HORMONAL INFLUENCE ON AUDITORY FUNCTION

Rakhi Lakshmi

Reg. No. MSHM0116

A Dissertation Submitted in part fulfillment of Final Year M.Sc. (Speech and Hearing), University of Mysore, Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING
MANASAGANGOTHRI
MYSORE - 570006

May - 2003

To Mother with Love

"M" is for the million things she gave me,
"O" means only that she's growing old,
"T" is for the tears she shed to save me,
"H" is for her heart of purest gold;
"E" is for her eyes, with love-light shining,
"R" means right, and right she'll always be,
Put them all together, they spell "MOTHER"
A word that means the world to me.

Howard Johnson

CERTIFICATE

This is to certify that this Dissertation entitled **"HORMONAL INFLUENCE ON AUDITORY FUNCTION"** is a bonafide work in part fulfillment for the degree of Master of Science (Speech and Hearing) of the student (Register No. MSHM 0l16).

Mysore

May, 2003

Director
All India Institute of Speech and Hearing
Mysore - 570 006

CERTIFICATE

This is to certify that this Dissertation entitled **"HORMONAL INFLUENCE ON AUDITORY FUNCTION"** has been prepared under my supervision and guidance. It is also certified that this Dissertation has not been submitted earlier in any other University for the award of any Diploma or Degree.

Guide

Mr. Animesh Barman

Lecturer
Department of Audiology
All India Institute of Speech and Hearing
Mysore - 570 006

Mysore

May, 2003

DECLARATION

This Dissertation entitled **"HORMONAL INFLUENCE ON AUDITORY FUNCTION"** is the result of my own study under the guidance of **Mr. Animesh Barman** Lecturer, Department of Audiology, All India Institute of Speech and Hearing, Mysore and not been submitted earlier in any other University for the award of any Diploma or Degree.

Mysore,

May, 2003 Reg. No. MSHMO116

ACKNOWLEDGMENTS

First and foremost I would like to express my gratitude and sincere thanks to my guide, my teacher, ***Mr. Animesh Barman,*** *Lecturer, Department of Audiology, All India Institute of Speech and Hearing, Mysore. You have been helping and guiding me right from the beginning Sir, words are not enough to thank you.....*

I am thankful to ***Dr. M. Jayaram,*** *Director, All India Institute of Speech and Hearing for permitting me to carry out this study.*

I am thankful to ***Dr. Asha Yathiraj,*** *HOD, Department of Audiology for permitting me to use the instruments for this study.*

My thanks to ***Acharya*** *sir, for helping me out with my statistical analysis and Mr. Joby for his timely help.*

Special thanks to all my subjects for their co-operation.

Dearest ***Acha,***

I have always felt your presence in the disguise of silent blessings you shower on me wherever I am and whatever I do. Your everfresh memories linger on and on..... / wish you were there with us today.

Dearest ***Amma, Rana, Raja*** *and* ***Rani,***

Like a flower which turns to the sun, now and again

I have turned to you to be warmed by the light of your care.

What I am today, I owe it all to your love and unfailing support.

I thank God for having blessed me with such a wonderful family.

Dearest ***Amala***

No words can express how great I feel about you.....For as long as we have known each other, you have given me true and honest friendship, right from the heart. . . . .You were always there with me when I needed you the most. I wish our friendship lasts forever.....

Dear ***Kripal*** *and* ***Kundan,*** *No matter what we do or where we go, all the times we had together will be cherished forever.*

Dearest ***Chandi, Mili, Vimi, Mamta,*** *When we were together, good times with lots of fun just happen naturally. Being with you is something always look forward to. . . . . / am so glad I have you all as my friends. . . . . .Ur friendship means so much to me.*

Gupta, *and* ***Sandeep*** *- Thank you friends for all your help and understanding.*

Dear ***Shereen, Amy, Rajani, Suma, Rahana, Deema, Mili, Bhuvna*** *and* ***Vandana****. . . . . I cherish the times spent with u. . . . . .gonna miss u guys!*

Special thanks to ***Softouch*** *for giving a final shape to this project.*

TABLE OF CONTENTS

	Page No.
INTRODUCTION	1-4
REVIEW OF LITERATURE	5-10
METHOD	11-15
RESULTS AND DISCUSSION	16-31
SUMMARY AND CONCLUSION	32-35
REFERENCES	

LIST OF TABLES

Table No.	Title	Page No
1.	The Mean (M) and Standard Deviation (SD) for puretone air conduction threshold at different octave frequencies during the 3 phases of menstrual cycle	16
2.	The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) for puretone air conduction thresholds at different octave frequencies during the 3 phases of menstrual cycle	17
3.	The Mean (M) and Standard Deviation (SD) of static compliance (SC) and peak pressure (PPr) during the 3 phases of menstrual cycle	19
4.	The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) of static compliance (SC) and peak pressure (PPr) during the 3 phases of menstrual cycle	19
5.	The Mean (M) and Standard Deviation (SD) of SNR values of DPOAE at different frequencies during the 3 phases of menstrual cycle	20
6.	The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) of SNR values of DPOAE at different frequencies during the 3 phases of menstrual cycle	20
7.	The Mean (M) and Standard Deviation (SD) of SNR values of TEOAE at different frequency bands during the 3 phases of menstrual cycle	21
8.	The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) of SNR values of TEOAE at different frequency bands during the 3 phases of menstrual cycle	21
9.	The Mean (M) and Standard Deviation (SD) of latency and amplitude of I, III, and V peak of ABR at a repetition rate of 11.1 /sec during the 3 phases of menstrual cycle	23

10.	The 't' value, significance level and the difference between the mean of 2 different phases (MD) of latency and amplitude of I, III, and V peak of ABR at a repetition rate of 11.1/sec during the 3 phases of menstrual cycle	24
11.	The Mean (M) and Standard Deviation (SD) of latency and amplitude of I, III, and V peak of ABR at a repetition rate of 65.1/sec during the 3 phases of menstrual cycle	25
12.	The 't' value, significance level and the difference between the mean of 2 different phases (MD) of latency and amplitude of I, III, and V peak of ABR at a repetition rate of 65.1/sec during the 3 phases of menstrual cycle	25
13.	The Mean (M) and Standard Deviation (SD) of latency and amplitude of I, III, and V peak of ABR at a repetition rate of 90.1/sec during the 3 phases of menstrual cycle	26
14.	The 't' value, significance level and the difference between the mean of 2 different phases (MD) of latency and amplitude of I, III, and V peak of ABR at a repetition rate of 90.1/sec during the 3 phases of menstrual cycle	26
15.	The Mean (M) and Standard Deviation (SD) of latency of contra wave V at the repetition rates 11.1/sec, 65.1/sec and 90.1/sec during the 3 phases of menstrual cycle	28
16.	The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) of latency of contra wave V at the repetition rates 11.1/sec, 65.1/sec and 90.1/sec during the 3 phases of menstrual cycle	28

INTRODUCTION

Hormonal changes occur throughout a woman's life. The direct role of gonadal hormones on sensory processing, including the auditory system, has been scarcely studied. Estrogen and other gonadal steroid substances may have direct effects upon the cochlea and central auditory system. They could influence central processing indirectly through other pathways and they could modulate blood flow in the cochlea and brain. There are some indications that the menstrual cycle can alter certain aspects of hearing, just as it does a wide array of other perceptual-motor and cognitive abilities (Hampson, 1990; Parlee, 1983)

