# COMPARATIVE STUDY OF BRAINSTEM AUDITORY EVOKED POTENTIAL AND SOMATOSENSORY EVOKED POTENTIAL IN TOTAL BLIND AND NORMAL SUBJECTS

*Dissertation submitted to*

**THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY**

*In partial fulfilment of the*

*Regulations for the award of the degree of*

**(M.D. PHYSIOLOGY)**
**BRANCH-V**

**THANJAVUR MEDICAL COLLEGE HOSPITAL**

**THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERISTY**

**CHENNAI, INDIA**

**APRIL – 2016**

# CERTIFICATE

This dissertation entitled **"Comparative study of Brainstem Auditory Evoked Potential and Somatosensory Evoked Potential in total blind and normal subjects"** is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the regulations for the award of M.D., Degree in physiology in the Examinations to be held during April 2016

This Dissertation is a record of fresh work done by the candidate **Dr. S. CHANDRABALAN,** during the course of the study (2013-2016). This work was carried out by the candidate himself under my supervision.

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## DECLARATION

I solemnly declare that the Dissertation titled **"Comparative study of Brainstem Auditory Evoked Potential and Somatosensory Evoked Potential in total blind and normal subjects"** is done by me at Thanjavur Medical College, Thanjavur

The Dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of requirements for the award of M.D. Degree (Branch V) in physiology

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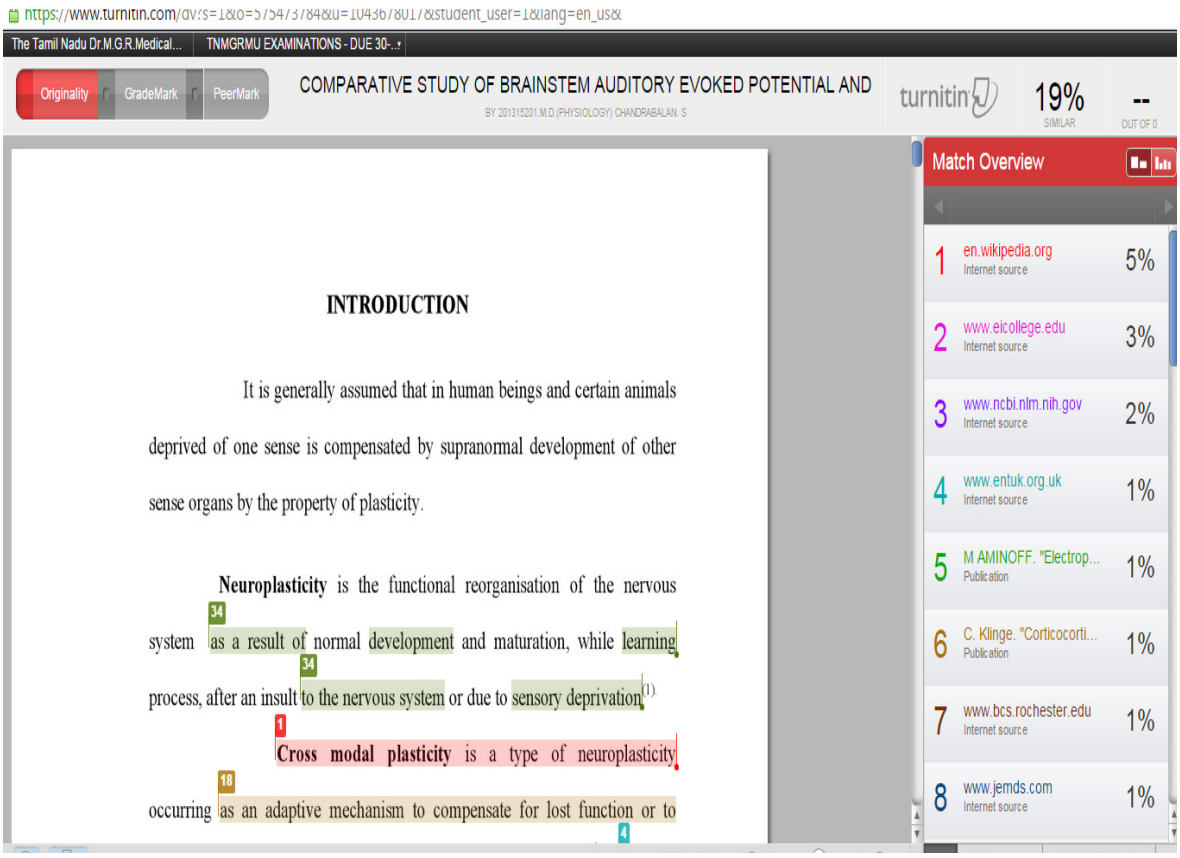
Last but never the least, I am immensely grateful to my ever loving and ever supporting wife and my children.

Finally, I thank almighty God for completion of this study and guidance at every step in my life.

# ANTI - PLAGIARISM ORIGINALITY REPORT



# Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA-613 001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

## INSTITUTIONAL ETHICAL COMMITTEE

## CERTIFICATE

Approval No. : 093

**This is to certify that The Research Proposal / Project titled**

COMPARATIVE STUDY OF BRAIN STEM AUDITORY EVOKED POTENTIAL AND SOMATOSENSORY EVOKED POTENTIAL IN TOTALLY BLIND AND NORMAL SUBJECTS.

**submitted by Dr.** S. CHANDRABALAN **of**

**Dept. of** PHYSIOLOGY **Thanjavur Medical College, Thanjavur**

**was approved by the Ethical Committee.**

Thanjavur

Dated : 19.9.2014.

THANJAVUR MEDICAL COLLEGE * THANJAVUR-4 *

**Secretary**

**Ethical Committee**

**TMC, Thanjavur.**

## CONTENTS

# INTRODUCTION

It is generally assumed that in human beings and certain animals deprived of one sense is compensated by supranormal development of other sense organs by the property of plasticity.

**Neuroplasticity** is the functional reorganisation of the nervous system as a result of normal development and maturation, while learning process, after an insult to the nervous system or due to sensory deprivation[1].

**Cross modal plasticity** is a type of neuroplasticity occurring as an adaptive mechanism to compensate for lost function or to maximize the remaining functions in the event of brain injury[2].It involves neural reorganisation in between various senses, followed by sensory deprivation. In these circumstances, there is a decrease in the neural representation of the deprived sense .This is compensated by increased representation of intact sense[3]

There are number of studies to provide experimental evidence for this compensatory plasticity in blind humans[4, 5, 6,7].

**Neuroplasticity** change is not uniform throughout the development period. There are certain period of time called critical period, which act as temporal window during postnatal development by allowing the experience to influence the end organisation of the neural connections[8].

During critical period, gross alteration in neural circuit is possible at the level of branching of axon and dendrite[9]. The resultant circuit seems to be stable and prevent the plasticity beyond adulthood[10]

For this, sensory input must be required during such critical period if normal development has to develop.The earlier the onset of blindness, the plasticity will be more involving the visual cortex and auditory cortex. Such plastic changes vary across brain systems, giving rise to highly specific alterations as a function of the nature of altered experience.

The probable reason for cross-modal plasticity is due to change in subcortical connectivity in brainstem and thalamus or due to change in cortico-cortical feedback or in the long-range connections between the primary cortexes .

These sensory changes are due to experience driven from peripheral activity rather than blindness. That is why individuals born blind show increased connectivity between their visual cortex and auditory cortex[11].

This leads to distribution of auditory processing between these two systems[12].

As a result of this increased representation, there is enhanced auditory ability of the blind, like improved identification of speech[11] .

## AIM AND OBJECTIVES

To compare the Brainstem auditory evoked potential response and Somatosensory evoked response after median nerve stimulation in total blind subjects with that of normal sighted subjects.

To evaluate, the plasticity changes in the auditory system and somatosensory system.

# REVIEW OF LITERATURE

Blindness is a major problem all over the world. The world health organization (WHO) (2002) estimates that for every five seconds, someone goes blind. India is a residence of World's largest number of blind people. Among, 45 million blind people found all over the World, around 15 million blind people were found in India [13]

In 2000,the number of blind persons in India was estimated to be around 18.7 millions and in 2010, the estimated blind persons increased to 24.1 millions and in 2020, the estimated blind persons may be increased to31.6 millions .[14]

According to 2001 census, Directorate General of Health services (DGHS) and Ministry of Health & Family Welfare, New Delhi ,had estimated that there were 1.4 million blind children in the world.[15]

Among them, 2,70,000 blind children were found in India. Its prevalence rate was estimated to be around 1.78% .The incidence rate of blindness was 1.29 % . Out of which 328 were in 0-14 yrs of age group and 2953 in 15-49 yrs of age group. The estimated prevalence rate of blindness is 16.7% per 1000 population .The number of newly occuring visually impaired cases per year is 19,386[13].

Blindness is defined as a condition in which the individual lacks vision. This lack can be due to diseases, trauma or genetic factors and can have effects of differing strength .World Health Organisation has classified blindness as follows

## W.H.O. CATEGORIES OF BLINDNESS [16]

<table>
<tr><td colspan="2" rowspan="2">CATEGORIES OF VISUAL IMPAIRMENT</td><td colspan="2">VISUAL ACUITY WITH BEST CORRECTION<br>( BETTER EYE)</td></tr>
<tr><td>MAXIMUM VISION (LESS THAN)</td><td>MINIMUM VISION (EQUAL TO OR MORE THAN)</td></tr>
<tr><td rowspan="5">BLINDNESS</td><td rowspan="2">3</td><td>3/60<br>FINGER COUNTING<br>AT 3 mtrs</td><td>1/60<br>FINGER COUNTING<br>AT 1 mtr</td></tr>
<tr><td colspan="2">OR VISUAL FIELD CONSTRICTED TO LESS THAN 10 DEGREE</td></tr>
<tr><td rowspan="2">4</td><td>1/60<br>FINGER COUNTING AT 1 mtr</td><td>P L<br>LIGHT PERCEPTION</td></tr>
<tr><td colspan="2">OR VISUAL FIELD CONSTRICTED TO LESS THAN 5 DEGREE</td></tr>
<tr><td>5</td><td colspan="2">NO LIGHT PERCEPTION</td></tr>
</table>

**Total blindness** of category V of WHO classification of blindness includes the person who are not able to see anything and not even light.

## CAUSES OF VISUAL IMPAIRMENT: [17]

### CONGENITAL

1.Toxoplasmic macular retino choroditis

2.Retinal dystrophies

3.Retinopathy of prematurity

4.Ocular malformation

5.Congenital glaucoma

6.Optic atrophy

7.Congenital cataracts,

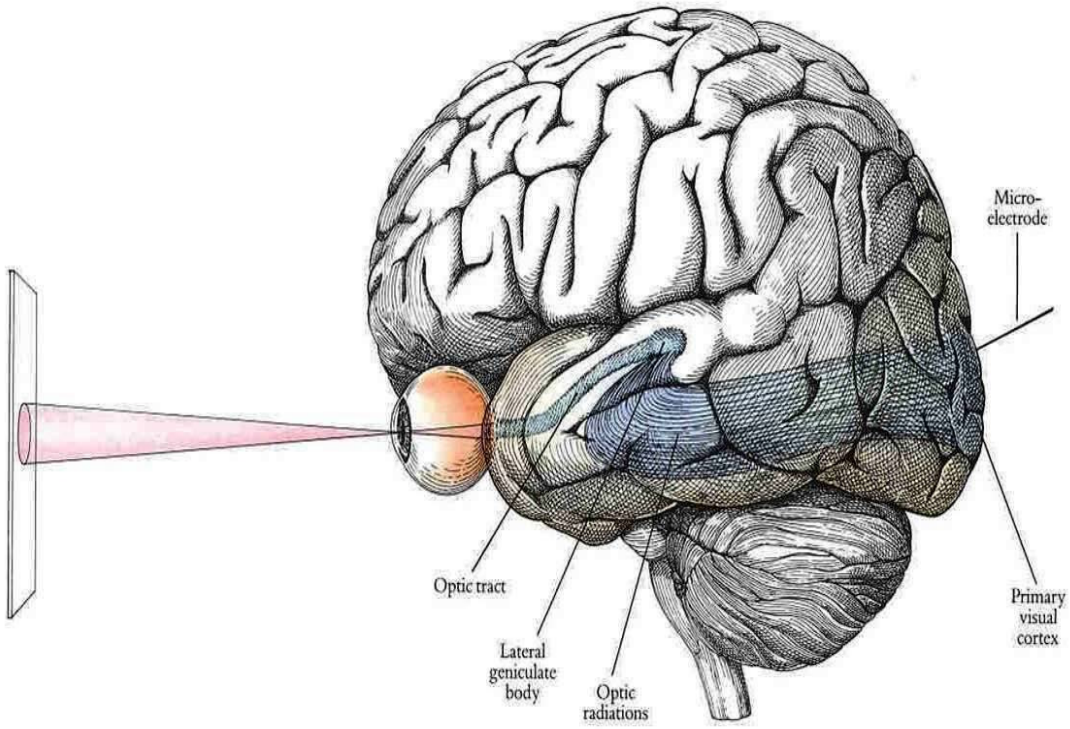
8.Opthalmia neonatarum

9.Vitamin A deficiency

### AQUIRED

Trauma

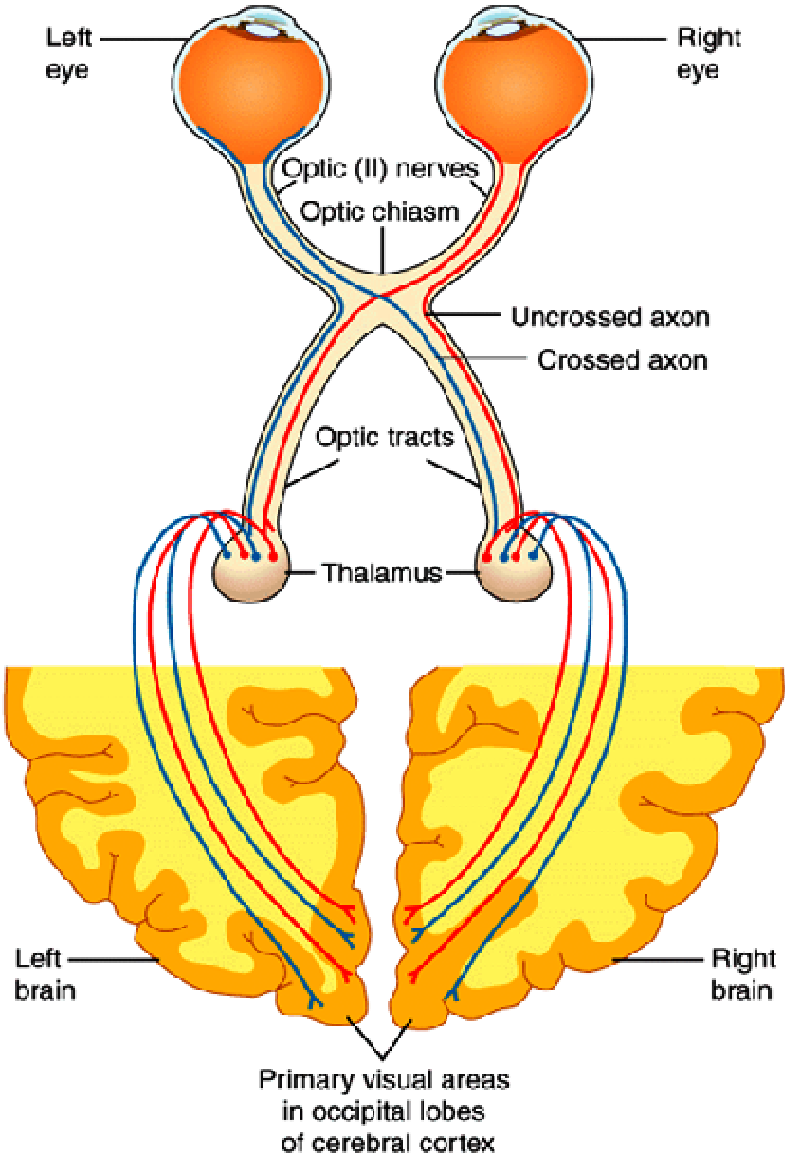
## VISUAL PROCESSING AND PRIMARY VISUAL CORTEX:[18]




Visual information reaches the visual cortex through the optic radiation which connects the lateral geniculate nucleus (LGN) to the primary visual cortex, (V1 or striate cortex).

The lateral geniculate nucleus is the major thalamic terminal for input into the visual cortex. In the lateral geniculate nucleus, a complete retinotopic representation of the opposite visual field is created. Information from the two eyes remains segregated.

## VISUAL PATHWAY[18]




Functionally V1 has six distinct layers. Layer 4 receives most of the visual input from the LGN. Visual information is processed contralaterally.

From V1 onwards, visual information flows through other cortical areas V2, V3, V4, and V5/MT[18].

