ methods, tonotopic.

I. Introduction

I.I Problem

Hearing impairments can be categorized by their type as conductive, sensorineural, or both. Also, a hearing impairment may exist in only one ear (unilateral) or in both ears (bilateral). A conductive hearing impairment is an impairment resulting from dysfunction in any of the mechanisms that normally conduct sound waves through the outer ear, the eardrum or the bones of the middle ear. A sensorineural hearing impairment is one resulting from dysfunction in the inner ear, especially the cochlea where sound vibrations are converted into neural signals, or in any part of the brain that subsequently processes these signals [ii].The vast majority of human sensorineural hearing loss is associated with abnormalities in the hair cells of the organ of Corti in the cochlea. This dysfunction may be present from birth due to genetic or developmental abnormalities, or arise through trauma or disease during the lifetime of an individual. Damage to parts of the brain that process auditory signals can lead to a condition in which sounds may be heard at normal thresholds, but the quality of the sound perceived is so poor that speech cannot be understood. Sensorineural hearing loss associated with abnormalities of the auditory system in the brain is called Central Hearing Impairment [ii].

1.2 Hyperglycemia Condition:

Apart from the factors mentioned above the hearing acuity can be progressively made worse by presence of prolonged hyperglycemia condition [i]. Hearing aids are programmed for a hearing impaired person taking into the consideration the age dependent deterioration of cochlear mechanics and completely ignoring the progressive deterioration added by prolonged hyperglycemia condition. Our study focuses on effect of diabetes on hearing acuity, cochlear damage by diabetes and locating diabetes affected parameters in mathematical modeling of cochlea. Paper is organized in following manner

Section 2 describes the test performed to study auditory acuity in type 2 diabetes mellitus and its results [i]. It describes the evaluation of a cross section of hyperglycemia subjects with age and sex-matched normoglycemic controls pure tone audiometric and compare the differences. Particularly it highlights the progressive deterioration of diabetic threshold over a period.

Section 3 describes the test performed to study the effect of type 2 diabetes mellitus on cochlear elements of ear and its results [iii]. It describes the factors especially the thickening of basilar membrane (BM) and loss of outer hair cell which are affected by prolonged hyperglycemic condition. Hyperglycemia appears to have effect on hearing loss and the proposed mechanism of microangiopathy, neuropathy or combination of both.

Section4 describes the general mathematical modelling of cochlea and its parameters related to BM and OHC. It identifies the model parameters affected by prolonged hyperglycemic condition. Future work remained to be carried is to vary the parameters to construct the model simulation for hearing acuity for diabetic compensation in hearing aid.

II. Auditory Acuity in Diabetic Condition

Pure tone audiometric assessment performed reveals the relation between hearing loss and diabetes [i]. Tests performed in [i] and its results with conclusion are presented in this section. Subjects with hyperglycemia experience the higher threshold than corresponding control subjects. Tests were performed with audiometer [i]. An audiometer is an electronic device which produces pure tones, the intensity of which can be increased or decreased in 5-Db steps. The amount of intensity that has to be raised above the normal level is a measure of the degree of hearing impairment at that frequency. It is charted in form of a graph called the "audiogram." The thresholds of bone conduction are a measure of the cochlear function. The difference in the thresholds of air and bone conduction is a measure of a degree of conductive deafness.

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01 Sept 2014

Earphones were used to test hearing by air conduction and a small vibrator was placed over the mastoid was used test hearing by bone conduction. All audio meters incorporate a calibration circuit, which allows the output sound level to be set at a test frequency. The signals presented to the subject by an audiometer are characterized by its frequency, sound pressure level and wave form which are all controlled.

As shown in Table1, there was a significant difference in the auditory thresholds at all frequencies from 250Hz to 8000Hz between type 2 diabetic subjects and control group and all the hyperglycemic subjects showed sensorineural hearing loss changes on audiogram. The effect size was large to very large. The controls, all had normal hearing thresholds, where as the cases showed a gradual increase in hearing loss starting at 250Hz and becoming pronounced as the frequency increased. This difference is highly statistically significantat1% confidence interval[i].