Two ovarian steroids have been proposed to be responsible for these changes estrogen or progesterone. During the first half (follicular phase) of the 28 day cycle, the follicle grows and matures under the dominant influence of follicle stimulating hormone and some luteinizing hormone. Early in this phase, follicular estrogen secretion is low, but towards the middle of the cycle, there is a burst of estrogen from the dominant follicle. If estrogen contributes to the change, then the effect would occur during this phase of the menstrual cycle. Following the rise in estrogen production, a surge in luteinizing hormone secretion occurs, which causes ovulation. After ovulation, marking the second phase of the cycle (luteal phase), the follicle undergoes luteinization and is converted to a progesterone-secreting structure, the corpus luteum. Progesterone acts to stimulate both endometrial gland and stroma maturation. If progesterone leads to the changes in hearing, then it is during this luteal phase that the change in auditory sensitivity would be seen. The corpus luteum involutes after about 12 days of secretion. With involution of the corpus luteum, the

fall in plasma estrogen and progesterone leads to withdrawal menstrual bleeding. Both estrogen and progesterone drop to their lowest level around the time of menses and the cycle begins again. Menstruation marks the end of the luteal phase and the beginning of the next follicular phase.

Thus woman with naturally occurring menstrual cycles can have systematic fluctuations in auditory threshold depending on the phase of their menstrual cycle (Swanson & Dengerink, 1988; Baker & Weiler, 1977). Davis and Ahroon (1982) and Miller and Gould (1967) reported better thresholds during the first half of the menstrual cycle and poorer thresholds during the luteal phase. In contrast Petiot and Parrot (1984) and Cox (1980) reported poorer thresholds at 4 KHz during the menstrual phase. During the menstrual phase both the hormone levels are low, at ovulation the progesterone levels are low but estrogen levels are high and during the luteal phase the progesterone levels are high and estrogen levels are moderately high (Swanson & Dengerink, 1988). Schiff, 1968 (cited in Cox, 1980) states hormonal influence on eustachian tube and cervical mucosa, which cause change in middle ear function. Even the latencies of waveform components of the auditory brainstem response were reported to be prolonged. (Elkind - Hirsch, Stoner, Stach & Jerger, 1992). In contrast a shortening of wave V latency was found during the luteal phase by Dehan and Jerger, 1990, while, no latency change as a function of the phase of the menstrual cycle was identified by Fagan and Church, 1986. It has been recently observed that the frequencies of spontaneous otoacoustic emissions may also exhibit changes during menstrual cycle (Bell, 1992; Haggerty, Lusted & Morton, 1993; Penner & Glotzbach, 1994). The intent of emphasizing the menstrual data here was to strengthen the implication that hormones have the potential to alter the function of

auditory system, which may throw light into hormonal processes primarily affecting other structures.

Need for the study

It is clear that the auditory system is also under the control of hormones by the fact that a number of auditory characteristics appear to fluctuate with the menstrual cycle. There are two hormones, estrogen and progesterone, which influence the hearing sensitivity by the systematic fluctuations in their levels across the menstrual cycle. The various studies on these hormonal influences on hearing sensitivity are contradictory. There is no agreement between the studies as to which hormones contribute more to the fluctuations in hearing sensitivity. This might be due to the methodological variation across the studies carried out. Thus it is essential to have a systematic study which will allow one to see whether these variation have any effect on auditory sensitivity during the menstrual phase and if its present then during which phase of the menstrual cycle it is more affected.

Further, no study has been done to supplement the results obtained in one test with the other tests. Also there is lack of studies on the effect of menstrual cycle on evoked OAEs. It is also essential to know whether progesterone or estrogen has more effect on auditory system. It is also important' to know whether hormonal effect is more on various auditory structures such as the middle ear, inner ear or higher levels in the auditory pathway. It is necessary to know how all these variations in hormonal level can affect hearing sensitivity and hence this study was taken up.

Aim of the study

1) To see the effect of hormonal changes on hearing sensitivity, whether these changes have more effect on behavioral, physiological or electrophysiological tests and what could be the possible reason for such changes.
2) Which hormone has more effect on hearing sensitivity.
3) To know the stable period during which the best hearing sensitivity could be obtained.

REVIEW OF LITERATURE

A number of investigators have studied the phenomenon of fluctuating auditory sensitivity as a function of the menstrual cycle.

Haggard and Gaston (1978) conducted a study to explain the changes in auditory perception during the menstrual cycle. The tasks involved were frequency J N D, tone modulation, octave matching and click lateralization. Results revealed no significant variation in the frequency JND task across the phases of menstrual cycle. But the remaining three tasks showed significant variations, though not in an entirely consistent pattern. One explanation could be that estrogen affect hearing by its influence on acetyl choline synthesis, which is a neurotransmitter in the efferent fibres of cochlea (Broverman, Klaiber, Kobayashi & Vogel, 1968, cited in Haggard & Gaston, 1978).

Another explanation is that of electrolyte variation affecting intercellular process. It might be possible that change in sodium and potassium metabolism could influence the process of axonal conduction and / or the availability of neurotransmitter at synapses. Changes in either of these could cause the effective conduction time to vary (Bruce & Russell, 1962, cited in Haggard & Gaston, 1978).

Baker and Weiler (1977), Davis and Ahroon (1982) and Miller and Gould (1967) reported better pure tone thresholds for normal cycling women during the first half of the menstrual cycle and poorer thresholds during the luteal phase i.e., hearing thresholds were poorest when progesterone was most elevated.

In contrast Petiot and Parrot (1984) and Cox (1980) reported poorer thresholds during the menstrual phase. Better thresholds were found in the postovulatory phase when progesterone is highest. In addition, these authors reported that 4 kHz thresholds were poorest during the menstrual phase when both estrogen and progesterone levels were lowest.

Parris (1964, cited in Cox, 1980) studied the possible change in auditory sensitivity that may occur during a normal menstrual cycle. He compared changes in pure tone air conduction thresholds at 5 time periods during the various stages of the menstrual cycle. The conclusion was that no significant effect on pure tone audiometric threshold could be attributed to various stages of the menstrual cycle.

Swanson and Dengerink (1988) examined the effects of ovarian hormones on auditory function in young women. A group of normally cycling women were tested during menstruation, at ovulation and during the luteal phase of their menstrual cycle. Results indicated significant cyclic fluctuations in auditory sensitivity, displaying better auditory thresholds at 4kHz during the later portions of the menstrual cycle than during menstruation. By selecting the chosen days, it would be possible to associate cyclic changes in hearing thresholds with specific hormones. From the present finding, it can be concluded that a higher level of endogenous estrogen is associated with better thresholds at 4kHz.

Several researchers also attempted to see the hormonal variation in middle ear structures. The middle ear pressure measurements were obtained at three specific

times during two consecutive menstrual cycles. (Cox, 1980) Results indicated higher negative middle ear pressure during the menstrual phase. This suggest the possibility that the natural increase in interstitial fluids, which generally begins 4 or 5 days before the onset of menses and culminates at or shortly after the onset of menses may be of sufficient magnitude to alter middle ear status. An increase of fluid within the tissue of the eustation tube would possibly reduce the ability of eustation tube to perform its normal physiologic function. If this increase in negative pressure were sufficient to cause slight retraction of the tympanic membrane, mechanical stiffness would have increased and possibly be responsible for the slight decrease in threshold noted during the menstrual phase.