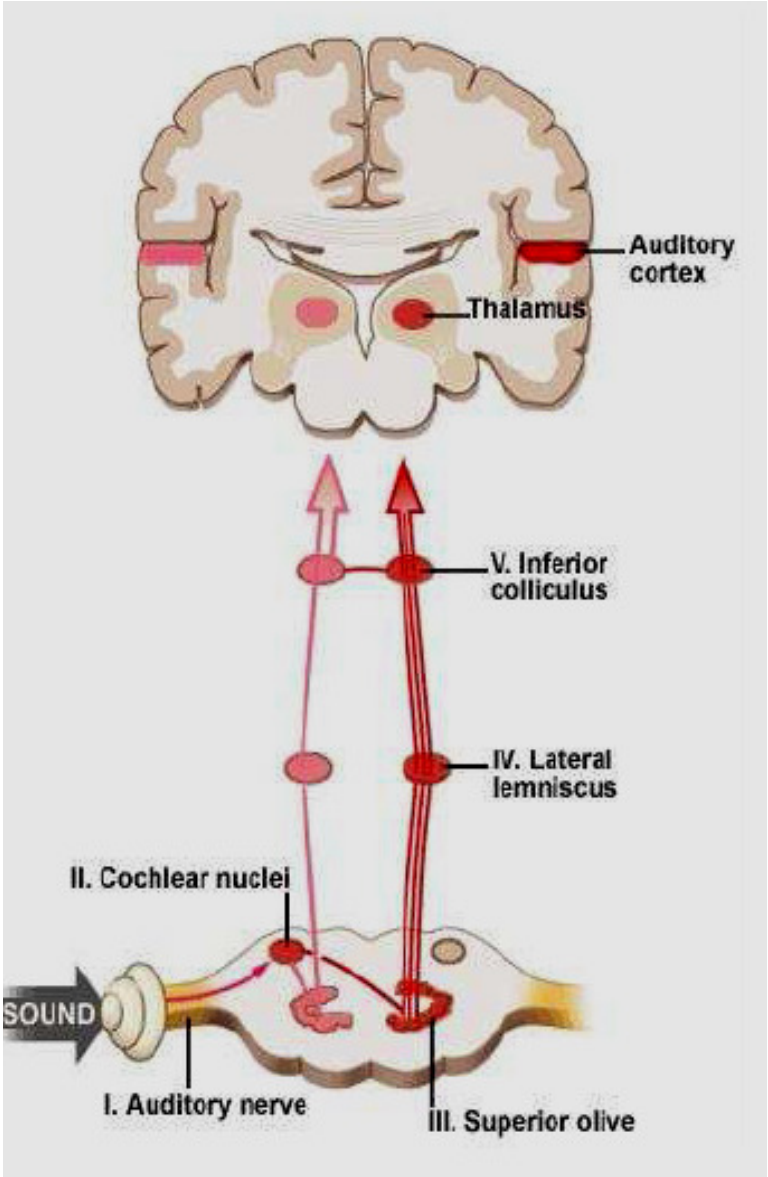
The primary visual cortex is situated at the posterior region of the Occipital cortex. In V1, neurons fire only when stimulated by a certain arrangement of active presynaptic cells like stimulation by a bar of light in a certain orientation. Similarly, basic information about e.g. colour is processed here and in V4[18]

Secondary visual areas (V2-V5), also named extrastriate visual cortex, process other visual primitives. As visual information is transferred onwards through visual hierarchy, the complexity of the processed stimulus features increases. WhileV1 responds to e.g. a line of light in a specific location and orientation, neurons in the lateral occipital complex respond selectively to complete objects, and other parts in the visual association cortex respond selectively to faces from a particular species or motion (V5).

As complexity increases, the level of processing of specialization is augmented. So, visual processing occurs simultaneously in two separate pathways i.e. dorsal and ventral pathway [19].

The dorsal pathway connects primary visual cortex and the posterior parietal cortex and it is essential for spatial localization. The ventral pathway responsible for object recognition leads to the inferior temporal gyrus (Kandel et al., 2000; Pinel2001[20,21]

# AUDITORY PATHWAY




In human sound reaches the brain via the ear. The frequency of sound that, humans can hear ranges from 20-20.000 Hz .Then, it travels through the outer, middle, and inner ear. [22]

In the Organ of Corti, the mechanical sound waves are converted into electric neural signal. Then, the dendrites of primary auditory neurons communicate with these signal. Later they are bundled into cochlear nerve after which they join the vestibular nerve and form vestibule cochlear nerve. The information is transferred through cochlear nuclei , superior olivary complex ,inferior colliculus and the lateral lemniscus to the thalamus.

At the level of cochlear nuclei, the sound waves cross to the contralateral side. Similarly, the superior olivary complex in brainstem allows sounds to be localised on azimuthal axis, depending up on intensity cue and auditory interaural delay it receives from both ears.

The inferior colliculus within the midbrain is divided into dorsal part nucleus and the central nucleus. The dorsal part receives both somatosensory and auditory input. The central nucleus is involved in auditory localisation.[22]

Within the thalamus, there is an oval structure found within diencephalaon which conveys sensory input to the medial geniculate nucleus (MGN).As it act as a primary sensory area it represents a major relay station for auditory system.

The medial geniculate nucleus ( MGN) consists of three subdivisions. Among which, the principal nucleus receives the auditory input. The other divisions receive multi-modal input. The principal nucleus of Medial Geniculte Nucleus is tonotopically arranged and cells are accurately tuned to particular frequency (Kandel, Schwartz, & Jessell, 2000; Pinel,2001).[20, 21]

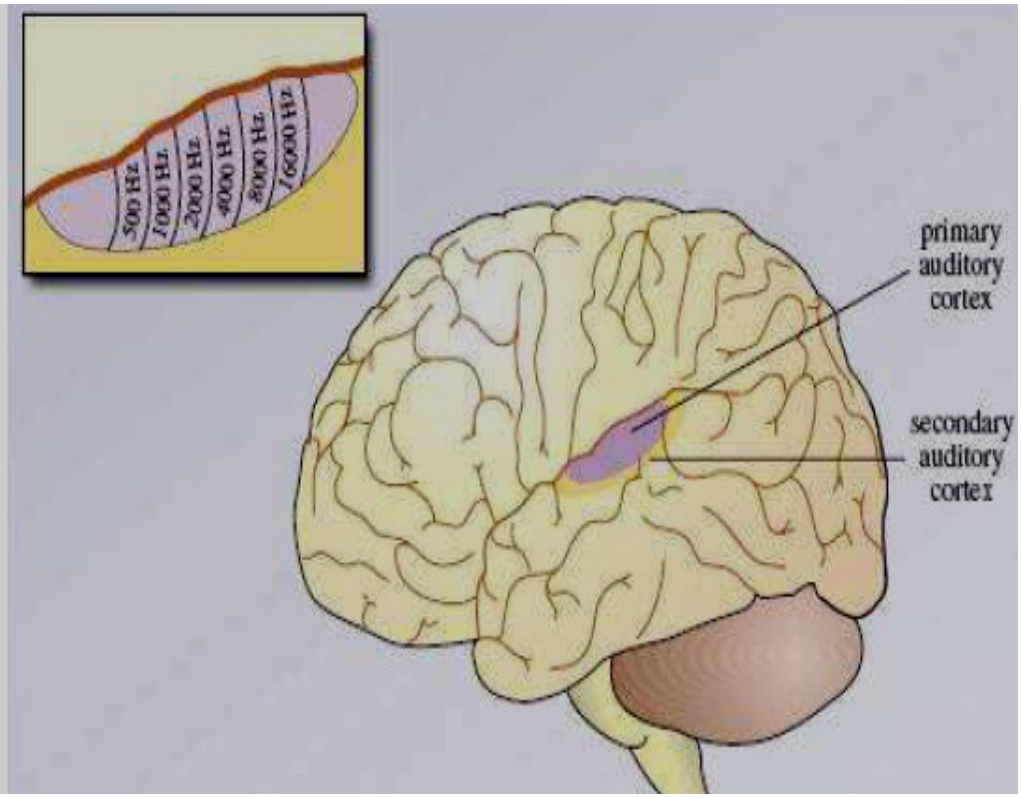
From median geniculte body, then the information is relayed to primary auditory cortex .It is located in transverse gyrus of Heschl which is situated within temporal lobe. This, auditory cortical area allows the sensation of auditory characteristics like pitch.

Thus, primary auditory cortex is composed of many functional columns (Schreiner, 1992)[23]. Neurons found within same column process sounds of same frequency. Further, they are tonotopically organized in similar to all the previous stages of auditory processing mentioned above.

Auditory neurons are spatially arranged in an order according to auditory frequencies they process. There is more evidence that its distinction into two different pathways with peculiar functions like in the visual cortex, can be found in auditory cortex. ( Kaas & Hackett, 1999; Rauschecker & Tian, 2000).[24,25]

Acoustic signals are recognized and distinguished as music speech, etc. after processed by Wernicke's area, in auditory association cortex found within temporal lobe. Besides primary auditory cortex and Wernicke's area, so many other areas of brain process sound information.

## AUDITORY PROCESSING AND AUDITORY CORTEX[22]



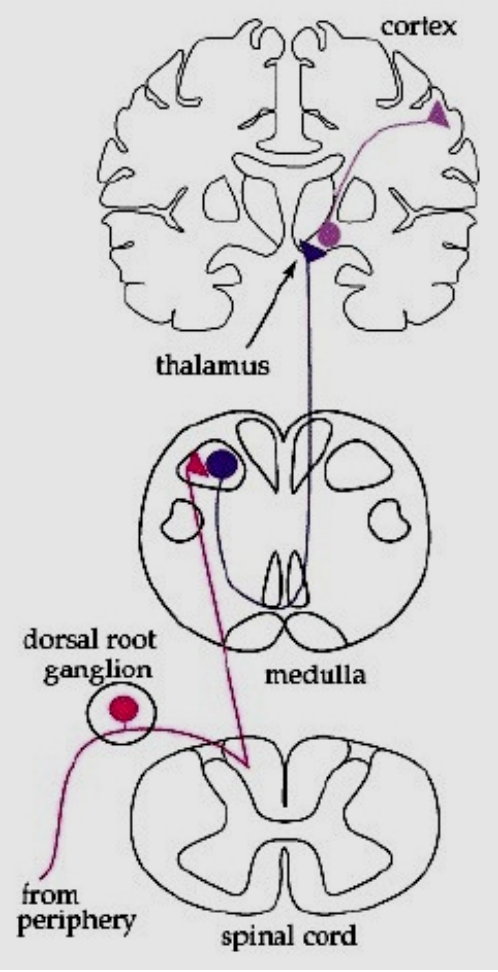

The auditory pathway is composed of cochlea and auditory processing centre the primary auditory cortex(A1) located within the brain along with the neurons and relay nuclei linking them.

When waves of sound reach cochlea, inner hair cells present on basilar membrane transduce these sound waves into electrical impulse. The frequency of the incoming waveform decides which parts of the basilar membrane with inner hair cells to be stimulated. Hair cells present at the base respond to high frequencies and those at the tip of basilar membrane respond to low

frequencies. Hence, the frequencies of the waves are mapped from low to high on the basilar membrane. This type of presentation is called 'tonotopic map.

In the auditory pathway, this topographic representation is replicated at successive levels. They act as a common site for neuroplasticity. The first region to receive auditory input is the primary auditory cortex which is tonotopically presented. From this area, increasingly complex feature of detection takes place when information moves towards the secondary (A2) and tertiary (A3) auditory cortexes , before it reaches auditory association area.

## SOMATOSENSORY SYSTEM [26]



The **somatosensory system** is composed of different sensation from the body like pain, light touch, temperature, pressure, muscle and joint position sense called proprioception In the spinal cord, these sensations are grouped into three different pathways with different targets in brain.

D**iscriminative touch** sensation includes pressure, touch, and vibration perception. It helps to read raised letters with fingertips and to describe the texture and shape of an object without visual clue.

**Pain and temperature**, sensation includes the sensations of tickle and itch.

P**roprioception** sensation includes receptors for , joint position, muscle stretch and tendon tension.It is responsible for sensation happening below body surface. This sensation primarily targets to the cerebellum.It needs feedback minute-by-minute by which the muscles are working. [26]

These sensations differ not only in their pathways, receptors and targets but also in their crossing level. Before going to the cerebral cortex, all sensory system will have to cross over at some level. The cerebral cortex generally operates on a contralateral basis. [26]

Discriminative touch sensory system crosses at the level of medulla. Pain sensory system crosses at the level of spinal cord. Proprioceptive sensory system of same side goes to the ipsilateral cerebellum, without crossing to opposite side.

Sensation from the periphery travel via sensory axons. All sensory neurons have cell bodies of their own. They are situated outside spinal cord in the form of clump. They are called **dorsal root ganglion**. Every spinal nerve has one such ganglion. The sensory neurons are peculiar because the signal will not pass through cell body. Instead of that, cell body sits on to one side but without dendrites. The signal then passes from distal axon process to proximal process directly.

Proximal end of axon enters into the dorsal half part of spinal cord. There it turns up the spinal cord towards brain. These axons constitute **primary afferents.** They are so called because they brought the signal into the spinal cord. Generally, the meaning of afferent is towards the brain and efferent is away from brain The axons usually ascend in dorsal side of white matter of spinal cord.

Finally, at the level of medulla, primary afferents synapse. The neurons that receive the synapse are called as secondary afferents. Immediately, the secondary afferents cross to form a new tract on the opposite side of brainstem.

This secondary afferent tract will ascend to thalamus and finally enter into cortex. In the thalamus, they synapses to form third order final neuron which will finally go in to the cerebral cortex.

The location of the pathway in the spinal cord has several names. The tracts on dorsal side of the spinal cord a called dorsal column or posterior column. The posterior column can be divided into two tracts namely, gracile fasciculus and cuneate fasciculus

The collection of axons situated in midline is tall and thin and is called gracile fasciculus. The wedge shaped outer tracts are called cuneate fasciculus.

The gracile fasciculus carry the information from lower half of body including legs and trunk. The cuneate fasciculus carry information from upper half including trunk and arms That is why we can see the cuneate fasciculus only at level of thoracic and cervical areas, though gracile fasciculus moves down into sacral cord.

At the level of medulla, gracile fasciculus axon synapses in gracile nucleus, and cuneate axon synapse in cuneate nucleus. After leaving these nuclei, these secondary afferents immediately cross, and line up along ventral medulla to form the medial lemniscus . Then it will ascend through the brainstem.

In pons, as the pontine nuclei enlarge beneath it, the medial lemniscus becomes flattened out. In the midbrain, the medial lemniscus is pushed away up dorsally and laterally to enter into the thalamus

In thalamus, the secondary afferents synapse to form ventrolateral posterior nucleus . This thalamocortical afferents travel through internal capsule to primary somatosensory cortex.

Primary somatosensory cortex is situated in post-central gyrus. It is nothing but a fold of cortex located posterior to central sulcus.

## NEUROPLASTICITY[27]

The capacity of the nervous system to modify its organization. These changes occur as a consequence of the normal development and maturation of the organism, the acquisition of new skills in immature and mature organisms, after damage to the nervous system or as a result of sensory deprivation

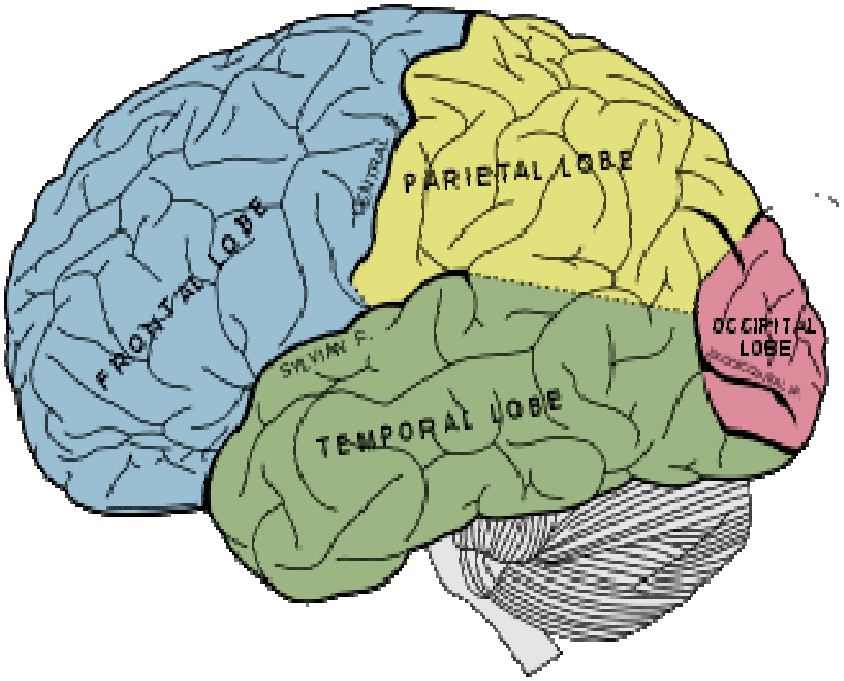
**Types of neural plasticity**:

1.Developmental

2.Intramodal

3.Crossmodal

## CROSS MODAL PLASTICITY[28]




**Cross modal plasticity** is the reorganization of neurons to integrate the function of two or more sensory systems by reorganising connections between the four major lobes in response to sensory loss.

Cross modal plasticity is a type of neuro plasticity and often occurs after sensory deprivation due to disease or brain damage. The reorganization of the neural network is greater following long-term deprivation of sense, such as congenital blindness or pre-lingual deafness.

In these circumstances, cross modal plasticity can strengthen other sensory systems to compensate for the lack of hearing or vision. This strengthening may be due to new connections that are formed to brain cortices that no longer receive sensory input.

## PLASTICITY IN THE BLIND[29]

Even though the blinds are not able to see, their visual cortex is still in use, with the information received from areas other than visual input.

Studies found that the volume of white matter with myelinated nerve connections were reduced in the optic tract, but not in the primary visual cortex itself. But, grey matter volume was reduced by 25% in primary visual cortex. The atrophy of grey matter with the neuron bodies, may be due to its connection with optic tract[30]. As, the eyes are no longer receive visual input, the nonuse of the concerned optic tract can cause loss of grey matter volume in the primary visual cortex. Similarly, the white matter is also atrophied. But, the primary visual cortex is less affected.

For example, blind individuals show enhanced attentional and perceptual sensitivity for identifying different auditory stimuli. Spatial detection of sound can be altered in the early blind by producing a virtual lesion in visual cortex by using transcranial magnetic stimulation[31].