Table 1: Effect of diabetes mellitus on auditory threshold in DB

Frequencies	Threshold in DB	Threshold in DB	Significance by student t
Hz	**Control**	**Cases**	
250	21.59 ± 3.48	29.33 ± 8.27	5.525
500	21.77 ± 3.37	31.83 ±6.85	80444
1000	20.49 ± 3.76	29.21 ±8.88	5.789
1500	19.27 ± 4.65	26.59± 8.32	4.913
2000	19.63 ± 3.64	29.82 ±8.78	6.886
3000	19.21 ± 4.62	27.59 ±8.65	5.468
4000	20.85 ± 4.62	34.21 ±9.96	7.951
6000	20.37 ± 3.69	36.46 ±10.98	8.897
8000	20.12 ± 3.75	35.24 ±12.39	7.476

It was observed that there was no statistically significant difference in auditory thresholds among type 2 diabetic patients and control group when analyzed according to their age groups, yet all type2 diabetic patients' auditory thresholds were higher than the control groups' thresholds.

As shown in Table2, from a frequency of 250Hz to 8000Hz, there was a significant difference between diabetic type2 patients with good control of their blood sugars [HbA1c values between 6% and 8%] versus type2 diabetic patients with poor control [HbA1c values greater than 8%]. The number of patients in the good control group was 20 and in the poor control group was 21. The significance levels are at 1% [i]

Table 2: Auditory threshold in DB HbA1C-wise

Frequencies	HbA1c	HbA1c	P value by
	(6%-8%)	(>8%)	student t
Hz	**Control**	**Cases**	
250	25.27±5.91	31.62±7.18	0.010
500	29.50±4.97	34.44±7.55	0.021
1000	26.13±6.31	32.78±9.69	0.016
1500	23.38±5.27	30.00±9.19	0.009
2000	26.38±7.28	33.78±8.41	0.006
3000	23.88±6.76	32.42±7.89	0.001
4000	30.63±8.91	39.44±8.93	0.004
6000	32.25±9.79	42.08±10.62	0.005
8000	29.88±11.2	41.94±11.59	0.002

Table 3 shows that there was no significant difference in the hearing thresholds between patients with short duration of type 2 diabetes [less than 10 years] versus long duration [greater than 10 years].

Table 3: Auditory threshold in duration-wise

Frequencies	Duration <10 years	Duration >10 years	P value by student t
Hz	**Control**	**Cases**	
250	27.73± 7.21	33.57± 7.34	.06
500	31.10 ±7.05	35.36 ±4.66	0.136
1000	28.60 ±8.9	32.14 ±8.83	0.343
1500	26.25 ±8.19	28.21 ±9.43	0.576
2000	29.56 ±8.36	31.07 ±11.07	0.682
3000	27.31± 8.42	28.93 ±10.29	0.658
4000	33.97 ±8.28	35.36 ±13.65	0.742
6000	35.36 ±10.32	40.36± 14.03	0.309
8000	34.71 ±11.99	37.86± 14.96	0.547

Table 4 shows that there is a trend towards a difference which is noted at higher frequencies (6000Hz and 8000Hz) when the effect of fasting blood sugar levels on auditory thresholds is considered [i].

Table 4: Auditory threshold in duration-wise

Frequencies	Blood sugar level	Duration >10 years	Significance
Hz			**ANOVA**
250	29.00 ±5.76	30.45 ±7.23	0.651
500	29.00 ±3.79	34.09 ±5.73	0.475
1000	29.00 ±8.22	31.59± 7.69	0.441
1500	27.00 ±4.81	28.18± 8.52	0.329
2000	31.00 ±6.02	32.77± 10.09	0.138
3000	26.00±2.82	31.14 ±7.77	0.125

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01 Sept 2014

4000	32.50 ±7.29	37.04 ±10.11	0.175
6000	32.00± 9.46	42.27 ±11.96	0.059
8000	33.00±11.51	40.45 ±12.29	0.090

Still the relationship between diabetes mellitus and hearing loss is controversial, because the pathogenic mechanism remains obscure. The variables influencing the auditory acuity statistically analyzed reveal the following:

1. Diabetes mellitus type2 raises auditory threshold in all frequencies between 250Hz and 8000Hz in all age groups in this study.
2. Patients with poor control [HbA1c greater than 8%] of their glycemic status have raised auditory thresholds.
3. The duration of diabetes does not affect auditory thresholds significantly in this study.