Studies have shown that the frequencies of spontaneous otoacoustic emissions (SOAEs) may also exhibit menstrual rhythms (Bell, 1992; Haggerty, Lusted & Morton, 1993; Penner & Glotzbach, 1994), although there was no discernable regularity during the menstrual cycle for amplitude variations of SOAEs. Changes in SOAE frequency may result from menstrually linked changes in the outer hair cells (OHC) that are sensitive to metabolic and hormonal changes (Haggerty, Lusted & Morton, 1993; Schuknet, 1974, cited in Penner, 1995)

Penner (1995) studied frequency variation of spontaneous oto acoustic emissions during a naturally occurring menstrual cycle, amenorrhea and oral contraception. Results indicated that frequencies declined before the onset of menstruation and rose to a peak near the suspected time of ovulation. The rise and fall of the SOAE frequencies in step with the menstrual cycle must be directly or indirectly due to ovarian hormones, which seems to affect the tuning of the cochlea.

Ovarian hormones also seem to have effect on the central nervous system (Laugel, Dengerink & Wright, 1987) and may thereby produce cyclic changes in hearing through control of cardiovascular effects, as Bell (1992) argues.

Numerous studies have shown that latencies of the waveform components of the auditory brainstem response (ABR), particularly wave V component, are consistently shorter in women than that in men (Beagley & Sheldrake, 1978; Stockard, Stockard & Sharborough 1978, cited in Elkind-Hirsch, Stoner, Stach & Jerger, 1992; Trune, Mitchell & Phillips, 1978; McClelland & McCrea, 1979; Jerger & Hall, 1980; Jerger & Johnson, 1988). Clinical studies suggest that female sex hormones may contribute to these functional differences (Fagan & Church, 1986; Dehan & Jerger, 1990).

Studies of the auditory brainstem response report variable alterations in latency during the menstrual cycle. A shortening of wave V latency was found during the luteal phase in one study (Dehan & Jerger, 1990), while, in another, no latency change as a function of the phase of the menstrual cycle was identified (Fagan & Church, 1986).

Elkind-Hirsch, Stoner, Stach and Jerger (1992) evaluated the impact of the menstrual cycle on ABR latency in nine normally cycling women. The testing was done during four different phases of the same menstrual cycle. Results revealed a significant lengthening of wave V peak latency during the mid-cycle estrogen peak. In addition the wave I-V interpeak intervals were also increased. Wave III peak latency increased slightly at mid-cycle and decreased during the pre-menstrual period. The

latency of wave I varied little with the monthly cycle. The changing hormone levels had no apparent effect on the amplitude of wave V. The source of the latency change appears to be in the central auditory neural pathways rather than in the auditory periphery. Wave I latency, which reflects peripheral conduction time, does not change significantly. However, waves III and V, which reflect central conduction time are increased at mid-cycle.

These findings suggest that an elevation in estrogen alters the speed with which sensory information travels through the auditory brainstem nuclei. One explanation as to how a rise in the circulating levels of estrogen could modify auditory conduction time is by the estrogen effect on neurotransmitter synthesis. Gamma-aminobutyric acid (GABA) is one such neurotransmitter that has been reported to play a role as an inhibitory neurotransmitter in the efferent fibres of the cochlea (Altschuler & Fex, 1986, cited in Elkind-Hirsch, Stoner, Stach & Jerger, 1992; Eybalin & Pujol, 1986; Schwartz & Ryan, 1986, cited in Elkind-Hirsch, Stoner, Stach & Jerger, 1992).

There appears to be an intricate interaction among sex steroids in regulating GABA mediated signals. When steroid levels are high in the bloodstream, it will be high in the cerebrospinal fluid (CSF) that bathes the brain. Steroids are very soluble in cell membranes and would be expected to cross the blood brain barrier and enter the CSF. Levels of ovarian hormones, estrogen in particular, could have affected ABR latencies by modulating GABA action. Fluctuations in circulating estrogen levels during the menstrual cycle might have influenced availability of GABA at the synapse and in turn, influenced conduction time. The biphasic response of GABA to estrogen

action helps to further explain the increase in latency. At mid-cycle, estrogen has a positive feedback action which most likely would have resulted in enhanced GABA secretion in the brain, whereas estrogen has a negative feedback action during the early follicular phase and late luteal phase. Though there is no clear cut agreement between the results obtained by several researchers, it is evident from the above review that in women with regular menstrual cycle, there are some changes either in peripheral or central auditory pathway due to alteration of either estrogen or progesterone. Thus, this study has been taken up to investigate the influence of such hormonal variation at the various levels of the auditory system.

METHOD

Women with regular menstrual cycle were tested 3 times during the menstrual cycle i.e, during the menstrual phase when progesterone and estrogen levels were lowest, at ovulation or mid cycle, when progesterone levels were low but estrogen levels were high, and during the luteal phase when progesterone levels were high and estrogen levels were moderately high to see the effect of these hormonal variation on auditory system.

Subjects

Fifteen female subjects in the age range of 18 to 25 years with normal menstrual cycle were taken. Subjects met the following criteria.

Selection criteria

- Regular cycles of 27 to 31 days, of length (Cox, 1980).
- No otological and medical history.
- No history of use of steroids.
- Normal hearing sensitivity (Pure tone threshold within 15dB at octave frequencies between 500 Hz to 8KHz).
- Immittance 'A' type with reflexes present.
- No observable emotional disturbances.
- No neurological abnormality reported.

Instrumentation

- A calibrated diagnostic audiometer, GSI-61 connected with the TDH-50P headphones was used to measure the hearing threshold through air conduction.
- A calibrated middle ear analyser GSI-33 (version 2) was used to assess middle ear status and estimate static compliance and peak pressure.
- GSI - 60 DPOAE analyser to record DPOAEs.

  ILO 292, DPEchoport plus (version 5) was used to record TEOAE

  Biologic navigator with TDH-39P headphones was used to record the auditory brainstem response of the subjects.

Test environment

The tests were carried out in acoustically treated airconditioned room where the ambient noise level measured was within the permissible level as recommended by ANSI, 1991 (cited in Wilber, 1994).

Procedures

Subjects were tested three times during a single menstrual cycle i.e., follicular or menstrual phase (cycle days 1-4), ovulation or midcycle (cycle days 12-15) and the luteal or pre-menstrual phase (22-25).

Prior to test, detailed case history was taken to confirm no otological and neurological history, steroid intake etc. Otoscopic examination was done to check any abnormality in the external auditory canal.

The tests carried out were PTA, immittance, DPOAE, TEOAE and ABR.

Puretone thresholds at frequencies ranging from 500 Hz to 8 kHz for air conduction were estimated. At near threshold, 2 dB step was used to estimate threshold. The method used to obtain puretone threshold was modified West lake and Hughson method (Jerger & Carhart, 1959, cited in Silman & Silverman, 1997).

For immittance measurements the subjects were made to sit comfortably on a chair and were instructed not to move their head, jaw, swallow or to talk. Tympanometry and reflexes were then measured and the static compliance and peak pressure were noted down.

For OAE recording, each subject was seated comfortably and was instructed to relax and minimize any extraneous movements during the test. The probe was inserted gently into the earcanal by selecting an appropriate probe tip. Probe fit was ensured to check adequate fitting of the probe into the ear canal.