The somatosensory cortex can recruit the visual cortex in assisting the tactile sensation. Thus, the cross modal plasticity rewind the network structure of the brain which in turn lead to more connections between somatosensory and visual cortex.

Besides this, the somatosensory cortex is acting as a hub region for nerve connections in the brain for the early blind and not for the sighted.[32] Using this cross-modal network, the early blind can react to tactile stimuli with greater speed and accuracy, as they are having more neural pathways to work.

The portion of the visual system that the somatosensory cortex can recruit is dorsal-visual stream. This dorsal visual stream is used by the sighted to recognise spatial information visually but the early blind use it for identification of 3D objects by tactile sensation[33].

However, both sighted and blind participants use the dorsal stream in processing spatial information, suggesting that cross modal plasticity in the blind re-routed the dorsal visual stream to coordinate with the sensation of touch rather than changing the overall function of the stream.

**EXPERIENCE DEPENDENCE**:

There are evidences that the level of cross modal plasticity between the somatosensory and visual cortices are dependent on experience. In a study by using tactile tongue devices to transmit spatial information, early blind

individuals were able to show activation of visual cortex after 1 week of training with the device[34].

Although, there were no cross modal connections at the beginning, later, the early blind could develop connections between the visual cortices and somatosensory while sighted controls couldn't to. Early or congenitally blind individuals have stronger cross modal connections when they began learning Braille at the earliest[35].

Earlier start of learning, allows for stronger connections to form so that early blind children can grow up using their sense of touch to read instead of using their sight. Sensory testing studies have shown that tactile spatial acuity is enhanced in blindness[36],[37] due to cross modal connections and this enhancement is experience-dependent[38].

## NON INVASIVE STUDIES FOR NEURAL PLASTICITY

### MAGNETIC RESONANCE IMAGING (MRI):

It is a non-invasive technique and it uses radio waves and magnetic fields in the place of ionising radiation.

Structural MRI method will allow creation of images of anatomical structures with excellent spatial resolution.

Magnetization transfer is very sensitive to the myelin content and hence it is useful in diagnosing early demyelination problem.

## FUNCTIONAL MAGNETIC RESONANCE IMAGING:

FMRI is an MRI-based technique that allows us to indirectly measure the activity of the brain. It was introduced in 1992 when several laboratories independently identified a mechanism that could be used for such non-invasive measurement (Bandetini, Wong, Hinks, Tikofsky, & Hyde, 1992 Frahm, Bruhn, Merboldt, & Hänicke, 1992; Kwong et al., 1992; Ogawa et al., 1992).[39.,40,41,42]

FMRI detects changes in blood flow and oxygenation occurring in response to neural activity. Underlying is the phenomenon that the brain area has an increased metabolism when active.

The physiological mechanisms underlying plastic changes in cortico-cortical connectivity will be seen as a non-invasive imaging approach here.

Developmental changes in local connectivity include pruning of the exuberant connections and masking of silent synapses.These changes differ between healthy and visually deprived individuals. (Bavelier & Neville,2002; Maurer, Lewis, & Mondloch, 2005)[43,44]

It has been suggested that opening of pre-existing connections and the shift in their connectivity might be the underlying rapid, early plastic changes e. (Merabet et al., 2008.[45]

If sustained and reinforced, these can lead to slower and more rigid structural changes like dendritic arborisation, sprouting and growth with rewiring of connections (Pascual-Leone, Amedi, Fregni, & Merabet, 2005). [46]

This line of thought would also fit with evidence, the functional crossmodal processing in the occipital cortex in sighted humans (Merabet et al., 2004; Zangaladze, Epstein, Grafton, & Sathian,1999).[47,48]

It may be possible, that the same connections underlie crossmodal processing in sighted but they remain comparably suppressed under conditions where vision is present.(Macaluso, Frith, & Driver, 2000; Merabet et al., 2004; Zangaladze et al., 1999). [48,49,50]

The earlier the sensory loss, the more striking are the neuroplastic effects found (Hensch, 2005).[51]

By this one can say that input can determine which connection gets either pruned or suppressed and which is left unchanged (Sharma, Angelucci, & Sur, 2000; Sur et al., 1988;von Melchner, Pallas, & Sur, 2000). [52,53,54]

If a person had no visual input, the normal pruning process could be disturbed, leaving exuberant connections, as also indicated by recent findings of increased thickness of the visual cortex of blind volunteers (Jiang et al., 2009).[55]

## PET (POSITRON EMISSION TOMOGRAPHY) & SPECT (SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY):

They are useful in evaluating structural lesions of the brain.

**Ptito M, et al** [56] in a PET study demonstrated the cross-modal plasticity in blinds by electro-tactile stimulation of tongue,By this he had shown that occipital area is a part of neural network in discrimination of touch sensations in association with posterior parietal cortex since there was an increase in regional blood flow(RBF) in occipital cortex and that this increase was attributed to the performance of particular task.

## ELECTROENCEPHALOGRAPHY: (EEG)[57]

Electroencephalography is a non-invasive technique in which the brain's activity is recorded from the scalp to evaluate the function of the brain. Thus are used to measure the brain electrical activity. The EEG displays spontaneous brain activity as a continuous graph of voltage and frequency changes occurring over time.

## EVOKED POTENTIALS:[57]

Evoked potentials are the electrical potentials difference recorded from the response to stimuli . Evoked potential recordings are useful in evaluating lesions in the afferent pathways under study. They assess the functional integrity of these pathways. These studies sometimes reveal the abnormalities missed by magnetic resonance imaging and vice versa. In patients with known

CNS pathology, evoked potentials studies may help to detect the lesions and also detect the structural abnormalities in many disorders

## BRAIN STEM AUDITORY EVOKED POTENTIAL:

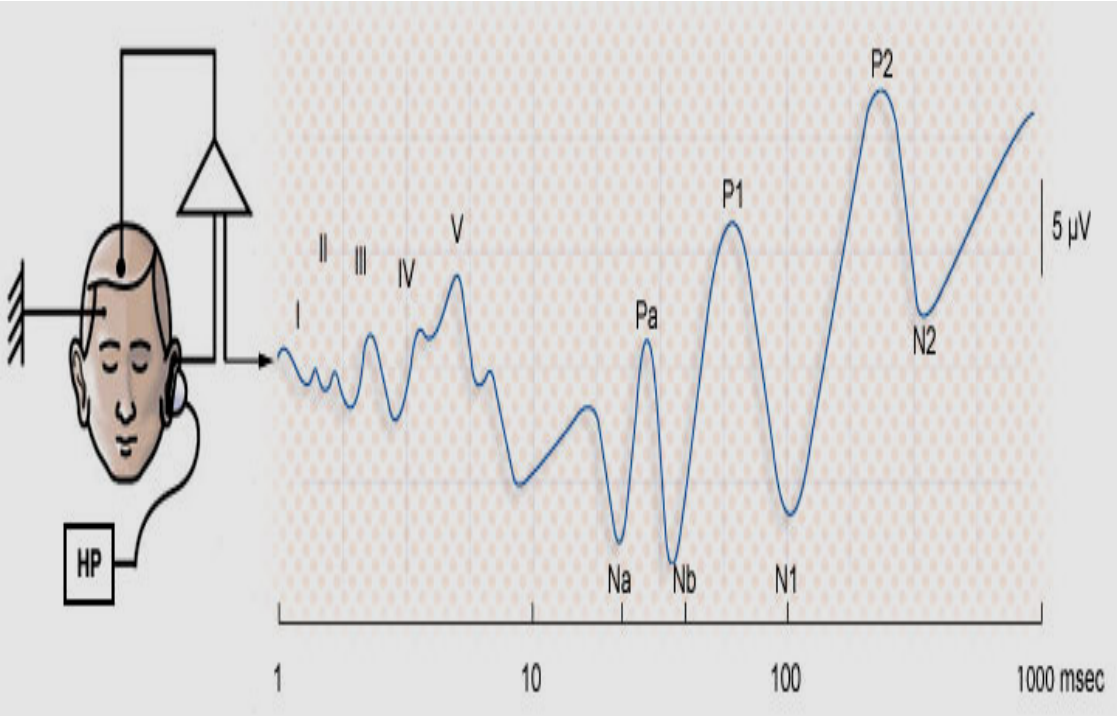
They are the potentials recorded from the ear and the scalp as a response to brief auditory stimulus in order to assess the integrity of the auditory pathway up to midbrain.

The evoked potentials that appear after transduction of the acoustic stimulus by the ear cells create an electrical signal which is carried via the auditory pathway to brainstem and from there to cerebral cortex . It comprises five or more waves within 10ms of the stimulus [58]. It may also described in relation to duration of onset of response [59.)]

This is useful to study noninvasively the function of the auditory system, like cochlea-auditory nerve pathway.This is responsible for extensive development of scalp recording of near and far field potentials

## EARLY LATENCY AUDITORY EVOKED POTENTIALS :[59)]

Early auditory evoked potentials is also called short latency auditory evoked potentials .It occurs within the first 10-12msec after an auditory stimulus.




**MID LATENCY AUDITORY EVOKED POTENTIALS**:

They are the potentials recorded between 12 m sec and 50 m sec after auditory stimulation. It can be recorded from either from transient or from high frequency stimuli. Mid latency auditory evoked potential is clinically useful in the assessment of threshold of hearing in infants and children and in the identification of central auditory pathway dysfunction and in evaluation of the central auditory pathway for cochlear implantation.

## LATE AUDITORY EVOKED POTENTIALS:

The waves that occur at 50msec or more after are called slow or late auditory EPs. They can be subdivided into

exogenous waves: 1. N1

2. P1

3. P2 which depends on external stimulus characters, and endogenous waves :

1. P300

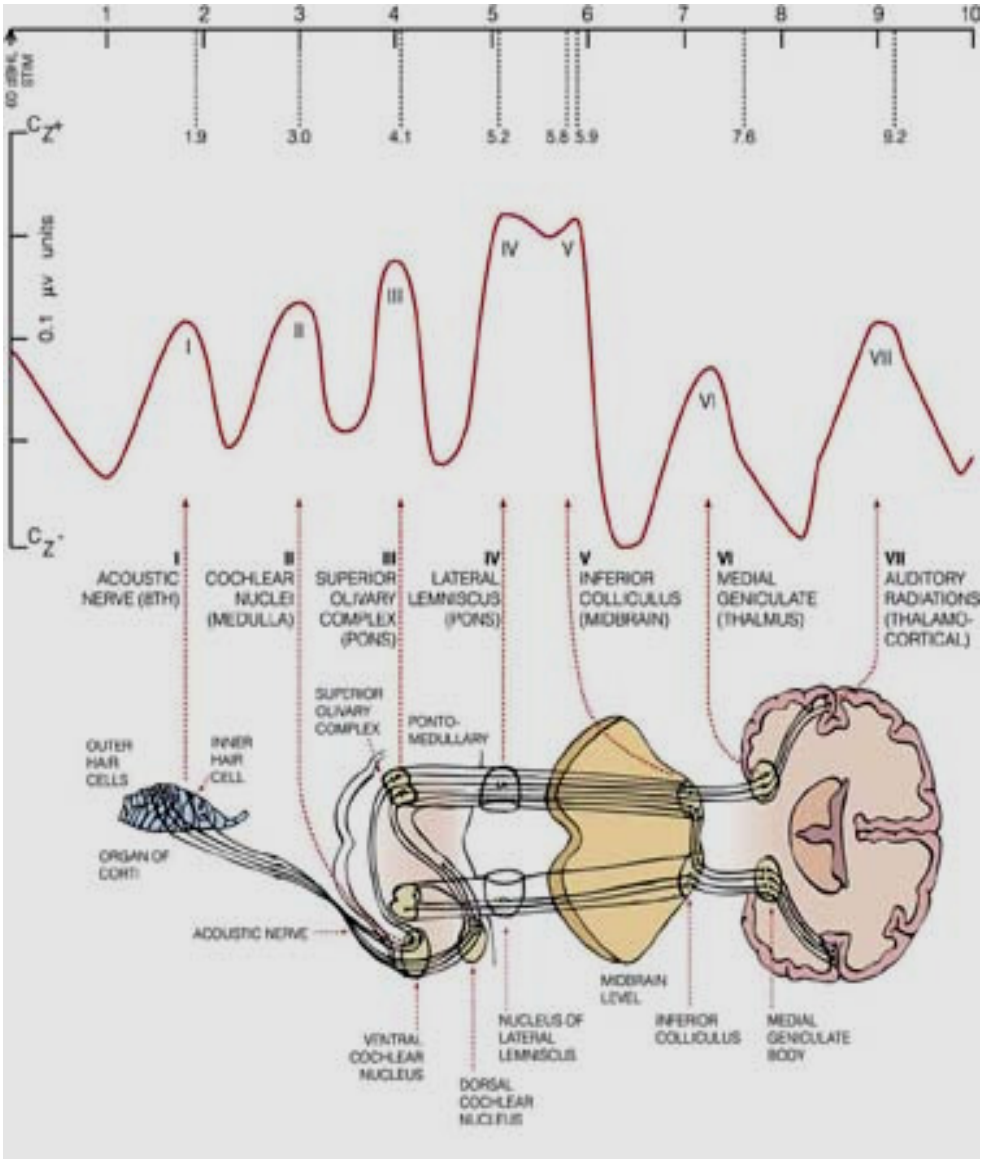
2. N400

3. cNV and the mismatch negativity, which depends on internal cognitive processes [60]

## NORMAL BRAINSTEM AUDITORY EVOKED POTENTIAL FINDINGS:

It consists of five or more distinct waves recorded within 10-12 ms of auditory stimulus. They are generated in different areas of the peripheral and central auditory pathways. This can be understood from the following diagram.

**BAER WAVE GENERATORS :**[61]




Wave I originates from the peripheral area of auditory pathway close to cochlea. Wave II originates from cochlear nucleus,

Wave III originates from superior olivary nucleus,

wave IV originates from lateral meniscus.

wave V originates from inferior colliculi [61]

The absolute latency, inter peak latencies and amplitude of wave forms of BAEPs were measured.

## INTERPRETATION OF BAEP:

It requires identification and measurement of waves I, III, and V and the measurement of I-V , I-III and I-V inter peak intervals. These values should be compared with the normal values for age and sex matched individuals.

Absence of wave I with normal wave V probably reflects technical problems in recording.

Absence of wave III is significant only when wave V is also missing or delayed.

Waves cannot be interpreted without checking the patients hearing status. Because conductive hearing loss and cochlear pathology may profoundly affect BAEP wave, latency and amplitude.

Latency-Intensity function is useful in differentiation of certain types of pathologies like

1. Conductive hearing loss is characterised by prolongation of wave I and wave V with latency- intensity curves parallel to the normal curve. The I-V and I-III inter peak intervals are normal.

2. Cochlear hearing loss is characterized by a recruiting curve for wave I which is either normal or mildly prolonged wave I latencies with loud clicks and greater delays with decreased intensity, resulting in a steep curve. Wave V is not completely affected. and its curve is little steep, resulting in a shortening of I-V interval

3.In retro-cochlear deficit type I , Wave I is prolonged with a deep latency-intensity function; Wave V is also prolonged; so the I-V interval is prolonged. This abnormality has been reported in lesions affecting the eight nerve.

4. In retro cochlear deficit type II wave I latency-intensity curve is normal. wave V and the I-V inter peak interval are prolonged. The latency-intensity function of wave v and the I-V interval is variable.

A delayed wave V with normal wave I latency indicates that the delay has occurred after wave.

Another type of abnormal BAEP is seen as normal wave I and absence of succeeding wave[60]

BAEP findings may be abnormal at a time when imaging studies show no definitive abnormality

## CLINICAL APPLICATION:

1.BAEP is useful in evaluating the integrity of the peripheral and central auditory pathway [60].

2.BAEP is used to detect subclinical brain stem pathology also[62]

3.To assess the hearing in uncooperative patients and very young children.