These results show the effect of hyperglycemia on auditory acuity may be explained by diabetic microangiopathy of the inner ear [iii].

III. COCHLEAR CHANGES DUE TO PROLONGED HYPERGLYCEMIA

The relationship between diabetes mellitus and sensorineural hearing loss has been studied for more than a century. Many authors agree that diabetes mellitus can lead to sensorineural hearing loss. Histopathology temporal bone studies in diabetic animal models have shown following reasons

1. Thickening of the basement membranes of capillaries
2. Loss of outer hair cells (OHCs)

These changes are described as cochlear changes in patients with type 2 diabetes mellitus. This section describes the test and results produced in [iv].

III.I Vessels of the Basilar Membrane

Morphometric measurements of the wall thickness of the vessels of the basilar membrane (VBM) were made in all turns of cochlea at the mid modular level and on the adjacent 2 sections.

Thickness of the vessel walls was measured using image analysis software and calculated using a modification of the method described by Robison. Results were presented as mean ±SD. Statistical evaluation was performed with the nonparametric Mann-Whitney test. The level of significance was set at P_0.05. Correlations were calculated with the Spearman rank correlation coefficient.

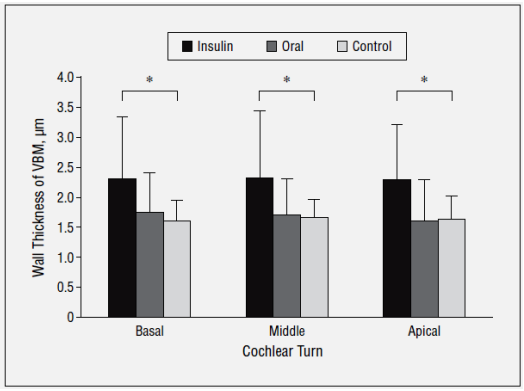

Figure1. Wall thickness of VBM on various cochlear turns

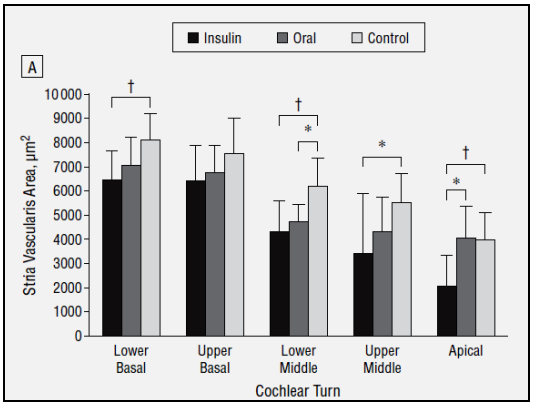

Figure2. Loss of OHC

Fig. 1 suggests that the insulin group had significant wall thickening of the VBM compared with the control group in the basal, middle, and apical turns of the cochlea (P=0.02, P=0.01, and P=0.04, respectively)

III.II Outer Hair Cell

Fig. 2 suggest a significantly greater loss of OHCs occurred in the insulin group compared with the control group in the lower basal (P0.001) and upper basal turns (P = 0.003).

The oral hypoglycemic agent group had a significantly greater loss of OHCs compared with the control group in the lower and upper basal turns of the cochlea (P=0.006 and P=0.04, respectively).

OHCs in the lower and upper basal turns of the cochlea in the diabetic groups compared with the control group [iv].

Study conducted demonstrates that type 2 diabetes mellitus results in changes of the cochlea, such as significant wall thickening of the VBM and OHC loss in the basal turn, that are likely to result in hearing loss [iv]. In addition, these pathologic changes of the insulin group showed a tendency to increase when compared with the oral hypoglycemic agent group. This study suggests that hearing loss in patients with type 2 diabetes mellitus may result from cochlear micro angiopathy [iv].

IV. MATHEMATICAL MODELING OF COCHLEA

Mathematical modeling of cochlea has been the topic of concern for so many years. The cochlea is the principle sensory organ of the mammalian auditory system. It is one of the most intricate and least understood mechanical systems in the body. Mathematical models of the cochlea are important tools in understanding the function of the cochlea because direct observation of cochlear vibrations is difficult.