Stimulus parameters

Stimuli used to evoke DPOAE : DPOAE was measured using two primary tones with frequencies of fl and f2 for different fl frequencies of 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz. The ratio of the frequencies of the two primaries (fl/f2) was kept constant at 1.2. This ratio was chosen because it is well documented in the literature that the ratio between 1.2 to 1.23 provide optimal DPOAE amplitude (Harris, Lonsbury-Martin, Stagner, Coats & Martin, 1999). In the present study intensity levels of the primaries were maintained at 65dB SPL and 55 dB SPL respectively for fl and f2. This level was advocated by Stover, Gorga, Neely and

Montoya (1996) to produce optimum results and artifacts were also found to be less at this level.

Stimuli used to evoke TEOAE

Stimuli - Clicks

Level - 75 dBSPL

No of sweeps-256

SNR at frequency bands of 1kHz, 2kHz and 4kHz were noted.

Subjects were made to sit comfortably for ABR recording and instructed to relax. Electrode sites were cleaned with cleaning gel and silver coated disc electrodes were placed with the ten-20 paste. Attempt has been made to keep the absolute electrode impedance within 5KΩ and inter electrode impedance within 2KΩ to obtain best response.

Different parameters used for ABR recording are as follows

Type - Clicks

Intensity - 80 dBnHL

Polarity - Rarefaction

Duration- 100 μs

No. of stimuli- 1500

No of channels-2

Montage : Cz/A_1 Cz/A_2

Non inverting - Cz (Vertex)

Inverting - A_1, A_2 (A_1 - left mastoid and A_2 - right mastoid)

Common - Fz (forehead)

Filter setting - 100 Hz to 3000 Hz

ABR recording was done at three different rates i.e., 11.1/ sec, 65.1/sec and 90.1/sec at 80 dBnHL. The amplitude and latency of wave I, III, V were noted and also the latency of contralateral wave V.

Analysis

The data collected were subjected to statistical analysis. A paired 't' test was administered. The mean, standard deviation, 't' value and the difference between the mean obtained in two different phases were noted for all the parameters during the 3 phases of menstrual cycle.

RESULTS AND DISCUSSION

The objective of the study was to see the effect of hormonal changes on hearing sensitivity. Fifteen female subjects were taken for the study and were tested during the 3 phases of a single menstrual cycle. The data obtained were tabulated and subjected to statistical analysis. Paired 't' test was administered and the mean, standard deviation, 't' value and the difference between the mean obtained in two different phases for the various parameters were noted and compared. Effect of different phases of menstrual cycle on different tests are illustrated below.

Pure tone threshold

Table I: The Mean (M) and Standard Deviation (SD) for pure tone air conduction thresholds at different octave frequencies during the 3 phases of menstrual cycle.

Frequency / Phases	500Hz		lKHz		2KHz		4KHz		8KHz	
	M	SD	M	SD	M	SD	M	SD	M	SD
Menstrual	5.47	3.15	5.0	3.7	4.93	4.35	5.53	5.7	5.87	6.32
Midcycle	6.13	4.03	5.7	3.4	5.3	4.1	3.93	3.73	8.07	9.1
Luteal	6.46	3.81	5.5	3.4	5.3	3.5	5.13	4.65	7.6	5.47

Table 2: The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) for pure tone air conduction thresholds at different octave frequencies during the 3 phases of menstrual cycle.

Phase	500Hz			1KHz			2KHz			4KHz			8KHz		
	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD
Menstrual - midcycle	1.2	NS	0.66	1.4	NS	0.7	1.4	NS	0.37	1.7	NS	1.6	1.6	NS	2.2
Midcycle - Luteal	0.7	NS	0.33	0.4	NS	0.2	0.4	NS	0.0	1.9	95%	1.2	0.3	NS	0.47
Menstrual - Luteal	2.0	NS	0.99	0.7	NS	0.5	0.7	NS	0.37	0.4	NS	0.4	2.3	95%	1.73

Table 1 and 2 indicates that the puretone airconduction thresholds were better during the menstrual phase for frequencies 500 Hz, 1 kHz, 2 kHz and 8 kHz whereas at 4 kHz better threshold was seen during the mid-cycle. The thresholds at 500 Hz, 1 kHz , 2 kHz and 8 kHz were lesser during the later half of the menstrual cycle i.e., during luteal and mid cycle phases and the 4 kHz threshold was poorest during the menstrual phase. Maximum variation in the difference between the mean of 2 phases is seen between menstrual and midcycle phases for all the frequencies except 500 Hz. At 500 Hz, maximum variation is seen between menstrual and luteal phases.

However the threshold variation observed between the phases failed to reach significant level at 500 Hz, 1 kHz and 2 kHz. But statistically significant difference was obtained at 4 kHz between mid-cycle and luteal phases ($P<0.05$) and at 8 kHz between menstrual and luteal phases ($P<0.05$). In general there is no doubt that higher thresholds were obtained during the later half of the menstrual cycle i.e., during mid-cycle and / or luteal phases than during menstruation except at 4KHz. The possibility of poorer threshold at 4 KHz during the menstrual cycle might be attributed to the effect of ovarian hormones on cochlear blood flow. It has been reported that there is

poor blood supply to the part of the cochlea that corresponds to 4 kHz region (Crow, Guild & Polvogot, 1934, cited in Behar, Chasin & Cheesman, 2000). Thus, there could be significant effect on blood flow to cochlea during the menstrual cycle which significantly affect the region which is responsible for the perception of 4KHz tone. Similar finding has also been reported by Petiot & Parrot (1984) and Cox (1980) stating that 4 KHz threshold was poorest during menstrual phase and better threshold during later phases. However, they have attributed this variation to the estrogen and progesterone level.

At the other frequencies, poorer threshold during luteal and midcycle phases could be attributed to the levels of endogenous estrogen which is either moderately high or high. Estrogen influences the synthesis of neurotransmitters like GABA which inturn affect the electromotility of OHCs which brings about a reduction in threshold.

These results are in agreement with those reported in literature by Baker and Weiler (1977), Davis and Ahroon (1982) and Miller and Gould (1967). They reported better thresholds during the menstrual phase and poorer thresholds during the later portion i.e. mid-cycle and luteal phase. They have attributed the poorer threshold to the elevation in progesterone. However the current study suggests that estrogen is the main hormone which has more influence on behavioural results than the progesterone. Similar explanation was also given by Swanson and Dengerink, 1988. They reported that a higher level of endogenous estrogen is associated with better thresholds at 4 KHz.

Immittance Findings

Table 3 : The Mean (M) and Standard Deviation (SD) of Static Compliance (SC) and Peak Pressure (PPr) during the 3 phases of menstrual cycle.

Phases	SC		PPr	
	M	SD	M	SD
Menstrual	0.68	0.34	3.5	14.3
Midcycle	0.68	0.28	3.67	8.3
Luteal	0.73	0.27	2.2	7.4

Table 4: The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) of static compliance and peak pressure during the 3 phases of menstrual cycle.

Phases	SC			PPr		
	't'	Sig	MD	't'	Sig	MD
Menstrual - Midcycle	0.1	NS	0.0	0.06	NS	0.17
Midcycle - Luteal	1.3	NS	0.05	1.0	NS	1.47
Menstrual - Luteal	1.1	NS	0.05	0.5	NS	1.3

The results of table 3 and 4 reveal, that the static compliance value was higher during the luteal phase and lowest during the menstrual phase. But this difference is not statistically significant. The mean peak pressure value obtained across the three phases were almost the same, which has also failed to show any significant difference. Though there was no significant mean variation in peak pressure across the phases, few subjects had more negative middle ear pressure during the menstrual phase.

