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Physiology

# POOR SCHOOL PERFORMANCE AND AUDITORY EVOKED POTENTIAL- A REVIEW

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**ABSTRACT** Poor school performance, defined as a lower than expected academic achievement for a given age, cognitive abilities, and schooling, is complex and multifactorial. Poor school performance can result in child with low self-esteem and can cause significant stress to the parents. It is essential that cause for the poor school performance to be identified and appropriate strategy or treatment plan to be started early so that child can perform with full potential. Disorders of the auditory system affect academic performance are well known. Unlike speech and other behavioral auditory processing tests, auditory evoked potential (AEP) can be recorded regardless of a child's developmental age or language, motivation or attention level. AEP can provide numerical data that can demonstrate central auditory nervous system maturation and, therefore can function as a biomarker of poor academic performance. Present study focuses on the researches that are done on the auditory evoked potential in poor school performance till recently.

**KEYWORDS :** Poor school performance; Low self-esteem; Auditory evoked potential (AEP); Academic performance.

## INTRODUCTION:

Deafness is the most common sensory disability in the world. Deafness affects speech development also. Deafness means loss of hearing and it may be partial or total. Hearing impairment cannot be seen and hence its effects are not visible to others, so deaf suffers in silence. Unlike blindness, deafness often provokes ridicules rather than sympathy. A deaf person is so isolated from family and friends and greeted by unsympathetic attitude, he is often depressed and needs psychological counselling. The consequences for a child born with hearing loss are quite severe. It is well established that a child with hearing loss cannot develop speech and language abilities. This puts the child at a disadvantage in school and limits future professional opportunities. [1]
In this review, use of AEP, its theory and practical application are discussed with special emphasis on poor school performing students.

### Basics of hearing.

The outer or external ear – wax in the ear canal is contributing 15% for deafness which is reversible.

The middle ear is mechanically linked by a chain of three tiny (the ossicles) to another membrane (the oval window) which closes the inner ear. 10% cause for deafness.

The hearing part of the inner ear is rolled up into a spiral canal called the cochlea, as it looks like a snail ('cochlea' is the Greek word for snail).

## AUDITORY BRAIN

Auditory messages are conveyed to the brain via two types of pathway: the primary auditory pathway which exclusively carries messages from the cochlea, and the non-primary pathway (also called the reticular sensory pathway) which carries all types of sensory messages.

## PRIMARY AUDITORY PATHWAYS

Schematically, this pathway is short (only 3 to 4 relays), fast (with large myelinated fibers), it ends in the primary auditory cortex.

The pathway carries messages from the cochlea, and each relay nucleus does a specific work of decoding and integration.

In human, the primary auditory cortex is located in the temporal area within the lateral sulcus.

The first relay of the primary auditory pathway occurs in the **cochlear nuclei** in the brain stem, which receive Type I spiral ganglion axons (auditory nerve); at this level an important decoding of the basic signal occurs: duration, intensity and frequency.

The second major relay in the brain stem is in the **superior olivary complex**: the majority of the auditory fibres synapse there having already crossed the midline.

Leaving this relay, a third neuron carries the message up to the level of the **superior colliculus** (mesencephalus).

These two relays play an essential role in the localization of sound.

A last relay, before the cortex, occurs in the **medial geniculate body** (thalamus); it's here that an important integration occurs: preparation of a motor response (e.g. vocal response).

The final neuron of the primary auditory pathway links the thalamus to the **auditory cortex**, where the message, already largely decoded during its passage through the previous neurons in the pathway, is recognized, memorized and perhaps integrated into a voluntary response.

## NON-PRIMARY PATHWAYS

From the cochlear nuclei, small fibers connect with the reticular formation where the auditory message joins all other sensory messages. The next relay is in the non-specific thalamus nuclei before the pathway ends in the polysensory (associative) cortex. The main function of these pathways, also connected to wake and motivation centers as well as to vegetative and hormonal systems, is to select the type of sensory message to be treated first.

For instance, when reading a book while listening to a record, this system allows the person to pay attention alternately to the most important task.

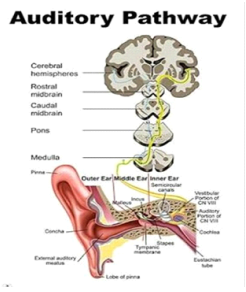



Fig:1

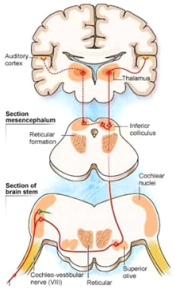



Fig:2

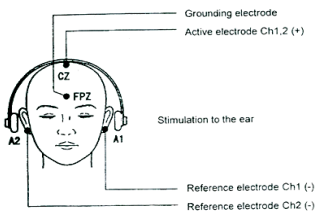



Fig:3

## AUDITORY EVOKED POTENTIALS (AEP)

Auditory evoked potentials (AEP) are records of the changes in electrical potentials in the auditory pathway (Fig1 &2) in response to an adequate external stimulus which can be recorded by surface electrodes (Fig. 3) on the scalp. They are stimulus evoked obligatory responses which are independent of attention or interest of the subject in the stimulus. They reflect the functional integrity of the auditory pathway.