The sound processing in the inner ear starts from input pressure waves that reach it through the middle ear. In mammals, frequency discrimination and active amplification are both performed in the cochlea, a cavity filled with a liquid medium, containing a tonotopically resonant vibrating membrane, the basilar membrane (BM). As shown in figure 4a and 4b when the input wave is a pure tone (i.e., it is made of a single frequency), the BM motion reaches its maximum amplitude at a specific

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01 Sept 2014

location, the characteristic place (CP) after a short transient, lasting a few milliseconds.[v] The frequency corresponding to CP is called characteristic frequency. By mapping each frequency component of a sound input to its characteristic place, the BM acts as a frequency analyzer.

The basilar membrane is also the base for the sensory cells of hearing, the hair cells. In mammals, the auditory hair cells are located within the organ of Corti on the BM. Cochlear hair cells come in two anatomically and functionally distinct types: the outer and inner hair cells (OHCs and IHCs). OHCs are the receptor potential triggers active vibrations of the cell body. This drives oscillations in the cells length and provide an ''active'' feedback amplification. OHCs do not send neural signals to the brain, but they are responsible of the nonlinear response to the above mentioned compressive nonlinear response of the BM. The amplification can be by movement of their hair bundles, or by an electrically driven motility of their cell bodies[v].

Table 3 suggest some of the physical parameters taken for various mathematical models developed over the time period [vi].

Table 3. Mathematical parameters

Parameters and denotation	Values	Unit
Cochlear duct height h	1.0	mm
Cochlear duct length L	25.0	mm
Fluid density ρ	1.0×10^{-3}	g/mm^3
BM mass per unit area $M(x)$	3.0×10^{-5}	g/mm^2
BM stiffness per unit area $S(x)$	$5.0 \times 10^{6} e^{-0.4x}$	$g/(mm^2 s^2)$
BM damping ratio ζ	0.2	
Tilt distance d	71.0×10^{-3}	mm
Segment length $\triangle$	1.0×10^{-2}	mm
OHC motility factor α	$0.0 - 0.2$	
Forward-to-backward ratio γ	0.3	

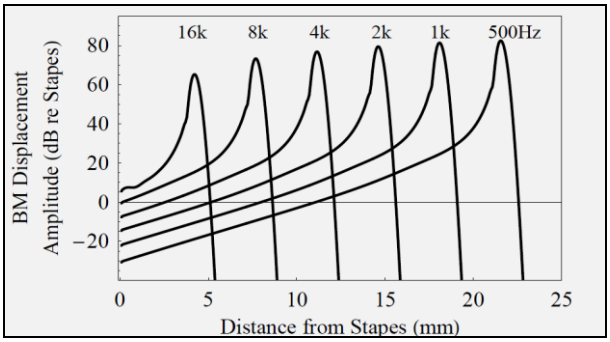

Figure4a. BM displacement

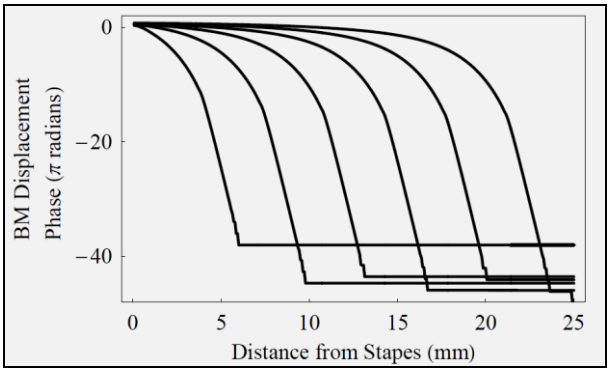

Figure4b. BM phase

Using the parameter from the table3 and WKBJ methods the dispersion equation for the cochlear model can be obtained as below:

$$\frac{k\tanh(kh)}{1+\alpha S(x)k\tanh(kh)(\gamma e^{ikd}-e^{-ikd})/(2\rho\omega^2)} = \frac{2\rho\omega^2}{S(x)+i\omega\beta(x)+(i\omega)^2 M(x)}$$

Where k is the complex wave number for a given input frequency ω. In the above dispersion equation one can input the single frequency ω and calculate the value of k from which the graphs in figure 4a and 4b can be obtained for BM displacement [vi].

