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# AUDITORY PLASTICITY AND COCHLEAR IMPLANTS IN CHILDREN- A REVIEW

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## ABSTRACT

**Aim:** The review article is aimed at highlighting the importance of Auditory Plasticity for the positive outcomes in cochlear implantation, which is intrinsic to the critical age period.

**Method:** Various electronic databases such as PubMed/ Medline, Science Direct, CINHALL etc. were used to extract the articles. The keywords used for the search engine were 'auditory plasticity', 'deprivation', and 'cortical re organization', ' cochlear implants'.

**Discussion:** The current review suggests that auditory plasticity is important in achieving proper wiring of acoustic sensation in the congenital hearing loss cases. Critical age period for the intake of new information and learning occurs before 3.5 years of age and post critical period the language learning is delayed.

**Conclusion:** Auditory plasticity and its related functions are critical in influencing the outcome after the CI in children. The advent of electrophysiology into clinical research has paved a path breaking journey in understanding the complex processing of auditory signals in the primary and secondary cortical areas.

**Key Words:** Auditory Plasticity, Auditory Deprivation, Cortical Reorganization, Cochlear Implants

## INTRODUCTION

Auditory plasticity is defined as the functional and structural changes occurring in the auditory cortex during the normal course of development. The ability of the auditory system to enhance the auditory sounds in the auditory cortex is pivotal for the successful speech perception in humans. The auditory plasticity is intrinsic to the critical age period, which defines the age period during which the neuronal and other functional developments occur in the brain for the normal wiring in the auditory system [1]. The interest in the research prospects in auditory plasticity has reached its prime since last two decades due to the introduction of the cochlear implants (CI) for the severe to profound hearing loss individuals. The success of the CI was determined based on the outcome in auditory and speech development in cochlear implantees. Poor outcome on CI was initially attributed to the performance of the implant or the speech processor used by the person. Little was known on the auditory plasticity and the effect of auditory stimulation in the auditory cortex, until technological advances allowed researchers to drive deep into the system of the complex auditory processing using fMRI, PET, and

evoked potential studies. Researchers in the field of auditory plasticity believe, a lot more intense research is required to unravel the mystery that presents in the primary auditory cortex; which in turn facilitates the hearing impaired to respond and process the complex speech signals.

The advent of electrophysiological measures has paved a path-breaking journey to understand the auditory development, auditory plasticity and critical age period in humans. A bulk of studies in literature on the critical age period and auditory plasticity was done on animals, especially rats and rodents [2,3,4]. The results were later extrapolated into human research. The basic research question that intrigued most of the researchers is that, if the deprived auditory cortex responded to the electrical stimulation through the CI or not. Also, if the poor outcome in CI patients was related to the reorganization of auditory cortex due to the long-standing auditory deprivation. They also researched if the other senses have taken over the functional areas of the auditory system. Such a set of complex research questions requires extensive research studies that would primarily focus on using non-invasive methods. This method being cost effective for the

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researchers is expected to create ripples in the field of auditory plasticity and CI.

## MATERIAL AND METHODS.

A detailed literature review was carried out to review the auditory plasticity and cochlear implants in children with respect to the outcome based in CI children. The review was focussed mainly on the following 4 areas; (a) auditory deprivation on the auditory cortical development and critical age period; (b) functional and metabolic changes in the auditory cortex due to auditory deprivation; (c) Electro physiological evidence in auditory plasticity after CI;(d) auditory decoupling hypotheses; (e) cortical reorganization and cross modal plasticity. The literature search was done independently by the author. Various electronic databases such as PubMed/ Medline, Science Direct, CINHAL etc. were used to extract the articles. The keywords used for the search engine were ‘auditory plasticity’, ‘deprivation’, and ‘cortical reorganization’, ‘cochlear implants’ The extracted articles were further screened to segregate the four key areas of the aim of the current review.

## RESULTS

All the extracted electronic articles were categorised according to the five core areas namely (a) auditory deprivation on the auditory cortical development and critical age period; (b) functional and metabolic changes in the auditory cortex due to auditory deprivation; (c) Electro physiological evidence in auditory plasticity after CI;(d) auditory decoupling hypotheses; (e) cortical reorganization and cross modal plasticity. Journals which are printed in English as a primary language as been wetted for the review. The final articles were reviewed independently by the authors.

## DISCUSSION

The aim of the current review article is to update on the auditory plasticity and its effect on post cochlear implantation in children. The reviewed articles on the specific areas were measured on the above mentioned four key areas and discussed in detail further.