The response to an auditory stimulus has been divided into 3 sequential time periods:

a) Early Latency (0-8 msec) or Auditory Brainstem Response
b) Middle Latency Response (8-50 msec)
c) Long Latency (50-300 msec) or Slow Vertex Response.[2]

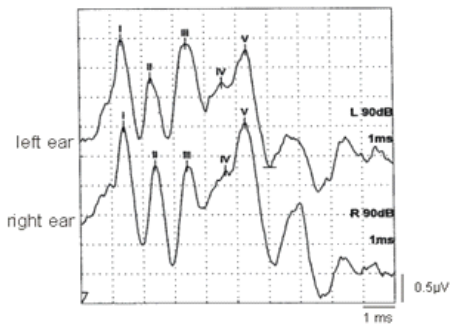
## AUDITORY BRAINSTEM RESPONSE (ABR)

The ABR waveforms are labeled from I-V (Fig. 4) and correspond to the sequential activation of peripheral, pontomedullary, pontine and midbrain portions of the auditory pathways.[3] The wave I is believed to reflect activity in the auditory nerve; waves II and III, activity in the cochlear and superior olivary nuclei and waves IV and V, activity in the lateral leminscus and inferior colliculus.[4](Table 1)

ABR abnormalities have been reported in children with learning problems. This could be due to delay in maturation of auditory pathway in these children. [5-7] Smaller amplitude for wave I, III and V in children with language and motor speech disorders and no change in latency was also observed. The latency time from onset of sound to superior olives (wave III) was observed to be longer in subjects with central language disturbances.[12] Latencies of wave II, III, IV and V, and interpeak latency I-V of ABR was found to be increased in poor performer females.[8]

In contrast, there was no ABR latency or amplitude differences found in primary school children with a learning disability. However, longer wave V latency, longer I-V interval and smaller V-I amplitude ratio was noted in children with risk factors related to brain damage. [9]

**Representative waveform of ABR {Fig:4}**




## MID LATENCY RESPONSE (MLR)

The possible generators of these responses are believed to be thalamus, primary auditory cortex and association cortex. [10]

Past studies have pointed to the simultaneous participation of multiple neural generators in eliciting the cortical response. The inferior colliculus, the medial geniculate body, the reticular formation and the primary auditory area participate in generating MLR together with other associated areas and corpus callosum. The reticular formation appears to be significantly related to primary and non-primary auditory pathways.

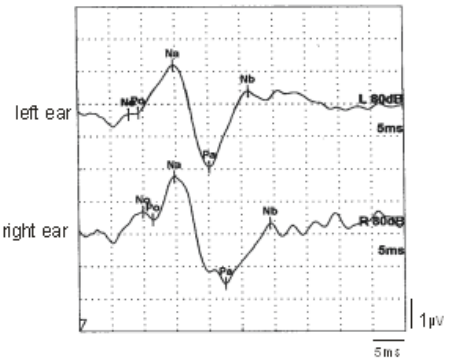
Multiple neural generators form two systems of neural generators involved in generating MLR. One of them is the subcortical portion of the auditory pathway, which develops early; the other is the cortical portion that develops later. Development of a primary neural generator varies among individuals, but is complete at around ages of 10-12 years.

The component waveforms of MLR are labeled as No, Po, Na, Pa, Nb and Pb. (Fig. 5, Table 1).Of the six component waves only Na/Pa complex have been constantly obtained in normal individuals. The No and Po are identified with difficulty due to interference with post-auricular muscle activity, whereas inter individual variability precludes reliable pattern recognition of Nb / Pb components. Pb is probably a cortical response that originates in association areas; it is, therefore, not fully developed until adulthood.

MLR abnormalities have been found in children with learning or speech/language disabilities. The mean Pa latencies of MLR in children with LD were significantly longer than normal children for contralateral recording derivation. MLR wave Na latency was longer and wave Nb amplitude was smaller in LD children.

Delayed $N_a$ and $N_b$ and $P_a$ latencies were measured in contralateral pathway to the left in children with reading and writing disorders. Children with learning disorders showed delayed latencies for $N_a$ wave in the left hemisphere. Differences in morphology of the waves $P_a$, $N_b$ and $P_b$ were observed in children with school complaints.

**Representative waveform of MLR {Fig:5}**

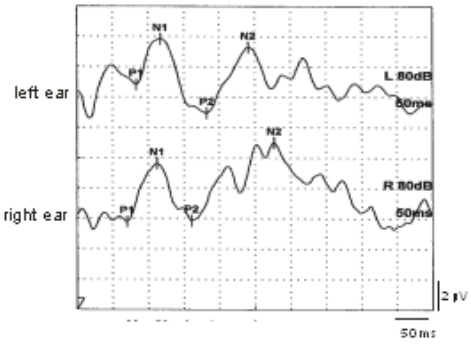



## SLOW VERTEX RESPONSE (SVR)

The long latency responses or SVR waveforms i.e. P1, N1, P2 and N2 (Fig 6) are widespread in their distribution over the fronto-central scalp area. Vaughan and Ritter suggested that these potentials arose from primary auditory cortex and temporo-parietal association area and had latency of 50-300msec.[11] (Table 1). The primary auditory cortex exerts a control over association cortex response through cortico-cortical and corticothalamocortical connections. The integrity of primary cortex was essential for generation of these potentials.