This is similar to the findings of Cox (1980). The reason attributed to this was an increase in interstitial fluids during the menstrual phase which alters the middle ear

status. The increase in the fluid also reduces the function of Eustachian tube to perform its normal function, i.e., it failed to equalize the middle ear pressure. As a result there will be an increase in negative middle ear pressure due to the partial absorption of air within the middle ear cavity. This increase in negative middle ear pressure reduces the mobility of tympanic membrane thus reducing the static compliance during the menstrual phase which is observed in the present study also.

Evoked OAEs

Table 5: The Mean (M) and Standard Deviation (SD) of SNR values of DPOAE at different frequencies during the 3 phases of menstrual cycle.

Frequency / Phase	500Hz		lKHz		2KHz		4KHz	
	M	SD	M	SD	M	SD	M	SD
Menstrual	12.82	5.5	25.8	6.8	26.43	4.87	26.33	8.5
Midcycle	13.24	6.4	25.24	6.7	26.72	4.9	25.07	9.6
Luteal	10.42	6.1	24.04	6.03	25.83	6.22	25.8	7.06

Table 6 : The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) of SNR values of DPOAE at different frequencies during the 3 phases of menstrual cycle.

Phases	500Hz			IKHz			2KHz			4KHz		
	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD
Menstrual - Midcycle	0.18	NS	0.42	0.65	NS	0.56	1.0	NS	0.29	0.02	NS	1.26
Midcycle Luteal	1.9	NS	2.82	0.93	NS	1.2	1.1	NS	0.89	0.19	NS	0.73
Menstrual - Luteal	1.9	NS	2.4	1.2	NS	1.76	0.35	NS	0.6	0.58	NS	0.53

Inspection of Table 5 and 6 shows that the SNR values of DPOAE were lowest during the midcycle or luteal phases and maximum during the menstrual phase

at all frequencies. The difference between the mean of DPOAE, SNR values obtained were minimum between menstrual and midcycle phases. However variations observed between the 2 phases did not show any significant difference.

Table 7: The Mean (M) and Standard Deviation (SD) of SNR values of TEOAE at different frequency bands during the 3 phases of menstrual cycle •

Frequency / Phases	l KHz		2KHz		4KHz	
	M	SD	M	SD	M	SD
Menstrual	8.72	5.06	13.17	5.5	12.78	6.87
Midcycle	9.28	5.8	10.06	5.87	10.61	7.72
Luteal	8.3	5.46	10.89	5.95	6.2	10.01

Table 8 : The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) of SNR values of TEOAE at different frequency bands during the 3 phases of menstrual cycle .

Phases	IKHz			2KHz			4KHz		
	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD
Menstrual - Midcycle	0.5	NS	0.56	4.1	99%	3.11	2.3	95%	2.17
Midcycle - Luteal	0.9	NS	0.98	0.7	NS	0.83	1.6	NS	4.41
Menstrual - Luteal	1.3	Ns	0.42	3.9	99%	2.28	3.0	99%	6.58

It can be seen in table 7 and 8, that the SNR values of TEOAE at 2 KHz and 4 KHz were better during the menstrual phase than the later portions of the menstrual cycle i.e., during mid-cycle and luteal phase. There is statistically significant difference seen between menstrual cycle and midcycle ($p < 0.01$) and between menstrual and luteal phase ($p < 0.01$) at 2KHz. Similar variation was also seen at 4KHz which is statistically significant between menstrual and midcycle ($p < 0.05$) and menstrual and luteal phase ($p < 0.01$).

Thus tables 5 to 8 indicate that there is variation in the SNR values of both DPOAE and TEOAEs. Better response was obtained during menstrual phase and poorer either during midcycle or luteal phase. This could be attributed to the fact that with the different phases, the level of estrogen and progesterone also varies. During the midcycle the estrogen is high and progesterone is low and during luteal phase progesterone is high and estrogen moderately high. Whereas during menstrual phase both the hormones are low. It has been reported that higher levels of estrogen has greater effect on synthesis of neurotransmitters like GABA. (Elkind-Hirsch, Stoner, Stach and Jerger, 1992). High levels of estrogen activate the release of GABA. This inturn could inhibit the function of OHCs which is known to be the generator of OAEs and thus leading to reduction of OAE amplitude during the midcycle or luteal phase.

However more variations are seen in TEOAE amplitude than DPOAE. This may be due to the fact that TEOAEs are more sensitive to subtle variation in the cochlea than DPOAEs, as TEOAEs are absent in 100% of ears with peripheral hearing loss exceeding 40 dBHL (Harris & Probst, 1997) whereas detectable DPOAE may be present for puretone threshold as high as 50-60 dB HL (Bonfils & Avan, 1992; Harris, 1990).

Researchers have explained the effect of estrogen on GABA but detailed information regarding function of progesterone and its effect on auditory system is still not clear. However we cannot completely rule out the effect of progesterone on cochlear physiology as minimum SNR values are also obtained for most of the frequencies during the luteal phase where the progesterone level is high and estrogen

level is moderately high. If the effect would have been due to progesterone, then the difference would be evident only during luteal phase as progesterone level is high during this phase. But since there is lower emissions reported in both midcycle and luteal phase, it may be inferred that the estrogen has more effect since it is either high or moderately high during both the phases.

Table 9: The Mean (M) and Standard Deviation (SD) of latency and amplitude of I, III and V peak of ABR at a repetition rate of 11.1 /sec during the 3 phases of menstrual cycle •

Wave	I				III				V			
	Latency		Amplitude		Latency		Amplitude		Latency		Amplitude	
Phase	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Menstrual	1.4	0.09	0.63	0.25	3.46	0.10	0.58	0.22	5.30	0.17	0.57	0.25
Midcycle	1.4	0.08	0.59	0.3	3.51	0.11	0.53	0.22	5.31	0.17	0.63	0.17
Luteal	1.4	0.1	0.63	0.25	3.52	0.14	0.58	0.3	5.32	0.19	0.6	0.18

Table 10: The 't' value, significance level and the difference between the mean of 2 different phases (MD) of latency and amplitude of I, III and V peak of ABR at a repetition rate of 11.1 /sec during the 3 phases.

Wave / Phase	I						III						V					
	Latency			Amplitude			Latency			Amplitude			Latency			Amplitude		
	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD
Menstrual - Midcycle	0.96	NS	0	1.3	NS	0.04	2.4	95%	0.05	0.1	NS	0.05	0.09	NS	0.01	1.98	95%	0.06
Midcycle - Luteal	0.9	NS	0	1.6	NS	0.04	0.22	NS	0.01	1.4	NS	0.05	3.5	NS	0.01	0.28	NS	0.03
Menstrual - Luteal	0.17	NS	0	0.44	NS	0.0	2.3	95%	0.06	0.25	NS	0	0.27	NS	0.02	1.0	NS	0.03

Tables 9 and 10 indicate that at a repetition rate of 11.1/sec, there was no variation in the latency of the I peak across the 3 phases of menstrual cycle. But there was a slight variation in the latency of III and V peak. Prolonged latency was seen during the luteal period for both III and V peak. Shortest latency was observed during the menstrual phase for both III and V peak. However the latency variation seen in wave III is statistically significant between menstrual and midcycle ($p<0.05$) and between menstrual and luteal ($p<0.05$) but wave V did not show any significant variation.