4. To detect the degree of hearing loss in infants [63]

**Anbarasi et al in 2012** showed that the shorter absolute latency of wave V of early evoked potential indicates the evidence of enhanced hearing ability and hence cross-modal plasticity at the level of inferior colliculus in blind people.[64]

**N. K. Manjunath et al in 1998** inferred that the peak latencies of the wave Pa and Nb were significantly reduced in congenitally blind subjects. [65]

**Naveen et al in 1998** have found that the Na and Pa middle latency auditory evoked potentials (MLAEP) were not significantly different in congenitally blind subjects when compared with the same values in normal sighted subjects but the Nb component of MLAEP had a significantly shorter latency in congenitally blind persons.[66]

**Manjula P1, Bharath T in 2014** have studied the evidence of neuro plasticity in the form of decreased latencies of MLAEP in the visually deprived, suggestive of much better information processing in the auditory system. [67]

**Anbarasi et al in 2014**,showed significantly shorter wave latencies for waves Po, No and Pa among males and for the wave Pa among females, an evidence of improved auditory activity in the dorso-medial part of Heschl gyrus in the areas of medial geniculate body and polysensory nuclei of the thalamus in the blind. [68]

**Fatemeh Heidari et al in2009** showed in their auditory evoked potential study, reduced latency of P300 in early blind subjects in comparison to sighted subjects due to faster rate of automatic processing and information categorization is faster because of sensory compensation. [69]

## SOMATOSENSORY EVOKED POTENTIAL:

These potentials are formed by large diameter sensory fibres on application of stimulus anywhere along their course, either in peripheral portion or in central portion of its pathway.[63]

Even though some fibres may follow extralemniscal pathway, it varies upon the functional integrity of group II cutaneous afferent fibres ,the fast-conducting large diameter group IA muscle afferent fibres and group II and on the posterior column of the spinal cord.[62]

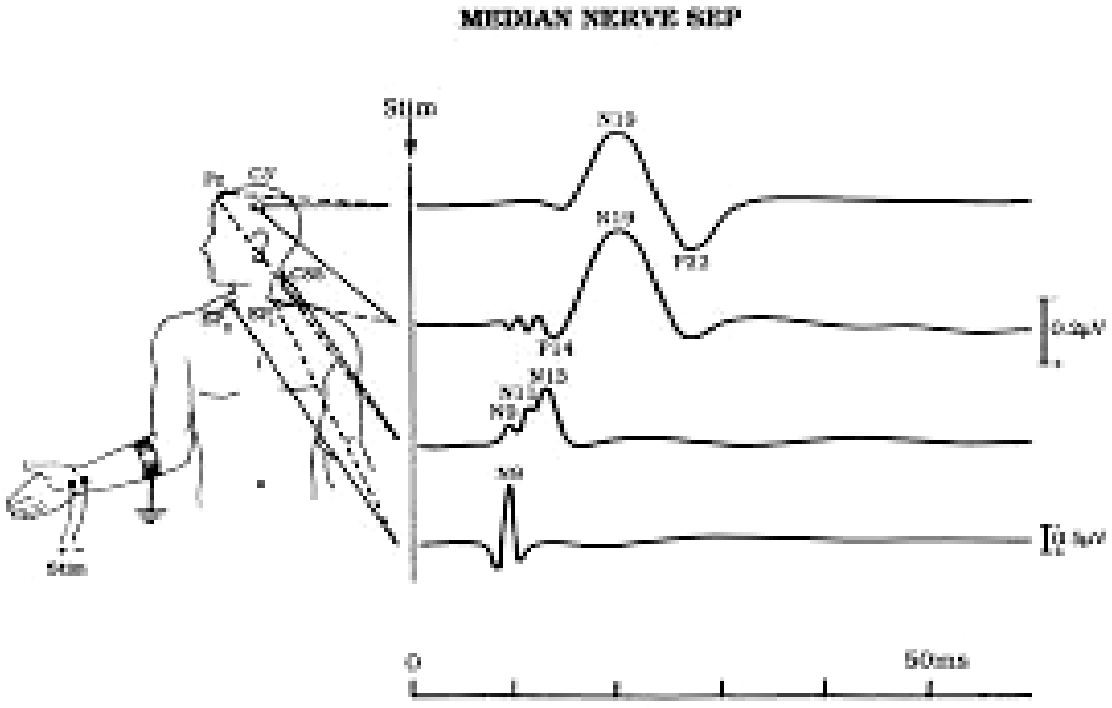
The potentials recorded have varied duration of latencies and they are named as short, intermediate and long latencies

Somatosensory evoked potentials are usually elicited by application of electrical stimulus to either median nerve or posterior tibial nerve.

**Normal SEP Findings**:

SEP components are defined by their polarity and latency.

**Components of median nerve SSEP:**




The components of median nerve SSEP recording useful in clinical interpretation are:

N9 - recorded as the afferent volley traversing the brachial plexus.

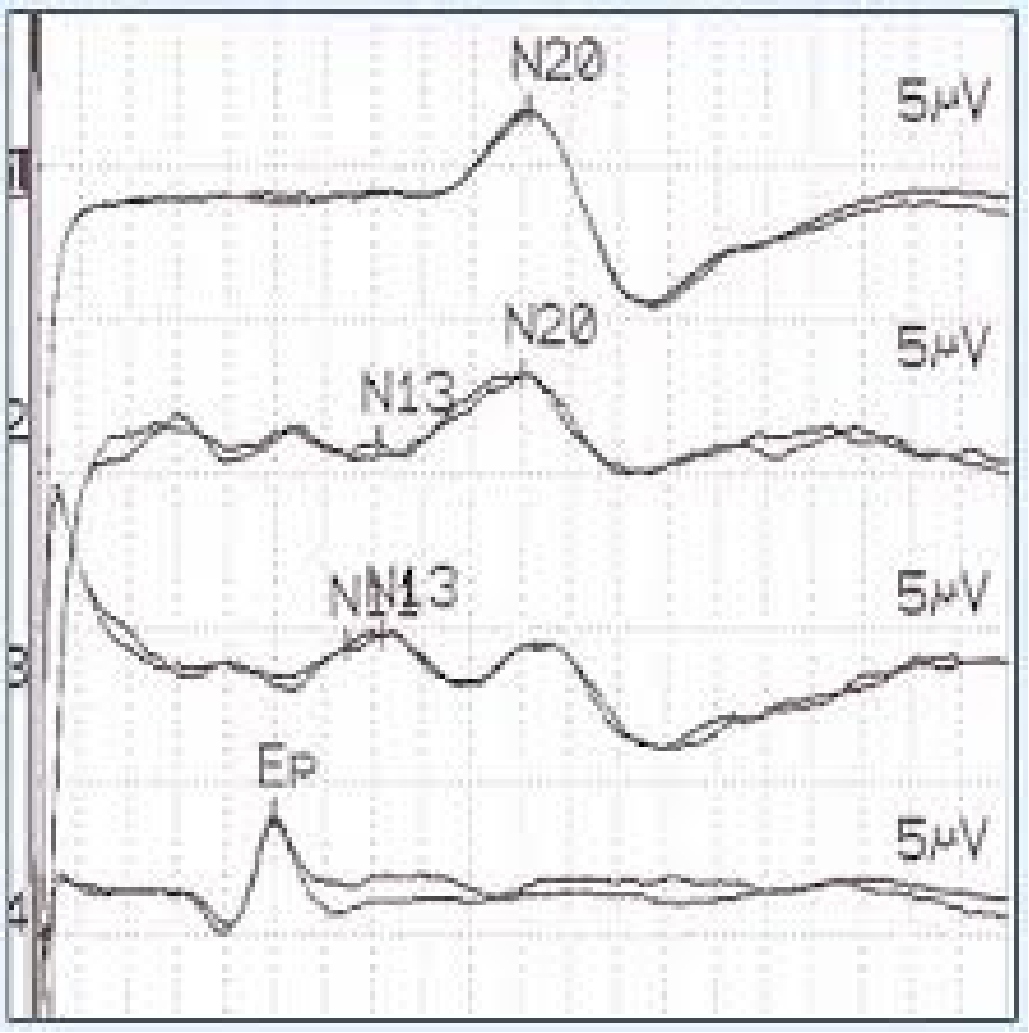
Nl3 - post synaptic activity in the central gray matter at the level of the cervical cord.

P14- generated from the lower brainstem, mostly from caudal medial lemniscus.

N18- post synaptic potentials arising in the rostral brain stem.

N20- indicates activation of the primary cortical Somatosensory area.

**Normal Median SEP:**




1. Erb potential, described as N9, is seen as a principle negative peak in EPi-EPc channel.

2.Spinal potential (Nl l) is seen as negative peak. It is recorded at spinous process of fifth cervical vertebra referred to as Epc.

3.The following negative wave is high in amplitude and is called as N13. Children in age group between 1 and 4 years usually have a prominent N11.

4. The P l4 Potential is seen as positive peak. It is widely distributed over the scalp and it is best recorded from Cc-.EPc (Scalp - non cephalic channel). It may be of low amplitude and inconspicuous in all recording channels in some normal individuals. It usually occurs 1milliseconds after N13.

5 .N l8 is a negative peak widely distributed over the scalp.It begins before N20 and usually evident in Cc-Epi and Cc-Epc channel.

6. N20 is seen as negative wave in cc-Fz and Cc-Ep. It is usually identified as portion of negative potentials, which is just preceding the sharp drop off towards p25 which is the succeeding cortical positive peak

7.N35,P45 and N60 is seen as series of cortical potentials after 40 ms.

**Parameters measured for analyzing median SEP:**

1..Latency

2.Peak Latency.

3.Amplitude

N9 latency is measured from stimulus artefact to its peak to the following positive deflection in EPi-EPc channel.

N13 latency is measured from the stimulus artefact to the peak in the C5Sp-EPc channel The amplitude can be measured from peak of Nl3 to the next deflection.

N20 latency is measured in Cc-Fz channel upto the point of maximum negativity just prior to the steep drop of P25 trough.

There are two important inter peak latencies (IPL), which are of clinical significance.

1. Brachial plexus to Spinal cord (N9-N13).

2.Certral Sensory Conduction Time (Nl3-N26)

**MEASUREMENT OF CONDUCTION TIME:**

The advantage of SEP recording in clinical routine is useful in calculation of the conduction time of the ascending volley in the central segments of the somatosensory pathways.

## UPPER LIMB SEPS:

Various montages and procedures have been adopted to measure the conduction time depending on whether to detect a slow conduction or to locate the site where conduction velocity has slowed down.

The conduction in the proximal segment of brachial plexus roots can be calculated by measuring the interval between the peaks of the supraclavicular N9 and the spinal Nl3 potentials.

Central conduction time is calculated by measuring interpeak between N13 and N20(54)

**Dayanand G et al in 2008** showed that there was increase in amplitude of N20 Potential & the increase was more on the right or dominant side14[70]

**Alvaro et al in 1993** showed in their Somatosensory Evoked Potential Study on right-sided stimulation there were significant increase in N20 & P22 suggesting activity dependant alteration in spatio-temporal components of signal processing[71]

**Dayananda Giriyappa etal in 2009** have demonstrated that totally blind Braille readers have larger N20 amplitude, suggestive of greater somatosensory cortical representation of the braille reading index finger.[72]

**Elbert T et al in 1995** have reported in their studies, decreased Somatosensory Evoked Potential latencies in totally blind individuals on recording Event related potentials . These findings were found to be linked with increased attention which in turn leads to quick processing of information while performing discrimination tasks of event related potentials.[73]

**Nikhat et al in 2013** showed decrease in latencies with increase in amplitudes at the level of cortex in somtosensory evoked potential due to improved information in the nervous system and the extent and synchronization of neural network involved in vision processing.[74]

# MATERIALS AND METHODS

## Type of study

This comparative, cross-sectional study was done in the department of Physiology Thanjavur Medical College, Thanjavur. This study was approved by Institutional Ethical Committee

## Subject selection

The study group consisted of 40 blind females of category 5 in menstrual age group ranging from 17 to 40years, recruited from local community , after getting informed and written consent. Subjects with any auditory pathology, known diabetes, mental retardation ,long duration of anaemia , those were on long term medication that may affect hearing and those who had undergone any kind of rehabilitation were **excluded** from the study.

Control group consisted of 40 age and sex matched students with normal vision who were from local community . For the control group same exclusion criteria were followed

## Methodology

The entire procedure was divided into history taking, examination of systems and recording of evoked potentials. It was done in the day time from 10.00 AM to 4.00 PM in the department of Physiology ,Thanjavur Medical College, Thanjavur.

General history details of the subjects, their educational status , any training, cause and duration of blindness, auditory problems, diabetes mellitus and other co-morbid conditions were asked in the study group subjects.

Simultaneously similar history except for details on blindness was elicited in control group subjects.

Usual anthropometric measurements (height and weight) were taken in both groups.

General physical examination, Systematic examination including cardiovascular system, respiratory system and central nervous system were done in both groups. Both the control and study group subjects were then evaluated for any ENT pathology by tuning fork tests and otoscopic examination and also for the presence of wax, if present it was promptly removed by appropriate treatment.

**Exclusion criteria:**

1.Diabetes mellitus.

2.Neurological disorders

3.Psychiatric illness.

4.Seizures.

5.Hypertension

6.Rehabilitation training

## EVOKED POTENTIAL RECORDING :

Both the groups of subjects were subjected to BAER and SSEP. The apparatus used in our study is MEDICAID systems Neuro perfect plus(EMG,NCV, EP)2000.

The evoked potentials were recorded in a closed dark room free from external noise. Electrodes Placement and recording of potentials were followed in accordance with10-20 electrode placement system .[61]

## Assessment of vision

The subjects of the study group showed absence of perception of hand movements and perception of light.

## Recording of BAER

The subject is seated comfortably. The electrical montage used for testing is based on 10-20 electrode placement system[61]

Channel 1: Cz-Ai

Channel 2: Cz-Ac,

Ground:20% from nasion Fz.

Head phones are placed on the ears for delivery of the auditory stimulus.

Examination of external ear, Rinne's test, and Weber's test were carried Out.

Subject is made to fully relax

Room should be quite and comfortable.

**Instrument setting for BAEP:**

| Settings | BAEP |
|---|---|
| Sweep | 5 msec |
| Sensitivity | 10µV |
| Low cut | 100 Hz |
| High cut | 10 Hz |
| Pulse | 11/sec |
| Pulse Width | 0.1 msec |
| Notch | ON |
| Decibels | 60 Db |
| Recordings | 100 average was recorded using click sound as a stimuli |

The contra lateral ear was masked with white noise of 50-60 dB.

**The wave peaks of BAER namely**,

Wave 1 (first positive wave followed by stimulus artefact),

Wave V (positive wave at approximately at 6 ms),

Wave III ((major peak  wave between wave I and wave V) and

Pa (positive wave at 25-45 ms) were recorded (10).

The amplitude of wave I and V was also noted. From them, inter peak latencies I-III,I-V and III-V are also calculated.