Most of the mathematical cochlear models discussed in [v] [vi] [vii] [viii] models the BM as having length dependent mass and stiffness denoted by *M(x)* and *S(x)*. Also important factor as per the models is α which determines the motility factor

Prolonged hyperglycemic condition leads to thickening of vessels of BM as shown in figure 1 which means progressively changing *M(x)* and *S(x)* making them time variant. It means that the model parameters *M(x)* and *S(x)* which are space variant will also be made time variant. Due to overall stiffness change the BM may not be able to peak at the same amplitude level as shown in the figure 4. This probably will explain the increased threshold levels for diabetic subjects obtained in table 1.

Mathematical models also include motility factor α and its effects on variation on peak value of BM amplitude. Study shows that as α reduces the BM peak at CP reduces [vi]. Various mathematical models treat α as a motility factor which plays important role in providing active mechanical feedback required for amplification which helps BM to achive peak in tonotopic manner. Hence α is the representation of OHC activity. Change in α changes peak achieved by BM as shown in figure 6

Prolonged hyperglycemic condition leads to loss of OHC as shown in Fig. 2. Loss of OHC will affect the motility factor α which in turn change the BM peak displacement. This may explain the increased threshold in Fig.1. Also variation in α may change position of CP and hence generate SNHL.

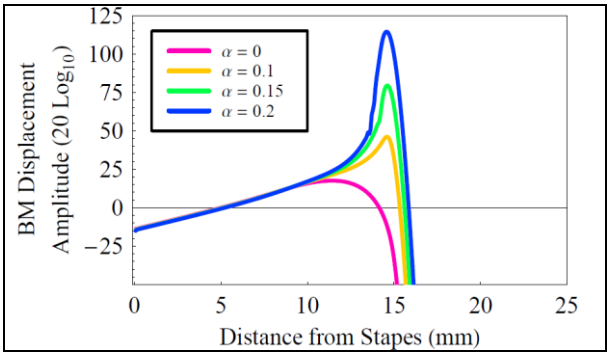

Figure 6 α variation changing peak amplitude of BM

V. Future Scope and Direction

As it is evident from the above discussion that the diabetes affects the hearing mechanism by affecting the thickness of BM and loss of OHC. Our research focuses on studying the effects of diabetics on stiffness of BM and motility factor α.

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We assume the mathematical model as shown in the Fig. 7 and try to perform the mathematical simulation which will calculate the deviation from the tonotopic relation shown in Fig. 8.

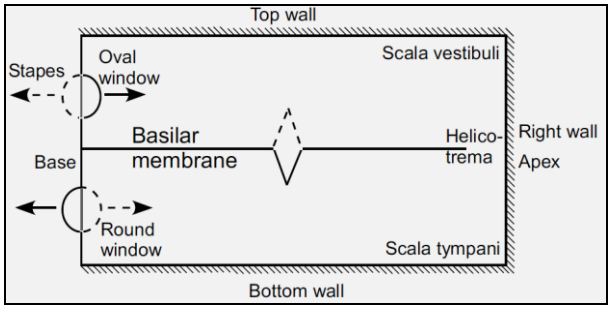

Figure 7 Physical cochlea model

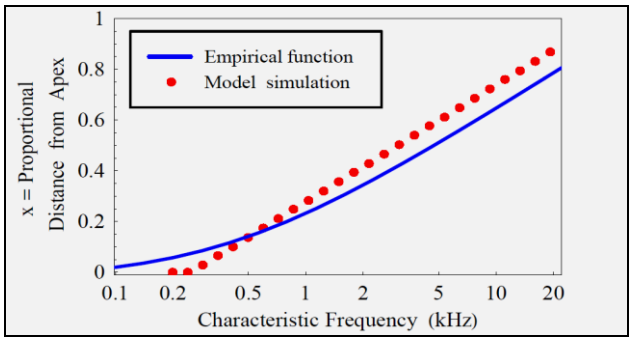

Figure8 Graphical relation between CP and CF

Conclusion

The diabetic affected parameters in cochlear models are the stiffness of BM and the motility factor α. Simulation on variation of the parameters on the BM amplitude response can throw light on correlation between diabetes and hearing acuity.

We propose that the realistic variations of α and properties of basilar membrane should be imposed on active non linear cochlear model proposed above and investigate the impairments caused by diabetes.

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