There was a statistically significant difference in the amplitude of wave V during the menstrual and midcycle ($P<0.05$). However there was no significant variation in the amplitude of Wave I and Wave III across the 3 phases of menstrual cycle. Maximum amplitude was obtained during the menstrual and luteal phases and minimum during the midcycle phase.

Table 11: The Mean (M) and Standard Deviation (SD) of latency and amplitude of I, III and V peak of ABR at a repetition rate of 65.1 /sec during the 3 phases of menstrual cycle.

Wave / Phase	I				III				V			
	Latency		Amplitude		Latency		Amplitude		Latency		Amplitude	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Menstrual	1.46	0.09	0.3	0.18	3.63	0.11	0.3	0.11	5.58	0.17	0.66	0.14
Midcycle	1.53	0.08	0.3	0.15	3.68	0.11	0.3	0.14	5.60	0.16	0.66	0.15
Luteal	1.52	0.12	0.36	0.16	3.65	0.13	0.4	0.18	5.59	0.19	0.68	0.16

Table 12: The `t' value, significance level and the difference between the mean of 2 different phases (MD) of latency and amplitude of I, III and V peak of ABR at a repetition rate of 65.1 /sec during the 3 phases of menstrual cycle.

Wave / Phase	I						III						V					
	Latency			Amplitude			Latency			Amplitude			Latency			Amplitude		
	`t'	Sig	MD	`t'	Sig	MD	`t'	Sig	MD	`t'	Sig	MD	`t'	Sig	MD	`t'	Sig	MD
Menstrual - Midcycle	2.8	99%	0.07	0.53	NS	0.0	1.4	NS	0.03	0.46	NS	0.0	1.4	NS	0.01	0.17	NS	0.0
Midcycle - Luteal	0.48	NS	0.01	1.7	NS	0.06	2.4	95%	0.05	1.0	NS	0.1	1.6	NS	0.02	0.13	NS	0.02
Menstrual - Luteal	2.6	95%	0.06	0.8	NS	0.06	1.2	NS	0.02	2.3	95%	0.1	0.19	NS	0.01	0.78	NS	0.02

Table 13: The Mean (M) and Standard Deviation (SD) of latency and amplitude of I, III and V peak of ABR at a repetition rate of 90.1 /sec during the 3 phases of menstrual cycle.

Wave / Phase	I				III				V			
	Latency		Amplitude		Latency		Amplitude		Latency		Amplitude	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Menstrual	1.52	0.12	0.24	0.15	3.67	0.12	0.28	0.10	5.65	0.16	0.71	0.22
Midcycle	1.65	0.44	0.29	0.17	3.73	0.12	0.3	0.15	5.69	0.19	0.73	0.23
Luteal	1.55	0.12	0.26	0.12	3.68	0.15	0.3	0.12	5.66	0.17	0.64	0.23

Table 14: The `t' value, significance level and the difference between the mean obtained in 2 different phases (MD) of latency and amplitude of I, III and V peak of ABR at a repetition rate of 90.1 /sec during the 3 phases of menstrual cycle.

Wave / Phase	I						III						V					
	Latency			Amplitude			Latency			Amplitude			Latency			Amplitude		
	`t'	Sig	MD	`t'	Sig	MD	`t'	Sig	MD	`t'	Sig	MD	`t'	Sig	MD	`t'	Sig	MD
Menstrual - Midcycle	1.3	NS	0.13	0.23	NS	0.05	2.2	95%	0.06	0.28	NS	0.02	1.6	NS	0.02	0.9	NS	0.02
Midcycle - Luteal	0.84	NS	0.1	0.63	NS	0.03	1.6	NS	0.05	0.27	NS	0.0	0.09	NS	0.03	0.56	NS	0.09
Menstrual - Luteal	0.74	NS	0.03	0.6	NS	0.02	1.5	NS	0.01	1.1	NS	0.02	0.59	NS	0.01	2.0	95%	0.07

Latency of wave I, III and V also varied at 65.1 / sec across the phases which is clear from the Table 11 and 12. Latency variation which was seen for wave I and III are statistically significant between menstrual and midcycle ($p < 0.01$) and between menstrual - luteal phase ($p < 0.05$) where as no significant difference was obtained for wave V across the phases. Maximum latency was obtained during the midcycle phase for all the peaks. Minimum latency was seen in the menstrual phase.

Maximum amplitude for wave I, III and V was obtained during the luteal phase and minimum amplitude during the menstrual or midcycle phases. But the amplitude variation was not statistically significant for any wave across the phases except between luteal and menstrual phases for wave III ($p < 0.05$).

At higher repetition rates i.e., at 90.1 / sec, variations in the latencies of I, III and V peak across the phases of the menstrual cycle is evident in Table 13 and 14. However, statistically significant difference in latency was seen only for Wave III between the menstrual phase and midcycle ($P<0.05$). Maximum latency was obtained during the luteal phase and minimum latency during the menstrual phase for all the peaks.

Statistically no significant difference in the amplitude of Wave I, III and V across the 3 phases was seen except for amplitude of Wave V between luteal phase and menstrual phase which was significant at ($P<0.05$). However maximum amplitude was obtained during the midcycle and minimum amplitude during the menstrual phase.

Table 15: The Mean (M) and Standard Deviation (SD) of latency of contra wave V at the repetition rates 11.1/sec, 65.1/sec and 90.1/sec during the 3 phases of menstrual cycle.

RR / Phase	11.1/sec		65.1/sec		90.1/sec	
	M	SD	M	SD	M	SD
Menstrual	5.36	0.18	5.6	0.19	5.67	0.24
Midcycle	5.38	0.16	5.6	0.17	5.68	0.17
Luteal	5.88	0.20	5.6	0.19	5.72	0.21

Table 16 : The 't' value, significance level and the difference between the mean obtained in 2 different phases (MD) of latency of contra wave V at the repetition rates 11.1/sec, 65.1/sec and 90.1/sec during the 3 phases of menstrual cycle.

RR / Phase	11.1/sec			65.1/sec			90.1/sec		
	't'	Sig	MD	't'	Sig	MD	't'	Sig	MD
Menstrual - Midcycle	1.8	NS	0.02	1.2	NS	0	2.3	95%	0.01
Midcycle - Luteal	1.3	NS	0.5	0.37	NS	0	1.1	NS	0.04
Menstrual - Luteal	0.08	NS	0.52	1.8	NS	0	0.6	NS	0.05

Table 15 and 16 indicate that there is statistically significant difference in latency of contra Wave V at a repetition rate of 90.1/sec between the menstrual and midcycle phase ($P<0.05$). The maximum latency was seen during the luteal phase at all the repetition rates and minimum latency was seen during the menstrual phase. The difference between mean obtained in 2 different phases was more during the menstrual and luteal phase at all repetition rates.

The latency was minimum during the menstrual phase and maximum during the midcycle or luteal phase.

Morphology

Don, Allen and Starr, 1977; Fowler and Noffsinger, 1983 have reported that ABR components I and V usually do not become indistinct with increased rate in normal subjects, but waves, II, III and IV may disappear at higher stimulus rates.

As the repetition rate increases morphology becomes poor. Similar pattern is observed in the present study also. But no significant abnormality interms of wave morphology is seen across the phases.