**BAEP waves generators:**

Wave I from Cochlear Nerve

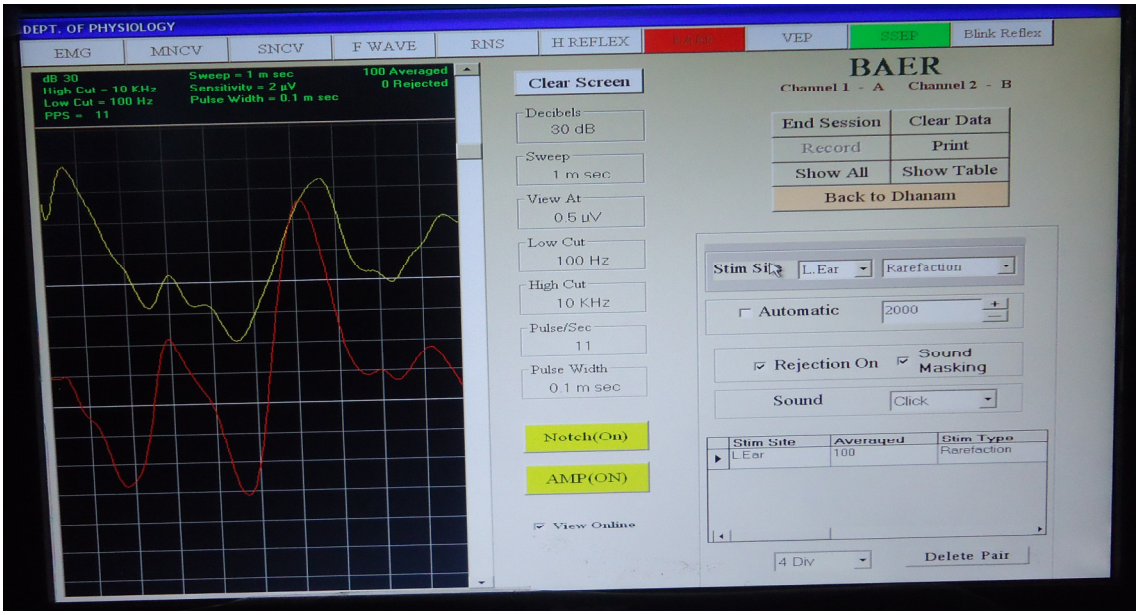
Wave II from Cochlear Nuclei

Wave III from Superior olivary nucleus,

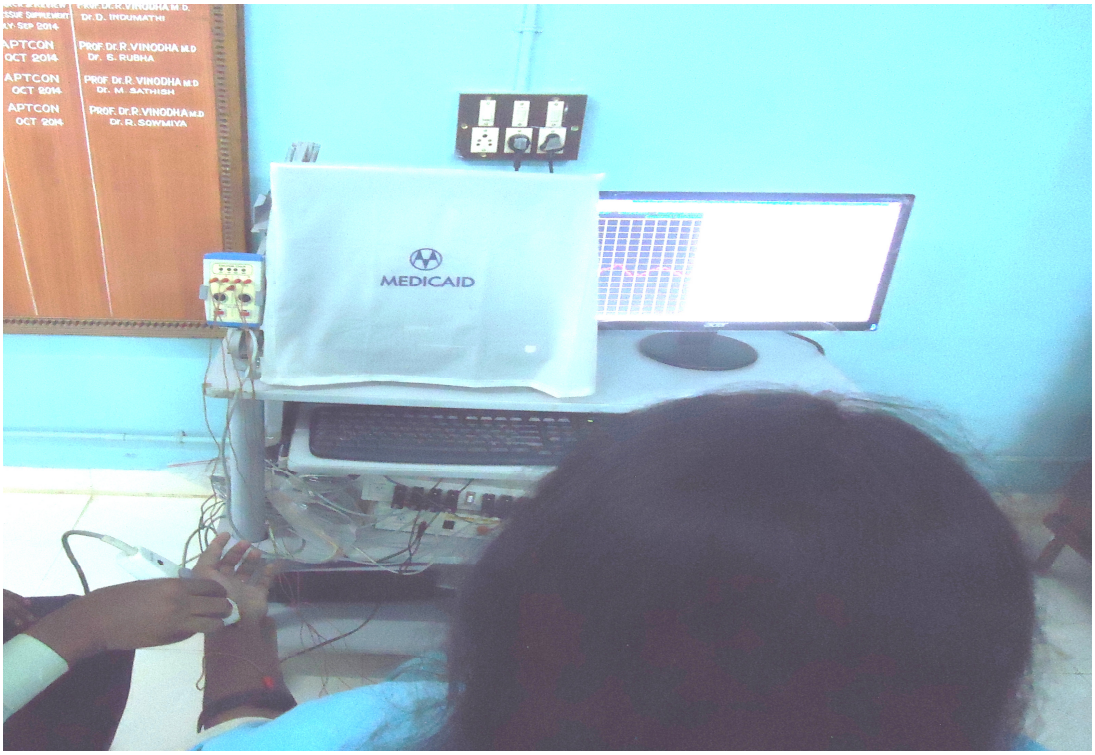
Wave IV from Lateral lemniscus and

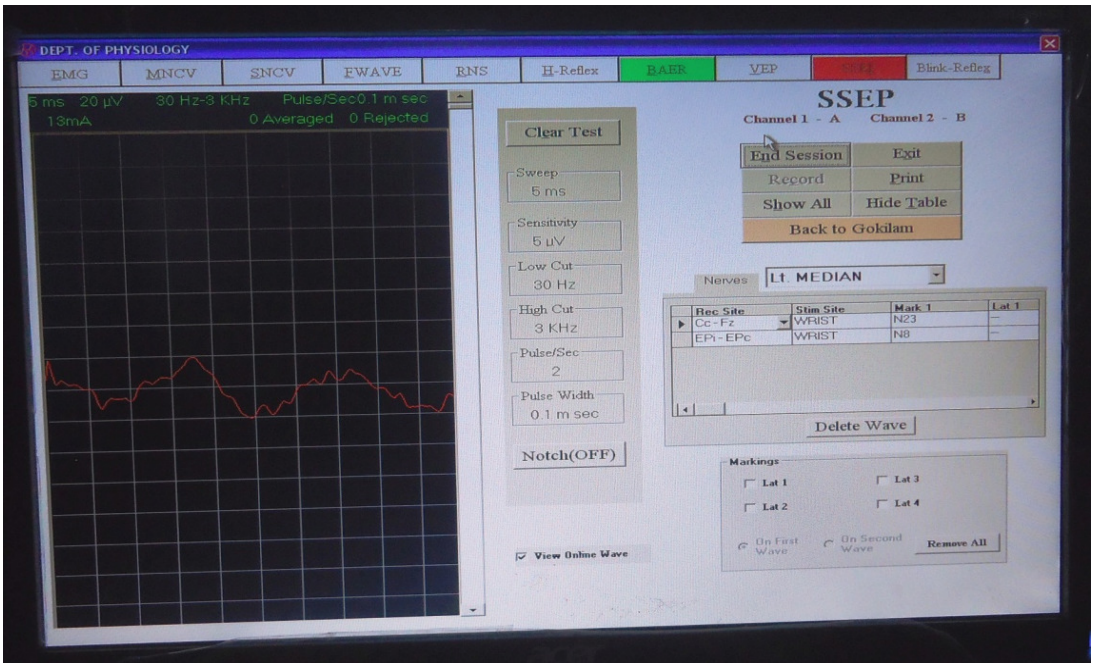
Wave V from Inferior colliculus

## RECORDING OF BRAINSTEM AUDITORY EVOKED POTENTIAL:

**Recording of somatosensory evoked response:**



History obtained to rule out nerve injury and Median nerve examination carried out.

Patient was seated comfortably on a chair.

Instructed to gently close eyes to relax all the head and neck muscles.

The room should be calm and comfortable.

The skin is prepared by abrading.

SSEP parameters are recorded using two channel digital polygraph Electrodes of the 2 channels were placed at appropriate sites after proper abrasion.

Placement of electrodes & recording of potentials were done in accordance with 10-20 electrode placement system.[61]

**RECORDING OF SSEP**: **Instrumental setting for SSEP**:

| | |
|---|---|
| Settings | SSEP |
| Sweep | 5 msec |
| Sensitivity | 10μV |
| Low cut | 30 Hz |
| High cut | 3 Hz |
| Pulse | 21/sec |
| Pulse Width | 0.1 msec |
| Notch | OF |
| Decibels | 60 Db |
| Recordings | 100 average was recorded using click sound as a stimuli |

Recordings 100 average was recorded using 5mAmp Current.

Parameters are recorded using two channel digital polygraph

Channel I :Cc-Fz to record N20

Channel II : Epi- Epc to record N9

From the waveform, the following are measured

N9 latency is measured in EPi-EPc channel from stimulus artefact to its peak

N20 latency is measured 1n Cc-Fz channel

N9 —Distal brachial plexus

N20 -thalamocortical radiation Cortical latency - N20

## Statistical analysis of data

Statistical analysis was done using SPSS version 18.0. The results of BAER and SSEP were analysed using Student's independent T test. P value of <0.05 was considered to be statistically significant.

# RESULTS

Out of the 80 subjects,40 were congenital blind forming study group and 40 were normal subjects forming control group.

In this congenital blind who form the study group were in the pre menstrual age group with mean 28.65±6.036 and the subjects in the control group were in the pre menstrual age group with mean 27.95±6.047

These mean values and standard deviation for study group and control group were tabulated.

These two groups differ significantly in some electrophysiological parameters like BAER and SSEP.

P- values were derived from data analysis by using statistical package SPSS version 18.Students T test was used to do statistical analysis. The statistical significance was considered significant at P value of 0.05

## DESCRIPTIVE STATISTICS

## Brainstem Auditory Evoked Potential Findings –CONTROL(n=40)

| LATENCY (ms) | SIDE | MINIMUM | MAXIMUM | MEAN | STANDARD DEVIATION |
|---|---|---|---|---|---|
| WAVE I | RIGHT | 1.58 | 1.75 | 1.652 | 0.04109 |
| | LEFT | 1.55 | 1.75 | 1.65775 | 0.03886 |
| WAVE III | RIGHT | 3.55 | 3.750 | 3.64725 | 0.04362 |
| | LEFT | 3.580 | 3.760 | 3.64725 | 0.04169 |
| WAVE V | RIGHT | 5.400 | 5,620 | 5.52825 | 0.05325 |
| | LEFT | 5.250 | 5.720 | 5.5655 | 0.08840 |
| WAVE I-III | RIGHT | 1.830 | 2.170 | 1.99538 | 0.06311 |
| | LEFT | 1.870 | 2.130 | 1.9895 | 0.05684 |
| WAVE III-V | RIGHT | 1.720 | 2.070 | 1.881 | 0.06864 |
| | LEFT | 1.600 | 2.070 | 1.91825 | 0.09769 |
| WAVE I-V | RIGHT | 3.720 | 4.040 | 3.87589 | 0.06858 |
| | LEFT | 3.590 | 4.030 | 3.90775 | 0.09119 |

**Brainstem Auditory Evoked Potential Findings –BLIND (n=40)**

| LATENCY (ms) | SIDE | MINIMUM | MAXIMUM | MEAN | STANDARD DEVIATION |
|---|---|---|---|---|---|
| WAVE I | RIGHT | 1.550 | 1.720 | 1.6475 | 0.03484 |
| | LEFT | 1.580 | 1.700 | 1.65025 | 0.03408 |
| WAVE III | RIGHT | 3.55 | 3.75 | 3.6425 | 0.04516 |
| | LEFT | 3.520 | 3.700 | 3.6365 | 0.03739 |
| WAVE V | RIGHT | 5.380 | 5.620 | 5.5015 | 0.05985 |
| | LEFT | 5.420 | 5.680 | 5.52025 | 0.07631 |
| WAVE I-III | RIGHT | 1.860 | 2.110 | 1.995 | 0.06489 |
| | LEFT | 1.840 | 2.120 | 1.98625 | 0.05471 |
| WAVE III-V | RIGHT | 1.700 | 2.00 | 1.859 | 0.06766 |
| | LEFT | 1.77 | 2.100 | 1.88375 | 0.06766 |
| WAVE I-V | RIGHT | 3.730 | 3.970 | 3.854 | 0.06126 |
| | LEFT | 3.72 | 4.060 | 3.87 | 0.09204 |

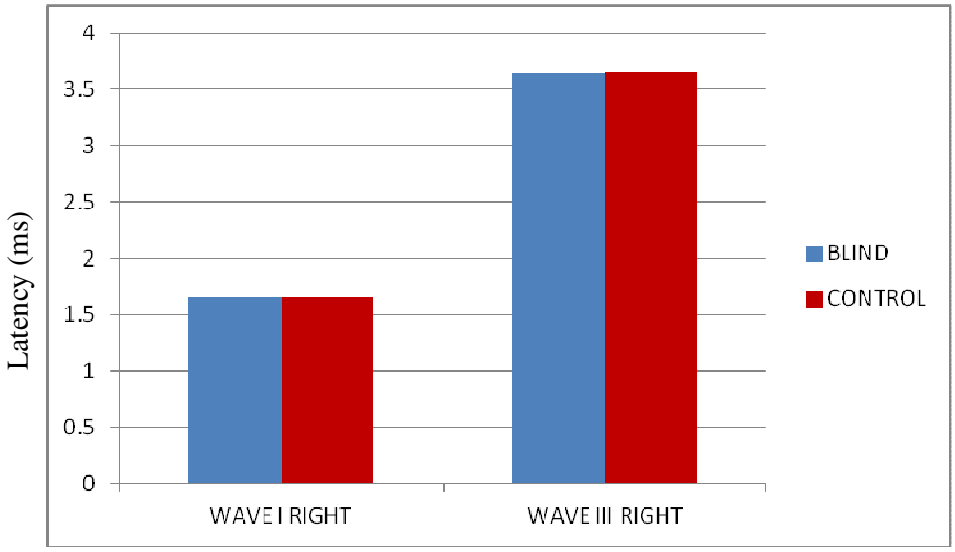
**Comparison Of Brainstem Auditory Evoked Potential Findings Between Control And Blind:**

| LATENCY(ms) | SIDE | CONTROL Mean±SD | BLIND Mean±SD | P-VALUE |
|---|---|---|---|---|
| WAVE- I | RIGHT | 1.652± 0.04109 | 1.6475±0.03484 | 0.5988 |
| | LEFT | 1.65775± 0.03886 | 1.65025±0.03408 | 0.3616 |
| WAVE-III | RIGHT | 3.64725± 0.04362 | 3.6425±0.04516 | 0.6377 |
| | LEFT | 3.64725± 0.04169 | 3.6365±0.03739 | 0.2284 |
| WAVE-V | RIGHT | 5.52825± 0.05325 | 5.5015±0.05985 | 0.0379 |
| | LEFT | 5.5655± 0.08840 | 5.52025±0.07631 | 0.0165 |
| WAVE I-III | RIGHT | 1.99538± 0.06311 | 1.995±0.06489 | 0.9788 |
| | LEFT | 1.9895± 0.05684 | 1.9862±0.05471 | 0.7951 |
| WAVE III-V | RIGHT | 1.881± 0.06864 | 1.859±0.06766 | 0.1529 |
| | LEFT | 1.91825± 0.09769 | 1.88375±0.06766 | 0.0982 |
| WAVE I-V | RIGHT | 3.87589± 0.06858 | 3.854±0.06126 | 0.1282 |
| | LEFT | 3.90775± 0.09119 | 3.87±0.09204 | 0.0692 |

The above table shows significant difference between blind and normal sighted control group in peak latency of wave V in Brainstem Auditory Evoked Potential.

## BRAINSTEM AUDITORY EVOKED POTENTIAL-RIGHT SIDE

## COMPARISON OF WAVE I AND WAVE III LATENCIES BETWEEN BLIND AND CONTROL GROUP

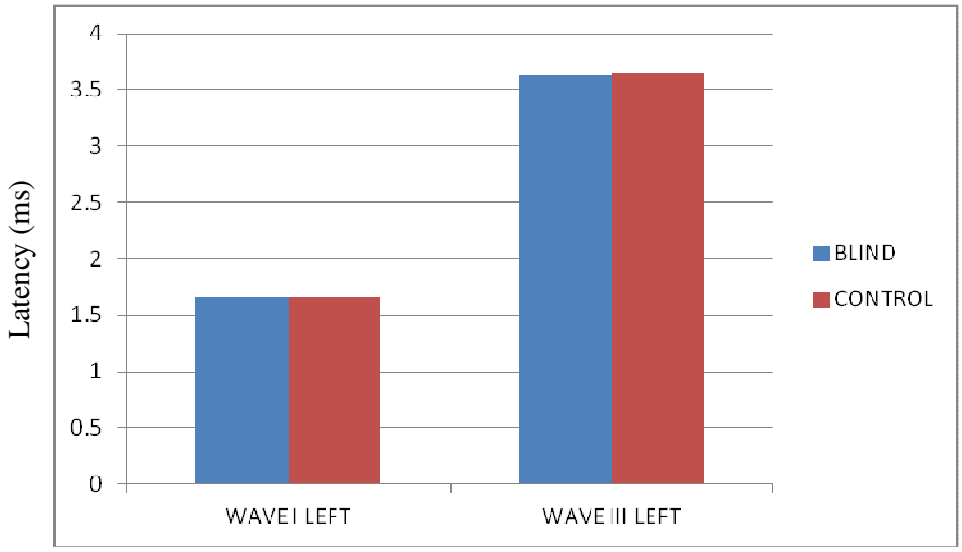



The above figure shows that

The mean value of wave I latency of Right in blind was 1.6475 ± 0.03484 and the mean value for control group was 1.652 ± 0.04109 in with P-value as 0.5988 which was found to be non –significant

The mean value of wave III latency Right side in blind was 3.6425±0.04516 and the mean value for control group was 3.64725±0.04362 in with P-value as 0.6377which was found to be non -significant

**BRAINSTEM AUDITORY EVOKED POTENTIAL-LEFT SIDECOMPARISON OF WAVE I AND WAVE III LATENCIES BETWEEN BLIND AND CONTROL GROUP**




The above figure shows that

The mean value of wave I latency of left side in blind was1.65025±0.03408

and the mean value for control group was1.65775±0.03886 with P-value of 0.3616 which was found to be non –significant

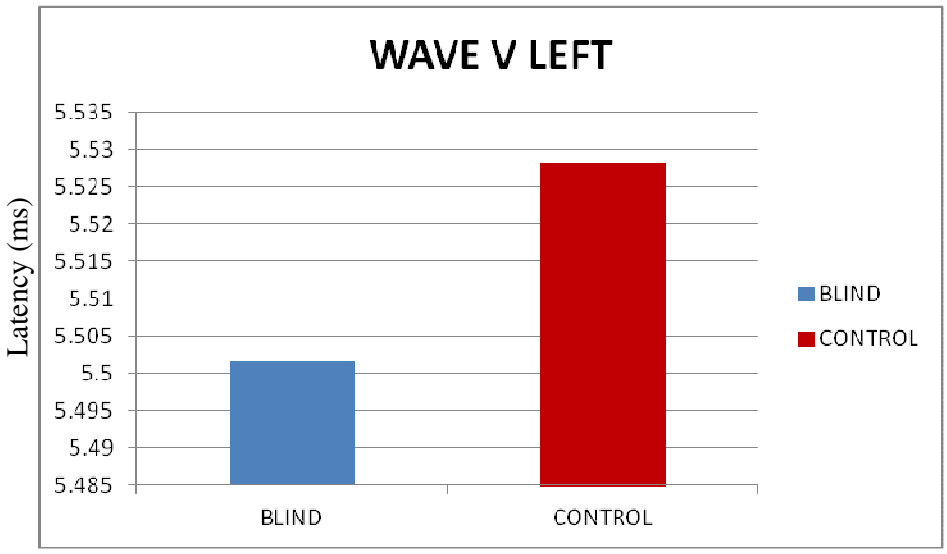
The mean value of wave III latency of left side in blind was 3.6365±0.03739

and the mean value for control group was 3.64725±0.04169 with P-value of 0.2284which was found to be non –significant

**BRAINSTEM AUDITORY EVOKED POTENTIAL-RIGHT SIDE**

**COMPARISON OF wave V LATENCY OF LEFT EAR**

**BETWEEN BLIND AND CONTROL GROUP**



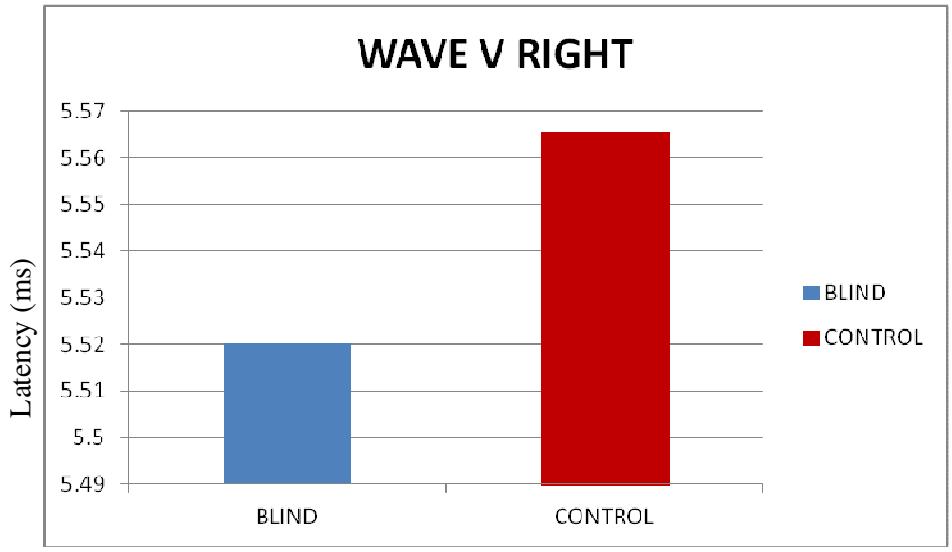

The above figure shows that

The mean value of wave V latency of left side in blind was5.52025±0.07631 and for control group the mean value was 5.5655±0.08840 with P-value of 0.0165 which was found to be significant