Researchers have found a variety of P1-N1-P2 and P3 abnormalities, including increased absolute and inter wave latencies, increased or decreased P2 amplitude, and increased hemispheric asymmetry in LD children. N1 and P2 latency and N2 latency was delayed.

**Representative waveform of SVR {Fig 6}**




## EVENT RELATED POTENTIALS (ERPs)

The term Event Related Potentials or ERPs refers to the responses evoked due to various mental work loads when a stimulus and the problem related with that stimulus are applied. They occur only when the subject is selectively attentive to the stimulus and are elicited in conditions where the subject has to distinguish a target stimulus from non target stimuli. [12]

### Components of ERPs

The long latency response to a rare auditory stimulus consists of different waves i.e. N1, P2, N2, and P3 (also called P300). The N1 and P2 components are believed to reflect the activity in neural areas that are activated by sensory modality and are independent of the subject's attention. The N2 component is related to the unexpectedness of the stimuli.

P3

P3 has attracted maximum attention because it is associated with psychological processing.[35] The P3 wave is believed to reflect cognitive processes underlying attention allocation and memory updating. Thus any event related potential includes an early sensory evoked potential and a late (cognitive) response P3 component.

Several studies have reported a smaller or later P300 in developmental dyslexics and in children with attention-deficit disorder, suggesting inefficient processing of task-relevant stimuli. P300 abnormalities were found among adults with childhood dyslexia only in those also suffering from attention-deficit disorder.

A significant latency increase for the N1, P2 and P3 components in the children with auditory processing disorders was found. The P300 responses occurred at significantly longer latency periods.

In LD children it was observed that for standard and deviant stimuli P1 latencies were shorter, P3 latencies were longer, and P3 amplitudes were smaller while for standard stimuli, N1 amplitude was smaller and P2 was earlier and for deviant stimuli, N1 was earlier and P2-N2 was smaller.

## DISCUSSION

ABR from various studies shows conflicting results. Stimulus and recording characteristics may account for the differences in these studies. ABR can be elicited by a wide variety of sound stimuli: click, pure tone, masked tone, and complex sounds (speech). According to Chandrasekaran and Kraus, the analysis of absolute latency values for ABR with speech stimuli can help differentiate clinical pictures, and allow for an objective measure of subcortical speech processing.[13] Researchers stress that this procedure can be used to evaluate auditory function and provide additional information in the diagnosis of learning disabilities and auditory processing disorders.[14]

Central auditory processing disorder (CAPD) refers to the mechanisms of the auditory system associated with the sound source localization, auditory discrimination, auditory pattern recognition, temporal aspects of audition (temporal resolution, masking, integration and sequencing) and performance decrements in response to competing signals or degraded signals.[15] CAPD is defined as a deficiency in one or more of these auditory mechanisms, which in children account for difficulty in learning language or learning to read. Studies on MLR, SVR and P300 response to target stimuli showed that latencies was prolonged suggesting prolonged time required for auditory processing which is crucial for understanding sound and responding to it.

Bruton conference (Jerger and Musiek, 2000) on auditory processing disorder (APD) concluded that electrophysiologic testing can assist in the differential diagnosis of APD in school-aged children. The Bruton conference recommended the use of ABR and MLR.[16]

**Table 1: Neural generators of evoked potential components**

| S.No. | | Name of the Components | Neural Generators |
|---|---|---|---|
| 1. | Short latency auditory evoked potential | Wave I | Auditory portion of the eight cranial nerve |
| | | Wave II | Near or the cochlear nucleus |
| | | Wave III | The lower pons through the superior olivary nuclei and trapezoid body. |
| | | Wave IV | The upper pons or lower midbrain, |
| | | Wave V | in the lateral lemniscus and the inferior colliculus; a contralateral brainstem generator for wave V is suggested. |
| 2. | Mid latency auditory evoked potential | Na | Medial geniculate body |
| | | Pa | Superior temporal gyrus. |
| | | Nb | Doro- posterior medial part of Heschl's gyrus that is the primary auditory cortex |
| 3 | Long latency auditory evoked potential | N1 | Secondary auditory cortex in the lateral Heschl's gyrus. |
| | | P1 | Bilateral parts of the auditory superior cortex |
| | | N2 | Mesencephalic – reticular activating system (RAS) |
| | | P2 | Anterior cingulate cortex |

## CONCLUSION

The present study has shown that auditory evoked potential assessment of children with poor school performance is important considering the fact that alterations in speech sound processing appear to be of crucial importance in the learning process. AEP is objective, reliable and does not require patient's conscious participation. Identification of auditory disorder is essential for any therapeutic strategy to be developed in these children, for prognosis and therapeutic effectiveness. However, future studies with varying parameters, for example, using speech stimuli is needed to validate the test.

**Conflicts of interests :** The author declare that there is no conflict of interests.

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