It can be seen from the above results on ABR that at all the repetition rates there is no significant variation in wave I latency and amplitude but significant variation in Wave III and slight variation but not significant in wave V latency. Though the variation in Wave III latency is statistically significant, it failed to reach clinical importance. In general latency seem to be shortest during the menstrual phase and longest either during midcycle or luteal phase. This can be attributed to the fact that the hormonal fluctuations during the different phases can cause the release of neurotransmitters like acetylcholine which inturn can alter the sodium and potassium permeability (Bruce & Russell, 1962, cited in Haggard & Gaston, 1978). The changes in sodium and potassium metabolism is evident during the midcycle or luteal phase. This alteration slowed down the conduction time resulting in prolonged latency as also supported by the result obtained by Broverman, Klaiber, Kobayashi and Vogel, 1968 (cited in Haggard & Gaston, 1978). This prolongation is more prominent at higher peaks than the lower peaks which is also seen in this study. That is, more during luteal or midcycle when estrogen and progesterone is high or moderately high. During the menstrual phase low level of estrogen and progesterone reduce the release

of acetyl choline resulting in faster conduction and reduced latency. More recently it has been suggested that an elevation in estrogen alters the speed with which sensory information travels through the auditory brainstem nuclei. One explanation is to how a rise in the circulating levels of estrogen could modify auditory conduction time is by the estrogen effect on neurotransmitter synthesis. GABA is one such neurotransmitter. GABA has been reported to play a role as an inhibitory neurotransmitter in the efferent fibres of the cochlea (Schwartz & Ryan, 1986, cited in Elkind-Hirsch, Stoner, Stach & Jerger, 1992). It is hard to say which hormone has more influence on conduction time as for some peaks and rates maximum latency is seen at midcycle or luteal phase and minimum during the menstrual phase. Progesterone is low during midcycle and high during the luteal phase whereas estrogen is either high or moderately high during midcycle and luteal phases. Hence it can be inferred that the estrogen has more influence on conduction time as prolonged latency is seen either during midcycle or luteal phase.

Thus, summarizing the results for puretone threshold, DPOAE and TEOAEs better response was obtained during menstrual phase and poorer during mid-cycle or luteal phase. During the midcycle and luteal phase both estrogen and progesterone is high or moderate levels and during the menstrual phase both hormones are low. It has been reported that more than progesterone, estrogen has greater impact on neurotransmitters like GABA (Elkind-Hirsch, Stoner, Stach & Jerger, 1992). High levels of estrogen activate the release of GABA and hence inhibiting the function of OHCs, which is known to be the generator of OAEs leading to reduction in OAE amplitude. And also by inhibiting the function of OHC hearing sensitivity would have also become poorer which is seen during this period whereas this effect is not seen

during the menstrual phase. Thus, the variation in puretone threshold and OAE amplitude suggest that hormonal changes have greater effect on cochlear function than middle ear as no significant variation either interms of static compliance and middle ear pressure is noted. At the same time it is seen that hormonal variations in neural activity may be minimum as in the present study we have failed to get any clinically significant difference either in III or V peak latency and amplitude which is supposed to be affected by alterations of sodium and potassium permeability or by levels of ovarian hormones, estrogen in particular, which could affect ABR latencies by modulating GABA action. At the same time this effect cannot be completely ruled out as statistically significant difference in latency is seen for wave III and slight prolongation is also seen for wave V.

SUMMARY AND CONCLUSION

Women with naturally occurring menstrual cycles have systematic fluctuations in auditory threshold depending on the phase of their menstrual cycle (Swanson & Densgerink, 1988). These fluctuations may be due to hormonal influences on cochlear blood flow (Laugel, Dengerink & Wright, 1987; Laugel, Wright & Dengerink, 1988). Two ovarian hormones have also been proposed to be responsible for these fluctuations in the threshold, i.e., estrogen and progesterone. The levels of these hormones vary depending upon the phase of the menstrual cycle.

Studies on middle ear function during menstrual cycle indicated higher negative middle ear pressure (Cox, 1980). Even the latencies of waveform components of auditory brainstem response were seem to have increased (Elkind-Hirsch, Stoner, Stach & Jerger, 1992). It has been recently reported that the frequencies of spontaneous otoacoustic emissions (SOAEs) may also exhibit changes during menstrual cycle (Bell, 1992; Haggerty, Lusted & Morton, 1993; Penner & Glotzbach, 1994).

Therefore it is necessary to know how all these variations can affect the hearing sensitivity at different levels of the auditory system. Also there has been no study to supplement the result obtained in one test with the other tests. Hence this study has been taken with the objective to see the effect of hormonal changes on hearing sensitivity, which hormone has more effect and which level of the auditory system is more affected by the hormonal fluctuations.

Fifteen female subjects with normal menstrual cycle were taken for the study. They were tested 3 times during a single menstrual cycle i.e, menstrual (cycle days 1-4), midcycle (12-15) and luteal phase (days 22-25). A battery of tests were administered which include pure tone audiometry, immittance, DPOAE, TEOAE, and ABR measurements. The puretone airconduction thresholds were obtained at 500 Hz, 1kHz, 2kHz, 4kHz and 8kHz. In the immittance, the static compliance and peak pressure values were obtained. In OAEs the SNR values at specific frequencies for both DPOAE and TEOAEs were noted. Auditory brainstem responses were obtained at three repetition rates i.e., 11.1/sec, 65.1/sec and 90.1/sec at 80 dBnHL. The tests were carried out in sound treated room using standard procedure and protocol.

The data obtained were subjected to statistical analysis. Paired 't' test was administered and the mean, standard deviation and 't' values of the various variables were noted and compared.

Results showed variations between the 3 phases of the menstrual cycle for pure tone threshold, SNR values of TEOAEs, and DPOAEs. The results are in agreement with the previous studies presented in literature by Baker and Weiler (1977), Davis and Ahroon (1982) and Miller and Gould (1967). Puretone threshold, TEOAE and DPOAE response seem to be better during the menstrual phase and poorer during the midcycle or luteal phase. This variation may be mainly due to physiological changes taking place within the cochlea due to the alteration of estrogen and progesterone level. They have attributed the poorer threshold to the elevation in progesterone. However, the present study suggests that estrogen is the main hormone which has more influence on behavioural, physiological and electrophysiological results than the progesterone.

However, there is only slight variation in the latency of waveform components of ABR. There is statistically significant difference in latency of only the wave III component. No clinically significant variations in the latency and amplitude of wave III and V is seen. This suggests that the variation in hormonal level also has some effect on neural activity but may not be as significant as on the cochlear physiology.

The hormones affect the synthesis of neurotransmitter like GABA which inhibit the function of OHC which inturn results in reduced OAE amplitude, reduced hearing sensitivity and also prolonged ABR latencies (Elkind-Hirsch, Stoner, Stach & Jerger, 1992) during the menstrual cycle. Hormones also affect the synthesis of neurotransmitter like acetyl choline which inturn alters the sodium potassium metabolism and thereby reduces the axonal conduction time (Bruce & Russell, 1962, cited in Haggard & Gaston, 1978). However it could not be explained whether estrogen or progesterone has significant effect on auditory system as variation is seen either during the midcycle or luteal phase. But since the levels of estrogen is either high or moderately high during the midcycle or luteal phase when compared to progesterone which is low during mid cycle and high during luteal phase, we can assume that estrogen has more influence on auditory function. Also the most stable period during which the best hearing sensitivity could be obtained is the menstrual phase, where both the hormones are at low levels. Thus it can be concluded that hormones have the potential to alter the function of auditory system especially at the cochlear level. It also provides a valuable window into hormonal processes primarily affecting other structures.