**BRAINSTEM AUDITORY EVOKED POTENTIAL-RIGHT SIDE**

**COMPARISON OF wave V LATENCY OF RIGHT EAR**

**BETWEEN BLIND AND CONTROL GROUP**

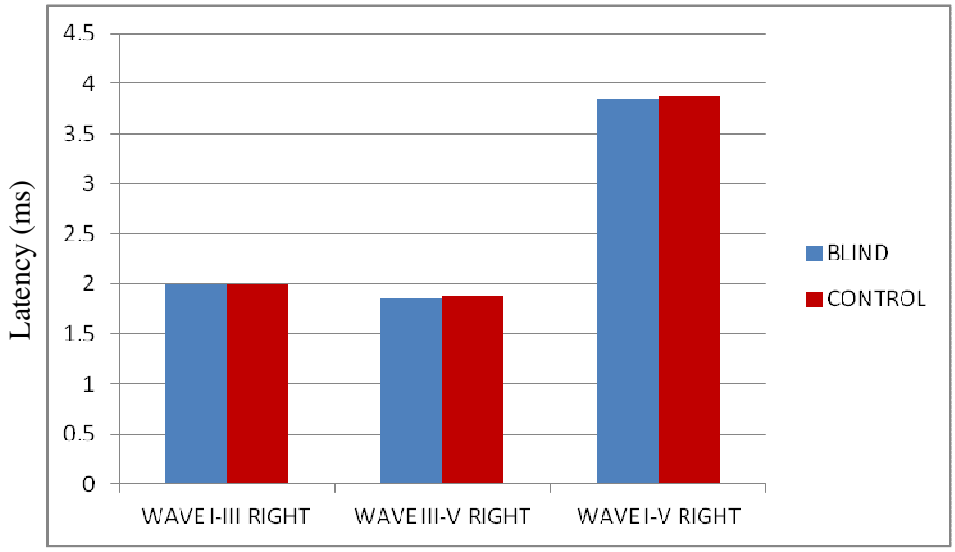



The above figure shows that

The mean value of wave V latency of right side in blind was 5.5015±0.05985 and for control group the mean value was 5.52825±0.05325 with P-value of 0.0379 which was found to be significant

**BRAINSTEM AUDITORY EVOKED POTENTIAL-RIGHT SIDE**

**COMPARISON OF INTER PEAK LATENCIES BETWEEN BLIND AND CONTROL GROUP**




The above figure shows that

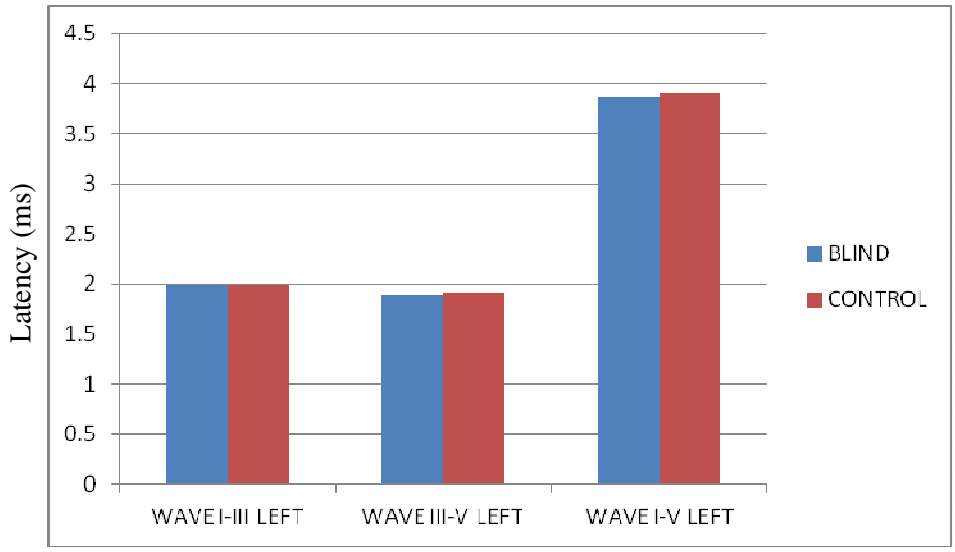
The mean value of inter peak latency of wave I-III on Right side in blind was 1.995±0.06489 and the mean value as 1.99538±0.06311 in control group with P-value of 0.9788 which was found to be non –significant

The mean value of inter peak latency of wave III-V on Right side in blind was 1.859±0.06766 and the mean value as 1.881±0.06864 in control group with P-value of 0.1529 which was found to be non -significant

The mean value of inter peak latency of wave I-V on Right side in blind was 3.854±0.06126 and the mean value as3.87589±0.06858 in control group with P-value of 0.1282 which was found to be non -significant

**BRAINSTEM AUDITORY EVOKED POTENTIAL-LEFT SIDE**

**COMPARISON OF INTER PEAK LATENCIES BETWEEN BLIND AND CONTROL GROUP**




The above figure shows that

The mean value of inter peak latency of wave I-III on Left side in blind was1.9862±0.05471 and the mean value for control group was 1.9895± 0.05684 with P-value of 0.7951 which was found to be non –significant

The mean value of inter peak latency of wave III-V on Left side in blind was 1.88375±0.06766 and the mean value for control group was 1.91825±0.09769 with P-value of 0.0982which was found to be non - significant

The mean value of inter peak latency of wave I-V on Left side in blind was 3.87±0.09204 and the mean value for control group as 3.90775±0.092119 with P-value of 0.0692 which was found to be non -significant

**Somatosensory Evoked Potential Findings –CONTROL (n=40)**

| LATENCY (ms) | SIDE | MINIMUM | MAXIMUM | MEAN | Standard Deviation |
|---|---|---|---|---|---|
| N 9 | Right | 8.280 | 9.480 | 9.04 | 0.3164 |
| | Left | 8.120 | 9.450 | 8.835 | 0.3979 |
| N22 | Right | 17.88 | 18.50 | 18.20325 | 0.1660 |
| | Left | 17.82 | 18.55 | 18.17925 | 0.2731 |
| N9-N22 | Right | 8.470 | 10.130 | 9.16325 | 0.3860 |
| | Left | 8.630 | 10.300 | 9.34425 | 0.4483 |

**Somatosensory Evoked Potential Findings in BLIND (n=40)**

| LATENCY (ms) | SIDE | MINIMUM | MAXIMUM | MEAN | Standard Deviation |
|---|---|---|---|---|---|
| N 9 | Right | 8.250 | 9.520 | 9.00025 | 0.3918 |
| | Left | 8.150 | 9.380 | 8.74575 | 0.3647 |
| N22 | Right | 17.820 | 18.820 | 18.09225 | 0.1809 |
| | Left | 17.350 | 17.250 | 17.957 | 0.2510 |
| N9-N22 | Right | 8.370 | 9.940 | 9.092 | 0.4486 |
| | Left | 8.460 | 10.00 | 9.21125 | 0.4155 |

**Comparison of Somatosensory Evoked Potential Findings between Blind and Control groups:**

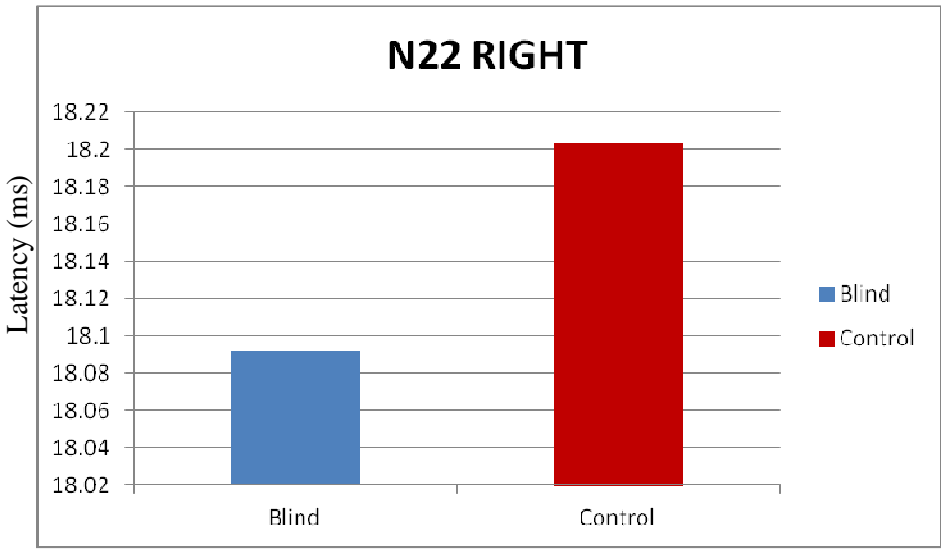
| LATENCY (ms) | SIDE | CONTROL Mean ± SD | BLIND Mean ± SD | P-VALUE |
|---|---|---|---|---|
| N 9 | RIGHT | 9.04± 0.3164 | 9.00025± 0.3918 | 0.6190 |
| | LEFT | 8.835± 0.3979 | 8.74575± 0.3647 | 0.2989 |
| N 22 | RIGHT | 18.20325± 0.1660 | 18.09225± 0.1809 | 0.0054 |
| | LEFT | 18.17925± 0.2731 | 17.957± 0.2510 | 0.0003 |
| N9 - N22 | RIGHT | 9.16325± 0.3860 | 9.092± 0.4486 | 0.4487 |
| | LEFT | 9.34425± 0.4483 | 9.21125± 0.4155 | 0.1727 |

The above table shows significant difference between blind and normal sighted group in latency of N22 on both sides of median nerve Somatosensory Evoked Potential

**SOMATOSENSORY EVOKED POTENTIAL -RIGHT SIDE**

**COMPARISON OF WAVE N 22 LATENCY**

**BETWEEN BLIND AND CONTROL GROUP**



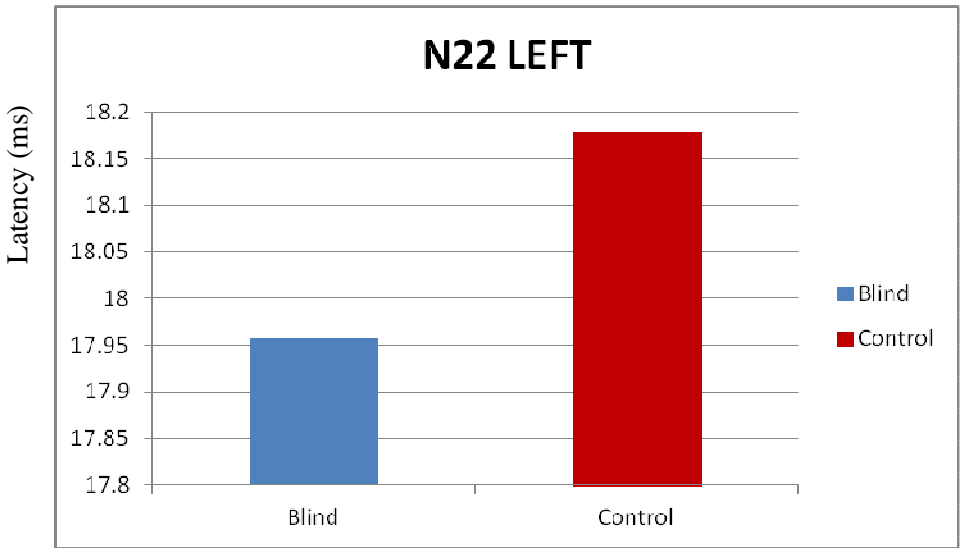

The above figure shows that

The mean value of wave N22 latency of Right side in blind was 18.09225±0.1809 and the mean value for control group was 18.20325±0.1660 with P-value of 0.0054 which was found to be non –significant

**SOMATOSENSORY EVOKED POTENTIAL -LEFT SIDE**

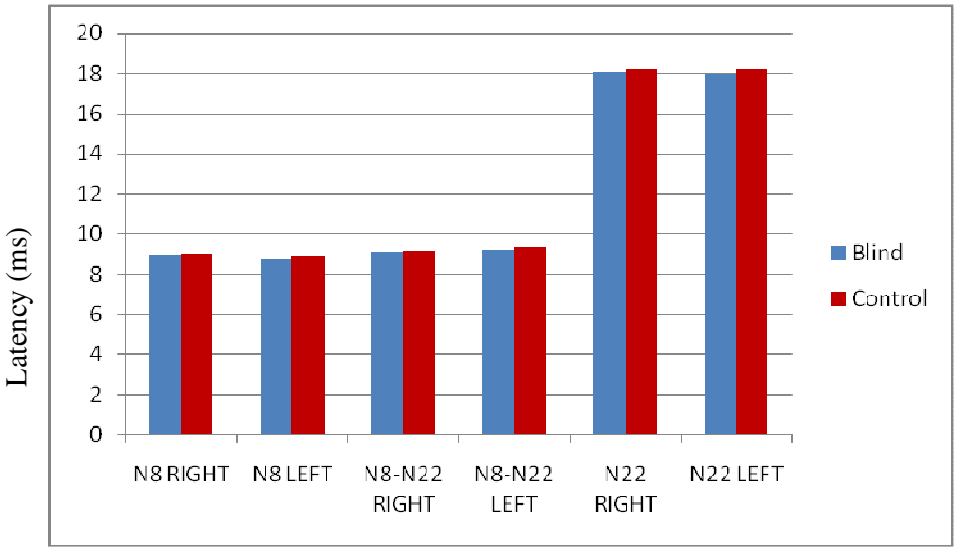
**COMPARISON OF WAVE N 22 LATENCY OF**

**BETWEEN BLIND AND CONTROL GROUP**




The mean value of wave N22 latency of Left side in blind was17.957±0.2510 and the mean value for control group was 18.17925± 0.2731 with P-value of 0.003 which was found to be non –significant

**SOMATOSENSORY EVOKED POTENTIAL COMPARISON OF LATENCY AND INTER PEAK LATENCY BETWEEN BLIND AND CONTROL GROUP**




The above figure shows that

The mean value of latency of wave N9 on Right side in blind was9.00025±0.3918 and the mean value control group for was9.04±0.3164 of same side with P-value of 0.6190 which was found to be non –significant.

The mean value of latency of wave N9- N22 on Left side in blind was8.74575±0.3647 and the mean value for control group was 8.835±0.3979 of same side with P-value as 0.2989 which was found to be non –significant

The mean value of inter peak latency of wave N9- N22 on Right side in blind was9.092±0.4486 and the mean value for control group was9.16325±0.3860 of same side with P-value as 0.4487 which was found to be non –significant.

The mean value of inter peak latency of wave N8- N22 on Left side in blind was 9.21125±0.4155 and the mean value for control group was 9.34425±0.4483 of same side with P-value as 0.1727 which was found to be non –significant.

# DISCUSSION

In this study, electrophysiological parameters were evaluated in 40 congenital total blind females. The results of electrophysiological parameters were compared with 40 age and sex matched normal sighted individuals with mean age of 28.65±6.036 for blind and mean age of 27.95±6.047 for control.

This study compares the absolute latencies and inter peak latencies of Somatosensory Evoked Potential and Brainstem Auditory Evoked Response in normal and congenitally blind individuals.

Though there are lot of information regarding cross modal plasticity in blind people, the aim of the present study is to prove the plasticity probably cross modal at the lower level of auditory system and along the tract of somatosensory system followed by visual deprivation.

Neuroplasticity is the capacity of the brain to restructure its neural pathways. It can be a normal process of learning and memory or it may be a compensatory mechanism for the lost sensory function which is called as Cross-modal plasticity. This neuroplasticity changes generally involve neurons at the cortical level and its cortico-cortical connections. It may involve the sub-cortical centres and also their connecting tracts [(75)].

Synapses are dynamic structures. It may increase or decrease in their number and complexity with use. The quantity of blood flow and molecular expression essential for the development of cortex depends on the particular neuron activity. The reason for the cortical representation occurring in blind appears to be due to cortical connections of sensory units to the cortex. These connections have extensive convergence and divergence that may become weak with disuse and strong with repeated use[76].

Thomas Elbert et al in their study showed expansion of the tonotopic area in the auditory cortex of the blind by showing decrease in the latency of the N1m component of the auditory-evoked magnetic response between blind and normal sighted due to the lack of visual input in association with enhanced auditory activity [77]

N. Lessard et al showed better localisation of sound in early blind by compensatory mechanism depending on the aetiology and extent of blindness(78)

Brigitte Röder et al in his electrophysiological study showed improved improved auditory spatial tuning in blind individuals by compensatory reorganisation of brain areas[79] .

Ptito M et al in their study using PET scan ,demonstrated the cross-modal plasticity in the visually impired individuals by electro-tactile stimulation of tongue and showed that occipital area forms a part of neural network in discriminating of touch sensation along with posterior parietal

cortex because of increased regional blood flow in occipital cortex and this was due to the performance of a task.[56]

Norihiro Sadato et al in their functional magnetic resonance imaging study between congenital and early-onset blind subjects revealed that there was an activation of the primary sensory area in association with polymodal association areas in congenital blind as compared with early onset blind individuals[80].

This study included only congenitally blind subjects with mean age of 28.65±6.6036 years and also found that the cross modal plasticity changes involving the auditory system and somatosensory system of all congenital blind subjects. This was evident by shortening of wave V latency in Brainstem auditory evoked response and shortening of wave N22 latency in somatosensory evoked potential of median nerve.

This was in consistent with the studies of Norihiro Sadato et al [80] Kujala T et al.[81],animal research by Volgyi et al.[82] Grunewald et al.[83] and Diana M. Kahn & Leah Krubitzer [84] who in their studies they had shown that cross modal plasticity changes were more in early blind.

So , the development of cortical reorganization may occur as the consequence of the lack of visual input in association with enhanced auditory activity, generated by long-term concentration of blind individuals on non visual cues to interact approximately with the environment.

**BAER Waveforms of Study And Control Groups:**

In my study, peak latencies of wave V of Brainstem Auditory Evoked Response were significantly lower with P- value of 0.004 in right ear and P-value 0.02 in left ear in blind subjects when compared to normal sighted.

Though, several studies [65,66,67,68,69] demonstrated the compensatory plastic changes in auditory system at cortical and sub cortical level ,the aim of our study was to know the plasticity changes at the low tier of auditory pathway in untrained congenital blind individuals. In our study this was proven by shortening of wave V latency of Brainstem Auditory Evoked Response on both sides of blind individuals.

This is similar to the study of B.Anbarasi et al [64] who showed in their study shortening of latency of wave V of Brainstem auditory evoked potential due to enhanced peripheral activity.

So, from our study, we could consider inferior colliculus , the major generator of wave V [61] as an acousticomotor nucleus with widespread connections with the spinal cord. the cerebellum, the vocalisation and somatosensory systems and also, our findings suggest that inferior colliculus is one among the important site of auditory processing and also an important site for the occurrence of neuronal and synaptic plasticity. We could also say that the cross modal plasticity in auditory system as shown by the shorter absolute latency of wave V in blind subjects when compared to normal sighted persons starts at the level of Inferior colliculus.

This was also supported by studies in anophthalmic mice [85] which showed that sub-cortical rerouting of connections leading to somatic and auditory activation of visual cortex .It also showed that Inferior colliculus and cuneate nucleus giving neural connection to lateral geniculate nucleus and influence the signal processing of cochlea.

HR Nagendra and K.V.Naveen et al [66] and showed that the wave V of BERA was not statistically significant and also wave Na of the Mid Latency Response, indicating that the brainstem and diencephalic areas which are known to generate these Waves appeared to be unchanged in the blind.

Neimeyer and Starling[85] also showed that peak latencies of Ni wave of Long Latency Response was lower in the congenitally blind without any change in lower level.

By these studies[66,85], we can suggest that they had tested the cortical reorganisation occurring in areas above the level of cortical relay.

Hence, the cross-modal plasticity found at the lower level of the auditory tract in our study may be due to increased dependence of auditory system by the blind subjects starting early in their lives to interact with the environment. Thus it favours the cross modal plasticity observed with increased dependence on one system.

## SSEP waveforms in study and control group:

Somatosensory evoked potentials are generated in areas of signal processing which corresponds to the synaptic junctions of numerous neurons. They are path-specific electrical signals which give good temporal resolution in milliseconds domain.

In our study, N22 Latency on both sides were significantly decreased in the congenitally blind when compared to control group with P-value of 0.0054 on Right side and P-value of 0.003 on Left side.

Dayananda G.et al [(70)]in his study on comparative median nerve somatosensory evoked potential between Braille reading total blind and normal subjects showed larger N20 amplitude and normal latency of N20 due to greater somatosensory cortical representation than normal sighted individuals.Similarly in his another study[(72)] ,when comparing the index finger somatosensory evoked potential in Braille reading total blind individuals and normal subjects showed larger N20 amplitude and normal latency of N20 due to greater somatosensory cortical representation than normal sighted individuals..

Elbert et al[(73)] in his study comparing the congenital blind and normal sighted individuals, showed a decrease in cortical latency while performing a task due to quick processing during the discrimination tasks of the event-related potentials.

Nikhat et al[74] in his study comparing the congenital blind and normal sighted individuals, demonstrated the shortening of wave N 20 latency and increased amplitude of wave N20 in somatosensory evoked potential due to improved information in the nervous system and the extent of neural network and its synchronization that are involved in processing of vision.

In all of the above studies[70,72,73,74] they had conducted their study in Braille reading blind individuals. But in our study we had studied the median nerve somatosensory evoked potential in non trained total blind, with the result showing decrease in latency of wave N22 probably due to quick processing of information at cortical level.

So, the decrease in N22 latencies of somatosensory evoked potentials of congenitally blind individuals may be due to the improved processing of information at the cortical level in the nervous system. It may also be due to change in the local connectivity by many local mechanisms like sprouting or un-masking of many silent synapses and also the change in modulatory effect of lateral connections

# CONCLUSION

The result of the present study shows that there is an evidence of Cross modal plasticity in blind persons involving their other sensory system like somatosensory system and auditory system.

The blind patients show shortening of wave V latency in Brainstem Auditory Evoked Potential study. Somatosensory Evoked Potential Study of median nerve also reveals shortening of N20 wave latency and augmentation of N20 amplitude.

These observations may be due to unmasking of neural circuit or may be due sprouting out of new neural connections at the level of cortex in somatosensory system and at the level of inferior colliculus in auditory system

This study suggests that earlier the onset of deficit more will be the development of plasticity changes. However further imaging studies are required to confirm these findings.

By these studies we can plan rehabilitation measures for the blind to improve the quality of their life.

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# PROFORMA

## C0MPARATIVE STUDY OF BRAINSTEM AUDITORY EVOKED POTENTIAL AND SOMATOSENSORY EVOKED POTENTIAL IN TOTAL BLIND AND NORMAL SUBJECTS

**Study Group/ Control Group**

**Name :** **Age:** **Sex:**

Address: Occupation:

H/O Present illness: H/o Giddiness

Past History: Known case DM/SHT/Pulmonary TB/Asthma

Personal History: Smoking/Alcohol/Betalnut chewing with or without tobacco

Family History:

**General Examination:**

Height: Weight:
Anaemic/Not anaemic

Cyanosis/No cyanosis

Clubbing/ No Clubbing

Jaundice/ No Jaundice

Pedal Edema/ No Pedal Edema

Generalised lymphadenopathy present/absent

**Vital Signs**: **PR:** **BP:** **RR:**

Examination of CVS:

Examination of RS:

Examination of Abdomen:

Examination of **CNS**:

**Somatosensory Evoked Potential:**

| Latency | Right Wrist | Left Wrist |
|---|---|---|
| N9 | | |
| N20 | | |
| N9-N20 | | |

**Brainstem Auditary Evoked potential :**

| Peak Latency | I | III | V |
|---|---|---|---|
| | | | |
| Interpeak Latency | I-III | III-V | I-V |
| | | | |

## ABBREVIATIONS USED IN THE STUDY:

| | | |
|---|---|---|
| **V 1** | **-** | **Primary visual cortex** |
| **A-1** | **-** | **Primary auditory cortex** |
| **MRI** | **-** | **Magnetic resonance imaging** |
| **PET** | **-** | **Positron emission tomography** |
| **EP** | **-** | **Evoked potential** |
| **BAEP** | **-** | **Braistem auditory evoked potential** |
| **SSEP** | **-** | **Somatosensory evoked potential** |
| **CMP** | **-** | **Cross modal plasticity** |
| **MGN** | **-** | **Medial geniculate nucleus** |
| **LGN** | **-** | **Lateral geniculate nucleus** |
| **IC** | **-** | **Inferior colliculus** |
| **EEG** | **-** | **Electroencephalography** |
| **FMRI** | **-** | **Functional magnetic resonance imaging** |

# INFORMED CONSENT

I Dr.S.Chandrabalan student in department of physiology, Thanjavur Medical College, Thanjavur is doing the the entitled Comparative study of Brainstem Auditory Evoked Potential and Somatosensory evoked potential in total blind and normal subjects.

I understand the procedure and voluntarily agree to participate in this study ,I also understand that this study is a noninvasive procedure and the possible adverse effects have been explained to me in details clearly in my own language

Signature of the subject

**SOMATO SENSORY EVOKED POTENTIAL**

**STUDY GROUP**

| | | LEFT SIDE | | | | RIGHT SIDE | | |
|---|---|---|---|---|---|---|---|---|
| | | LATENCY | LATENCY | IPL | | LATENCY | LATENCY | IPL |
| Sl.No. | AGE | N8LS | N22LS | N8-N22LS | | N8RS | N22RS | N8-N22RS |
| 1 | 24 | 8.62 | 17.88 | 9.26 | | 8.52 | 18.22 | 9.7 |
| 2 | 32 | 9.32 | 18.12 | 8.8 | | 9.35 | 18.15 | 8.8 |
| 3 | 36 | 8.52 | 17.65 | 9.13 | | 9.22 | 18.2 | 8.98 |
| 4 | 18 | 8.65 | 18.12 | 9.47 | | 8.62 | 18.12 | 9.5 |
| 5 | 28 | 8.25 | 17.62 | 9.37 | | 9.48 | 18.2 | 8.72 |
| 6 | 38 | 8.62 | 18.22 | 9.6 | | 8.82 | 18.15 | 9.33 |
| 7 | 22 | 9.32 | 18.22 | 8.9 | | 9.45 | 17.98 | 8.53 |
| 8 | 28 | 8.86 | 18.12 | 9.26 | | 8.52 | 18.15 | 9.63 |
| 9 | 18 | 9.32 | 17.78 | 8.46 | | 9.35 | 17.85 | 8.5 |
| 10 | 21 | 9.25 | 18.22 | 8.97 | | 8.65 | 18.12 | 9.47 |
| 11 | 36 | 8.42 | 17.82 | 9.4 | | 9.13 | 17.85 | 8.72 |
| 12 | 25 | 8.22 | 18.22 | 10 | | 9.25 | 18.25 | 9 |
| 13 | 31 | 9.25 | 18.2 | 8.95 | | 9.42 | 17.82 | 8.4 |
| 14 | 37 | 9.12 | 17.78 | 8.66 | | 9.32 | 18.15 | 8.83 |
| 15 | 29 | 8.15 | 17.88 | 9.73 | | 8.72 | 17.92 | 9.2 |
| 16 | 23 | 8.26 | 18.12 | 9.86 | | 8.82 | 18.18 | 9.36 |
| 17 | 22 | 8.72 | 18.25 | 9.53 | | 8.82 | 18.15 | 9.33 |
| 18 | 29 | 8.52 | 17.82 | 9.3 | | 9.42 | 17.85 | 8.43 |
| 19 | 20 | 8.55 | 18.12 | 9.57 | | 8.62 | 18.18 | 9.56 |
| 20 | 37 | 9.32 | 18.12 | 8.8 | | 9.25 | 17.85 | 8.6 |
| 21 | 31 | 8.65 | 17.75 | 9.1 | | 9.52 | 18.22 | 8.7 |
| 22 | 33 | 9.35 | 18.12 | 8.77 | | 8.65 | 17.98 | 9.33 |
| 23 | 24 | 8.15 | 17.92 | 9.77 | | 8.25 | 17.92 | 9.67 |
| 24 | 29 | 8.24 | 18.2 | 9.96 | | 9.32 | 18.82 | 9.5 |
| 25 | 25 | 8.82 | 17.85 | 9.03 | | 8.82 | 18.18 | 9.36 |
| 26 | 33 | 8.65 | 18.12 | 9.47 | | 9.22 | 17.95 | 8.73 |
| 27 | 23 | 9.38 | 17.85 | 8.47 | | 8.62 | 17.98 | 9.36 |
| 28 | 25 | 8.76 | 17.82 | 9.06 | | 9.42 | 18.18 | 8.76 |
| 29 | 35 | 8.45 | 18.15 | 9.7 | | 8.62 | 17.98 | 9.36 |
| 30 | 33 | 8.35 | 17.72 | 9.37 | | 9.35 | 18.12 | 8.77 |
| 31 | 27 | 8.86 | 18 | 9.14 | | 8.68 | 18.22 | 9.54 |
| 32 | 29 | 8.52 | 17.52 | 9 | | 9.45 | 17.82 | 8.37 |
| 33 | 35 | 8.65 | 18.22 | 9.57 | | 8.92 | 18.22 | 9.3 |
| 34 | 27 | 8.72 | 17.35 | 8.63 | | 9.48 | 17.95 | 8.47 |
| 35 | 39 | 8.88 | 18.22 | 9.34 | | 8.28 | 18.22 | 9.94 |
| 36 | 21 | 8.92 | 17.45 | 8.53 | | 9.22 | 18.15 | 8.93 |
| 37 | 23 | 8.68 | 18.22 | 9.54 | | 8.95 | 18.12 | 9.17 |
| 38 | 36 | 8.82 | 17.85 | 9.03 | | 9.45 | 17.98 | 8.53 |
| 39 | 37 | 8.86 | 18.15 | 9.29 | | 8.76 | 18.22 | 9.46 |
| 40 | 27 | 8.86 | 17.52 | 8.66 | | 8.28 | 18.12 | 9.84 |

**SOMATO SENSORY EVOKED POTENTIAL**

**CONTROL GROUP**

| | | LEFT SIDE | | | | RIGHT SIDE | | |
|---|---|---|---|---|---|---|---|---|
| | | LATENCY | LATENCY | IPL | | LATENCY | LATENCY | IPL |
| Sl.No | AGE | N8LC | N22LC | N8-N22LC | | N8RC | N22RC | N8-N22RC |
| 1 | 18 | 8.25 | 17.82 | 9.57 | | 9.12 | 18.2 | 9.08 |
| 2 | 18 | 9.2 | 18.12 | 8.92 | | 9.32 | 18.22 | 8.9 |
| 3 | 36 | 8.48 | 17.82 | 9.34 | | 9.42 | 18.12 | 8.7 |
| 4 | 28 | 8.52 | 17.85 | 9.33 | | 8.68 | 17.92 | 9.24 |
| 5 | 24 | 8.35 | 18.22 | 9.87 | | 9.18 | 18.21 | 9.03 |
| 6 | 36 | 9.22 | 17.85 | 8.63 | | 9.12 | 18.25 | 9.13 |
| 7 | 32 | 9.2 | 18.2 | 9 | | 8.82 | 18.12 | 9.3 |
| 8 | 28 | 8.12 | 17.85 | 9.73 | | 9.12 | 18.22 | 9.1 |
| 9 | 19 | 9.12 | 18.48 | 9.36 | | 8.92 | 18.12 | 9.2 |
| 10 | 27 | 8.22 | 18.12 | 9.9 | | 9.48 | 17.95 | 8.47 |
| 11 | 35 | 9.12 | 17.88 | 8.76 | | 8.86 | 18.15 | 9.29 |
| 12 | 22 | 8.62 | 18.22 | 9.6 | | 9.12 | 18.22 | 9.1 |
| 13 | 19 | 9.32 | 18.15 | 8.83 | | 8.75 | 18.15 | 9.4 |
| 14 | 23 | 9.12 | 18.12 | 9 | | 9.38 | 18.21 | 8.83 |
| 15 | 29 | 8.42 | 18.22 | 9.8 | | 8.72 | 18.15 | 9.43 |
| 16 | 35 | 9.15 | 17.85 | 8.7 | | 9.32 | 17.95 | 8.63 |
| 17 | 33 | 9.22 | 18.22 | 9 | | 8.95 | 18.15 | 9.2 |
| 18 | 31 | 8.35 | 18.12 | 9.77 | | 9.12 | 18.12 | 9 |
| 19 | 28 | 9.15 | 17.88 | 8.73 | | 8.72 | 18.2 | 9.48 |
| 20 | 24 | 9.12 | 17.82 | 8.7 | | 9.38 | 17.88 | 8.5 |
| 21 | 23 | 8.52 | 18.22 | 9.7 | | 8.75 | 18.22 | 9.47 |
| 22 | 34 | 8.12 | 18.15 | 10.03 | | 9.35 | 18.18 | 8.83 |
| 23 | 26 | 9.42 | 18.12 | 8.7 | | 8.92 | 18.18 | 9.26 |
| 24 | 39 | 8.45 | 17.9 | 9.45 | | 9.32 | 17.92 | 8.6 |
| 25 | 25 | 8.92 | 18.52 | 9.6 | | 8.65 | 18.18 | 9.53 |
| 26 | 29 | 8.52 | 18.48 | 9.96 | | 9.38 | 18.15 | 8.77 |
| 27 | 18 | 9.15 | 17.85 | 8.7 | | 8.65 | 18.12 | 9.47 |
| 28 | 31 | 8.82 | 17.85 | 9.03 | | 9.35 | 18.22 | 8.87 |
| 29 | 21 | 8.92 | 18.2 | 9.28 | | 8.92 | 18.22 | 9.3 |
| 30 | 24 | 8.25 | 18.55 | 10.3 | | 9.35 | 17.98 | 8.63 |
| 31 | 33 | 9.15 | 18.5 | 9.35 | | 8.82 | 18.18 | 9.36 |
| 32 | 26 | 8.65 | 18.52 | 9.87 | | 9.35 | 18.5 | 9.15 |
| 33 | 32 | 9.22 | 18.55 | 9.33 | | 9.38 | 18.45 | 9.07 |
| 34 | 19 | 9.18 | 18.52 | 9.34 | | 8.35 | 18.48 | 10.13 |
| 35 | 34 | 8.92 | 18.51 | 9.59 | | 9.28 | 18.5 | 9.22 |
| 36 | 27 | 9.45 | 18.52 | 9.07 | | 8.28 | 18.38 | 10.1 |
| 37 | 35 | 8.65 | 17.85 | 9.2 | | 9.15 | 18.38 | 9.23 |
| 38 | 37 | 9.25 | 18.48 | 9.23 | | 8.58 | 18.48 | 9.9 |
| 39 | 28 | 8.62 | 18.55 | 9.93 | | 9.35 | 18.42 | 9.07 |
| 40 | 32 | 8.95 | 18.52 | 9.57 | | 8.92 | 18.48 | 9.56 |