Implication

- The present study emphasise the hormonal variations that take place during the menstrual cycle. It also suggests about the audiological tests which can be used to assess the normal variations of hormones, especially estrogen during the cycle.
- This study also highlight the hormones which are responsible for the changes in the function of the auditory system and also the level at which the maximum effect is seen.
- The present study also throw light into the variations in the responses that can be observed during the menstrual cycle, thus suggesting the most stable period during which one can expect better results in all the audiological tests.
- It also give some guidelines to the future researches about how the effect of other agents like steroids, contraceptives etc on hormone synthesis and hearing sensitivity can be studied.

REFERENCES

Baker, M.A., & Weiler, E.M. (1977). Sex of listener and hormonal correlates of auditory thresholds. *British Journal of Audiology,* 11, 65-68.

Beagley, H.A., & Sheldrake, J.B. (1978). Differences in brainstem response latency with age and sex. *British Journal of Audiology,* 12, 69-77.

Behar, A., Chasin, M, & Cheesman, M. (2000). *Noise control - A primer.* San Diego : Singular publishing group.

Bell, A. (1992). Circadian and menstrual rhythms in frequency variation of spontaneous otoacoustic emissions from human ears. *Hearing Research,* 58, 91-100.

Bonfils, P., & Avan, P. (1992). Distortion product otoacoustic emissions : Values for clinical use. *Archives of otolaryngology - Head and Neck Surgery,* 118, 1069-1076.

Cox, J.R. (1980). Hormonal influence on auditory function. *Ear and Hearing,* 1, 219-222.

Davis, M.J., & Ahroon, W.A. (1982). Fluctuations in susceptibility to noise-induced temporary threshold shift as influenced by the menstrual cycle. *Journal of Auditory Research,* 22, 173-187.

Dehan, C.P., & Jerger, J. (1990). Analysis of gender differences in the auditory brainstem response. *Laryngoscope,* 100, 18-24.

Don, M., Allen, A.R., & Starr, A. (1977). Effect of click rate on the latency of auditory brainstem responses in humans. *Annals of Otology, Rhinology and Laryngology,* 86, 186-195.

Elkind-Hirsch, K.E., Stoner, W.R., Stach, B.A., & Jerger, J.F. (1992). Estrogen influences auditory brainstem response during the normal menstrual cycle. In B.R. Alford & S. Jerger (Eds.), *Clinical Audiology- A Jerger perspective* (pp. 411-416). San Diego: Singular publishing group, Inc.

Eybalin, M., & Pujol, R. (1986). Cochlear neuroactive substances. *Archives of otorhinolaryngology*, 246, 228-234.

Fagan, P.L., & Church, G.T. (1986). Effect of the menstrual cycle on the auditory brainstem response. *Audiology*, 25, 321-328.

Fowler, C, & Noffsinger, D. (1983). Effects of stimulus repetition rate and frequency on the auditory brainstem response in normal, cochlear impaired and VIII nerve / brainstem impaired subjects. *Journal of Speech and Hearing Research*, 26, 560-567.

Haggard, M., & Gaston, J.B. (1978). Changes in auditory perception in the menstrual cycle. *British Journal of Audiology*, 12, 105-118.

Haggerty, H.S., Lusted, H.S., & Morton, S.C. (1993). Statistical quantification of 24-hour and monthly variation of spontaneous otoacousic emission frequency in humans. *Hearing Research*, 70, 31-49.

Hampson, E. (1990). Variations in sex-related cognitive abilities across the menstrual cycle. *Brain and Cognition,* 14, 26-43.

Harris, F.P. (1990). Distortion product otoacoustic emissions in humans with high frequency sensorineural hearing loss. *Journal of Speech and Hearing Research*, 33, 594-600.

Harris, F.P., & Probst, R. (1997). Otoacoustic emissions and audiometric outcomes. In M.S. Robinette & T.J. Glattke (Eds.), *Otoacoustic emissions: Clinical applications* (pp. 151-180). New York: Thieme.

Harris, F.P., Lonsbury-Martin, B.L., Stagner, B.B., Coats, A.C., & Martin, G.K. (1999). Acoustic distortion products in humans: Systematic changes in amplitude as a function of fl/f2 ratio. *Journal of Acoustic Society of America,* 85, 220-229.

Jerger, J., & Hall, J. (1980). Effects of age and sex on auditory brainstem response, *Audiology,* 25, 321-328.

Jerger, J., & Johnson, K. (1988). Interactions of age, gender and sensorineural hearing loss on ABR latency. *Ear and Hearing,* 9, 168-175.

Laugel, G.R., Dengerink, H.A., & Wright, J.W. (1987). Ovarian steroid and vasoconstrictor effects on cochlear blood flow. *Hearing Research,* 31, 245-252.

Laugel, G.R., Wright, J.W., & Dengerink, H.A. (1988). Vasoconstriction and progesterone effects on laser doppler measures of cochlear blood flow. *Acta Otolaryngologica,* 106, 34-39.

McClelland, R.J., & McCrea, R.S. (1979). Inter subject variability of the auditory - evoked brainstem potentials. *Audiology,* 18, 462-471.

Miller, M.H., & Gould, W.J. (1967). Fluctuating sensorineural hearing impairment associated with the menstrual cycle. *Journal of Auditory Research,* 7, 373-385.

Parlee, M.B. (1983). Menstrual rhythms in sensory processes: A review of fluctuations in vision, olfaction, audition, taste and touch. *Psychological Bulletin,* 93, 539-548.

Penner, M.J. (1995). Frequency variation of spontaneous otoacoustic emissions during a naturally occurring menstrual cycle, amenorrhea, and oral contraception: A brief report. *Ear and Hearing,* 16, 428-432.

Penner, M.J., & Glotzbach, L. (1994). Covariation of tinnitus pitch and the associated emission : A case study. *Otolaryngology - Head & Neck Surgery,* 100, 304-309.

Petiot, J.C, & Parrot, J.E. (1984). Effects of ovarian and contraceptive cycles on absolute thresholds, auditory fatigue and recovery from temporary threshold shifts at 4 and 6 KHz. *Audiology*, 23, 581-598.

Silman, S., & Silverman, C.A. (1997). *Auditory Diagnosis principles and Application.* London : Singular publishing group, Inc.

Stover, L., Gorga, M.P., Neely, ST., & Montoya, D. (1996). Towards optimizing the clinical utility of distortion product otoacoustic emission. *Journal of Acoustic Society of America,* 100,956-967.

Swanson, S.J., & Dengerink, H.A. (1988). Changes in puretone thresholds and temporary threshold shifts as a function of menstrual cycle and oral contraceptives. *Journal of Speech and Hearing Research,* 31, 569-574.

Trune, D.R., Mitchell, C, & Phillips, D.S. (1978). The relative importance of head size, gender and age on the auditory brainstem response. *Hearing Research,* 32, 165-174.

Wilber, L.A. (1994). Calibration, puretone, speech and noise signals. In J. Katz (Ed.). *Handbook of clinical audiology* (pp. 73-97). Baltimore: Williams and Wilkins.