**BRAIN STEM AUDITORY EVOKED POTENTIAL**

| | | | RIGHT SIDE | | | | STUDY | | | LEFT SIDE | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | LATENCY | | INTER | PEAK | LATENCY | | | LATENCY | | INTER | PEAK | LATENCY |
| S.NO | AGE | IR | IIIR | VR | I-IIIR | III-VR | I-VR | | IL | IIIL | VL | I-IIIL | III-VL | I-VL |
| 1 | 24 | 1.65 | 3.62 | 5.38 | 1.97 | 1.76 | 3.73 | | 1.6 | 3.65 | 5.45 | 2.05 | 1.8 | 3.85 |
| 2 | 32 | 1.62 | 3.68 | 5.52 | 2.06 | 1.84 | 3.9 | | 1.68 | 3.68 | 5.49 | 2 | 1.81 | 3.81 |
| 3 | 36 | 1.68 | 3.62 | 5.55 | 1.94 | 1.93 | 3.87 | | 1.64 | 3.65 | 5.42 | 2.01 | 1.77 | 3.78 |
| 4 | 18 | 1.62 | 3.68 | 5.52 | 2.06 | 1.84 | 3.9 | | 1.68 | 3.62 | 5.52 | 1.94 | 1.9 | 3.84 |
| 5 | 28 | 1.55 | 3.66 | 5.48 | 2.11 | 1.82 | 3.93 | | 1.62 | 3.62 | 5.55 | 2 | 1.93 | 3.93 |
| 6 | 38 | 1.68 | 3.65 | 5.52 | 1.97 | 1.87 | 3.84 | | 1.58 | 3.65 | 5.58 | 2.07 | 1.93 | 4 |
| 7 | 22 | 1.65 | 3.62 | 5.46 | 1.97 | 1.84 | 3.81 | | 1.68 | 3.68 | 5.48 | 2 | 1.8 | 3.8 |
| 8 | 28 | 1.68 | 3.62 | 5.48 | 1.94 | 1.86 | 3.8 | | 1.65 | 3.62 | 5.62 | 1.97 | 2 | 3.97 |
| 9 | 18 | 1.62 | 3.55 | 5.42 | 1.93 | 1.87 | 3.8 | | 1.62 | 3.7 | 5.48 | 2.08 | 1.78 | 3.86 |
| 10 | 21 | 1.65 | 3.68 | 5.52 | 2.03 | 1.84 | 3.87 | | 1.65 | 3.62 | 5.62 | 1.97 | 2 | 3.97 |
| 11 | 36 | 1.68 | 3.58 | 5.58 | 1.9 | 2 | 3.9 | | 1.68 | 3.58 | 5.65 | 1.9 | 2.07 | 3.97 |
| 12 | 25 | 1.62 | 3.7 | 5.52 | 2.08 | 1.82 | 3.9 | | 1.6 | 3.62 | 5.58 | 2.02 | 1.96 | 3.98 |
| 13 | 31 | 1.68 | 3.58 | 5.42 | 1.9 | 1.84 | 3.74 | | 1.68 | 3.65 | 5.62 | 1.97 | 1.97 | 3.94 |
| 14 | 37 | 1.62 | 3.65 | 5.58 | 2.03 | 1.93 | 3.96 | | 1.7 | 3.62 | 5.42 | 1.92 | 1.8 | 3.72 |
| 15 | 29 | 1.65 | 3.62 | 5.52 | 1.97 | 1.9 | 3.87 | | 1.58 | 3.68 | 5.62 | 2.1 | 1.94 | 4.04 |
| 16 | 23 | 1.62 | 3.7 | 5.52 | 2.08 | 1.82 | 3.9 | | 1.58 | 3.7 | 5.64 | 2.12 | 1.94 | 4.06 |
| 17 | 22 | 1.68 | 3.65 | 5.58 | 1.97 | 1.93 | 3.9 | | 1.68 | 3.62 | 5.52 | 1.94 | 1.9 | 3.84 |
| 18 | 29 | 1.65 | 3.65 | 5.52 | 2 | 1.87 | 3.87 | | 1.68 | 3.52 | 5.48 | 1.84 | 1.96 | 3.8 |
| 19 | 20 | 1.65 | 3.68 | 5.62 | 2.03 | 1.94 | 3.97 | | 1.62 | 3.58 | 5.58 | 1.96 | 2 | 3.96 |
| 20 | 37 | 1.65 | 3.62 | 5.48 | 1.97 | 1.86 | 3.83 | | 1.65 | 3.58 | 5.68 | 1.93 | 2.1 | 4.03 |
| 21 | 31 | 1.65 | 3.58 | 5.52 | 1.93 | 1.94 | 3.87 | | 1.65 | 3.62 | 5.64 | 1.97 | 2.02 | 3.99 |

**BRAIN STEM AUDITORY EVOKED POTENTIAL**

| | | | RIGHT SIDE | | | | STUDY | | LEFT SIDE | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | LATENCY | | INTER | PEAK | LATENCY | | | LATENCY | | INTER | PEAK | LATENCY |
| S.NO | AGE | IR | IIIR | VR | I-IIIR | III-VR | I-VR | | IL | IIIL | VL | I-IIIL | III-VL | I-VL |
| 22 | 33 | 1.65 | 3.65 | 5.58 | 2 | 1.93 | 3.93 | | 1.65 | 3.68 | 5.52 | 2.03 | 1.84 | 3.87 |
| 23 | 24 | 1.72 | 3.62 | 5.56 | 1.9 | 1.94 | 3.84 | | 1.62 | 3.6 | 5.46 | 1.98 | 1.86 | 3.84 |
| 24 | 29 | 1.62 | 3.68 | 5.42 | 2.06 | 1.74 | 3.8 | | 1.65 | 3.62 | 5.52 | 1.97 | 1.9 | 3.87 |
| 25 | 25 | 1.68 | 3.65 | 5.62 | 1.97 | 1.97 | 3.94 | | 1.7 | 3.68 | 5.58 | 1.98 | 1.9 | 3.88 |
| 26 | 33 | 1.65 | 3.58 | 5.45 | 1.93 | 1.87 | 3.8 | | 1.65 | 3.65 | 5.48 | 2 | 1.83 | 3.83 |
| 27 | 23 | 1.65 | 3.65 | 5.55 | 2 | 1.9 | 3.9 | | 1.65 | 3.68 | 5.62 | 2.03 | 1.94 | 3.97 |
| 28 | 25 | 1.62 | 3.72 | 5.42 | 2.1 | 1.7 | 3.8 | | 1.68 | 3.63 | 5.48 | 1.95 | 1.85 | 3.8 |
| 29 | 35 | 1.68 | 3.58 | 5.52 | 1.9 | 1.94 | 3.84 | | 1.68 | 3.65 | 5.42 | 1.97 | 1.77 | 3.74 |
| 30 | 33 | 1.65 | 3.65 | 5.46 | 2 | 1.81 | 3.81 | | 1.62 | 3.65 | 5.48 | 2.03 | 1.83 | 3.86 |
| 31 | 27 | 1.68 | 3.75 | 5.48 | 2.07 | 1.73 | 3.8 | | 1.65 | 3.6 | 5.45 | 1.95 | 1.85 | 3.8 |
| 32 | 29 | 1.62 | 3.65 | 5.48 | 2.03 | 1.83 | 3.86 | | 1.68 | 3.68 | 5.48 | 2 | 1.8 | 3.8 |
| 33 | 35 | 1.68 | 3.65 | 5.45 | 1.97 | 1.8 | 3.77 | | 1.68 | 3.62 | 5.42 | 1.94 | 1.8 | 3.74 |
| 34 | 27 | 1.65 | 3.62 | 5.48 | 1.97 | 1.86 | 3.83 | | 1.62 | 3.68 | 5.5 | 2.06 | 1.82 | 3.88 |
| 35 | 39 | 1.65 | 3.65 | 5.42 | 2 | 1.77 | 3.77 | | 1.68 | 3.65 | 5.45 | 1.97 | 1.8 | 3.77 |
| 36 | 21 | 1.65 | 3.72 | 5.6 | 2.07 | 1.88 | 3.95 | | 1.65 | 3.62 | 5.48 | 1.97 | 1.86 | 3.83 |
| 37 | 23 | 1.62 | 3.58 | 5.45 | 1.96 | 1.87 | 3.83 | | 1.68 | 3.62 | 5.48 | 1.94 | 1.86 | 3.8 |
| 38 | 36 | 1.58 | 3.68 | 5.48 | 2.1 | 1.8 | 3.9 | | 1.62 | 3.62 | 5.45 | 2 | 1.83 | 3.83 |
| 39 | 37 | 1.58 | 3.65 | 5.45 | 2.07 | 1.8 | 3.87 | | 1.65 | 3.62 | 5.48 | 1.97 | 1.86 | 3.83 |
| 40 | 27 | 1.72 | 3.58 | 5.48 | 1.86 | 1.9 | 3.76 | | 1.7 | 3.65 | 5.42 | 1.95 | 1.77 | 3.72 |

**BRAIN STEM AUDITORY EVOKED POTENTIAL**

**CONTROL**

| | | | RIGHT SIDE | | | | | | LEFT SIDE | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | LATENCY | | INTER | PEAK | LATENCY | | | LATENCY | | INTER | PEAK | LATENCY |
| S.NO | AGE | IRC | IIIRC | VRC | I-IIIRC | III-VRC | I-VRC | | ILC | IIILC | VLC | I-IIIC | III-VLC | I-VLC |
| 1 | 18 | 1.68 | 3.68 | 5.52 | 2 | 1.84 | 3.84 | | 1.68 | 3.65 | 5.52 | 1.97 | 1.87 | 3.84 |
| 2 | 18 | 1.68 | 3.68 | 5.4 | 2 | 1.72 | 3.72 | | 1.68 | 3.68 | 5.45 | 2 | 1.77 | 3.77 |
| 3 | 36 | 1.58 | 3.65 | 5.6 | 2.07 | 1.95 | 4.02 | | 1.68 | 3.62 | 5.52 | 1.94 | 1.9 | 3.84 |
| 4 | 28 | 1.62 | 3.62 | 5.52 | 2 | 1.9 | 3.9 | | 1.62 | 3.75 | 5.54 | 2.13 | 1.79 | 3.92 |
| 5 | 24 | 1.62 | 3.68 | 5.48 | 2.06 | 1.8 | 3.86 | | 1.62 | 3.65 | 5.45 | 2.03 | 1.8 | 3.83 |
| 6 | 36 | 1.68 | 3.65 | 5.54 | 1.97 | 1.89 | 3.86 | | 1.68 | 3.58 | 5.58 | 1.9 | 2 | 3.9 |
| 7 | 32 | 1.72 | 3.62 | 5.48 | 1.9 | 1.86 | 3.76 | | 1.62 | 3.62 | 5.58 | 2 | 1.96 | 3.96 |
| 8 | 28 | 1.66 | 3.68 | 5.52 | 2.02 | 1.84 | 3.86 | | 1.62 | 3.58 | 5.58 | 1.96 | 2 | 3.96 |
| 9 | 19 | 1.65 | 3.64 | 5.45 | 1.99 | 1.81 | 3.8 | | 1.75 | 3.65 | 5.54 | 1.9 | 1.89 | 3.79 |
| 10 | 27 | 1.68 | 3.64 | 5.48 | 1.96 | 1.84 | 3.8 | | 1.68 | 3.62 | 5.52 | 1.94 | 1.9 | 3.84 |
| 11 | 35 | 1.72 | 3.72 | 5.62 | 2 | 1.9 | 3.9 | | 1.62 | 3.65 | 5.45 | 2.03 | 1.8 | 3.83 |
| 12 | 22 | 1.62 | 3.58 | 5.45 | 1.96 | 1.87 | 3.83 | | 1.68 | 3.68 | 5.62 | 2 | 1.94 | 3.94 |
| 13 | 19 | 1.68 | 3.65 | 5.52 | 1.97 | 1.87 | 3.84 | | 1.68 | 3.72 | 5.55 | 2.04 | 1.83 | 3.87 |
| 14 | 23 | 1.66 | 3.55 | 5.52 | 1.89 | 1.97 | 3.86 | | 1.62 | 3.62 | 5.52 | 2 | 1.9 | 3.9 |
| 15 | 29 | 1.62 | 3.72 | 5.55 | 2.1 | 1.83 | 3.93 | | 1.66 | 3.65 | 5.25 | 1.99 | 1.6 | 3.59 |
| 16 | 35 | 1.65 | 3.68 | 5.45 | 2.03 | 1.77 | 3.8 | | 1.55 | 3.62 | 5.52 | 2.07 | 1.9 | 3.97 |
| 17 | 33 | 1.62 | 3.62 | 5.52 | 2 | 1.9 | 3.9 | | 1.72 | 3.72 | 5.62 | 2 | 1.9 | 3.9 |
| 18 | 31 | 1.68 | 3.62 | 5.58 | 1.94 | 1.96 | 3.9 | | 1.65 | 3.62 | 5.65 | 1.97 | 2.03 | 4 |
| 19 | 28 | 1.65 | 3.68 | 5.48 | 2.03 | 1.8 | 3.83 | | 1.68 | 3.62 | 5.55 | 1.94 | 1.93 | 3.87 |
| 20 | 24 | 1.65 | 3.62 | 5.52 | 1.97 | 1.9 | 3.87 | | 1.65 | 3.62 | 5.55 | 1.97 | 1.93 | 3.9 |
| 21 | 23 | 1.68 | 3.65 | 5.48 | 1.97 | 1.83 | 3.8 | | 1.68 | 3.58 | 5.52 | 1.9 | 1.94 | 3.84 |

**BRAIN STEM AUDITORY EVOKED POTENTIAL**

**CONTROL**

| | | | RIGHT SIDE | | | | | | LEFT SIDE | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | LATENCY | | INTER | PEAK | LATENCY | | | LATENCY | | INTER | PEAK | LATENCY |
| S.NO | AGE | IRC | IIIRC | VRC | I-IIIRC | III-VRC | I-VRC | | ILC | IIILC | VLC | I-IIIC | III-VLC | I-VLC |
| 22 | 34 | 1.58 | 3.68 | 5.52 | 2.1 | 1.84 | 3.94 | | 1.65 | 3.65 | 5.65 | 2 | 2 | 4 |
| 23 | 26 | 1.75 | 3.62 | 5.61 | 1.87 | 1.99 | 3.86 | | 1.65 | 3.68 | 5.42 | 2.03 | 1.74 | 3.77 |
| 24 | 39 | 1.68 | 3.65 | 5.52 | 1.97 | 1.87 | 3.84 | | 1.65 | 3.62 | 5.55 | 1.97 | 1.93 | 3.9 |
| 25 | 25 | 1.68 | 3.68 | 5.48 | 2 | 1.8 | 3.8 | | 1.68 | 3.65 | 5.45 | 1.97 | 1.8 | 3.77 |
| 26 | 29 | 1.65 | 3.65 | 5.52 | 2 | 1.87 | 3.87 | | 1.65 | 3.72 | 5.55 | 2.07 | 1.83 | 3.9 |
| 27 | 18 | 1.62 | 3.58 | 5.48 | 1.96 | 1.9 | 3.86 | | 1.75 | 3.62 | 5.65 | 1.87 | 2.03 | 3.9 |
| 28 | 31 | 1.65 | 3.62 | 5.58 | 1.97 | 1.96 | 3.93 | | 1.68 | 3.65 | 5.62 | 1.97 | 1.97 | 3.94 |
| 29 | 21 | 1.72 | 3.55 | 5.62 | 1.83 | 2.07 | 3.9 | | 1.58 | 3.62 | 5.52 | 2.04 | 1.9 | 3.94 |
| 30 | 24 | 1.65 | 3.65 | 5.6 | 2 | 1.95 | 3.95 | | 1.65 | 3.62 | 5.65 | 1.97 | 2.03 | 4 |
| 31 | 33 | 1.68 | 3.68 | 5.52 | 2 | 1.84 | 3.84 | | 1.65 | 3.65 | 5.65 | 2 | 2 | 4 |
| 32 | 26 | 1.62 | 3.62 | 5.55 | 2 | 1.93 | 3.93 | | 1.65 | 3.62 | 5.62 | 1.97 | 2 | 3.97 |
| 33 | 32 | 1.65 | 3.72 | 5.52 | 2.07 | 1.8 | 3.87 | | 1.65 | 3.65 | 5.65 | 2 | 2 | 4 |
| 34 | 19 | 1.58 | 3.62 | 5.62 | 2.04 | 2 | 4.04 | | 1.65 | 3.62 | 5.65 | 1.97 | 2.03 | 4 |
| 35 | 34 | 1.68 | 3.65 | 5.55 | 1.97 | 1.9 | 3.87 | | 1.62 | 3.7 | 5.65 | 2.08 | 1.95 | 4.03 |
| 36 | 27 | 1.58 | 3.62 | 5.52 | 2.04 | 1.9 | 3.94 | | 1.7 | 3.65 | 5.72 | 1.95 | 2.07 | 4.02 |
| 37 | 35 | 1.65 | 3.68 | 5.55 | 2.03 | 1.87 | 3.9 | | 1.68 | 3.62 | 5.65 | 1.94 | 2.03 | 3.97 |
| 38 | 37 | 1.58 | 3.75 | 5.62 | 2.17 | 1.87 | 4.04 | | 1.62 | 3.75 | 5.65 | 2.13 | 1.9 | 4.03 |
| 39 | 28 | 1.65 | 3.62 | 5.55 | 1.97 | 1.93 | 3.9 | | 1.65 | 3.65 | 5.62 | 2 | 1.97 | 3.97 |
| 40 | 32 | 1.632 | 3.62 | 5.52 | 1.988 | 1.9 | 3.888 | | 1.68 | 3.65 | 5.62 | 1.97 | 1.97 | 3.94 |