### a) Auditory deprivation and critical age period

Auditory plasticity is intrinsic to the critical age period, and defines the ability of the auditory systems to undergo certain plastic changes that may be irreversible in nature. These changes may not be retrained in later life. During this period the human brain undergoes various structural and functional changes through the natural course of development [5]. The

development of sound detection and localisation along with other segmental and suprasegmental features in speech are all wired during this period. This period prepares the humans to face the competitive acoustic world through the normal course of action. Any disturbance in the natural development of the central auditory system or the auditory cortex may result in the tonotopic changes in the auditory system. This shall subsequently alter the acoustic image in the auditory cortex [6]. Therefore, the accurate and precise flow of acoustic signals during the early years of life is critical in wiring and strengthening the auditory centres in the auditory cortex. Gaps in this flow may lead to the auditory derivation, which will in turn stall the normal auditory development. The arguments advocating the development of auditory function during the critical period is well established in the literature and enormous studies have been done on rats to identify its effects and further the results were extrapolated to understand the human nature [7, 8]. One of the recent studies by Kral [3] states that initial post-natal exposure to the auditory system in rats is critical in developing the auditory plasticity in them, as the auditory centres were highly sensitive during this period compared to the other centres in the brain. Therefore, the initial acoustic stimulation would enhance the overall wiring of the auditory cortex. This would encapsulate the functioning of the later stages. Study done by *Meridith & Allamn* [4], in rats proved that the initial post-natal auditory stimulation during the first 13 days changes the sound representation in the auditory systems. However, if the system is exposed post 30 days, no differences in the representation of the acoustic image were found. This supports the notion that lack of early auditory stimulation would lead to the auditory deprivation [9, 10]. The outcome of all these experiments directs to an important fact that the early acoustic exposure is critical for the auditory cortical development in rats during the early days of life.

It is believed that attenuation or blockages of ongoing acoustic signal in the auditory system will misalign the wiring of auditory afferent development in children during the critical period [11]. These facts have been well explained in various studies by the authors with examples of long-standing middle ear pathology- Otitis media in children. These children have shown potential decline in various auditory processing tasks and have shown poor academic performance. Such lack of exposure incidents have later progressed to auditory processing disorders in children. Thus, reaffirming the fact that any disturbance in the incoming signals during the critical age period would affect the normal course of auditory development. Kral and Sharma [12] reported that in hearing impaired children with lack of acoustic stimulation, there is a disassociation of the primary auditory cortex from that of higher cortical centres in the brain. This was predominantly observed in children who were implanted late and found to be having poor outcome from the CI. They believe that the

normal cortical development is simply based on the stimulus driven learning. Thus, lack of stimulation during the early years of life would end up in auditory deprivation. Rewiring of such areas during the rehabilitation in children post cochlear implantation would therefore be an enormous task.

The effect of auditory deprivation is enormous and plays an important role in determining the outcome from any form of habilitation/rehabilitation, be it through hearing aids or cochlear implants. During the early learning period, an exposure to a new stimulus would largely reorganise the acoustic representation in the brain [13, 14]. In this process both cortical and subcortical regions play a significant role. The rewiring of the system to understand the acoustic features and later categorize into the environmentally important sounds is essential for the overall representation of the acoustic image in the brain [15]. This processing is easily done in the plastic brain in young children than in those who have crossed the critical age period.

### **b) Functional changes in the auditory cortex**

Auditory deprivation will lead to various changes in the alignment of the auditory cortex and central auditory nervous system (CANS). The lack of stimulation is expected to disassociate the morphological structure. It also brings about various other metabolic changes in the system which is essential for the synaptogenesis and maturation of the higher order centres. The resulting alterations due to auditory deprivation include changes in the volume of neurons in the spiral ganglion, changes in the neuronal projections in the CANS and auditory cortex, the regulation of intracortical and thalamocortical neural activities, pruning in pyramidal cells, decoupling of cortico-cortical neural fibres and so on [3,16]. These changes will have detrimental effects on the auditory function and relate to the auditory rewiring or reorganisation. The excitatory and inhibitory changes due to auditory deprivation are profound as they are expected to play a major role in synaptic pruning and for the accurate transmission of the acoustic sensation to the brain.

All these factors need to be taken into account prior to the cochlear implantation in children, especially congenial hearing loss cases. During the early years of life, the auditory cortex and CANS undergoes various developmental changes, one such process is the synaptogenesis. Synapses undergo continuous development and disappear after few days. These continual processes in the central auditory pathway and the auditory cortex are traced using various functional measures for better comprehension [17]. It is believed that in cats, during the initial months, synaptogenesis occur in the auditory cortex. In deaf animals during the same period there was a delay noticed of approximately 2 months. This in turn proves the notion that lack of auditory stimulation shall lead to functional changes in the auditory system. This

substantiates the need for early identification and stimulation through cochlear implantation in the congenital hearing loss individuals. This rewiring of the auditory system would also have a pronounced decrease in the synaptic plasticity in the auditory cortex, followed by changes in the excitatory and inhibitory function in the auditory cortex [11, 18]. Such molecular level changes hugely relied on the input signal from the peripheral structures. The lack of auditory streaming of signals would eventually enhance the strengthening of visual cortical functions. The changes in the interplay between the cortico cortical structures will also affect the processing of acoustics signals into the brain. It is a proven phenomenon that the perceptual skills are purely dependent on the interaction between the top down and bottom up process. Such a complex interaction allows the individuals to understand the highly time varying speech sounds in various complex situations.

### **c) Electrophysiological evidence on Auditory plasticity after Cochlear implantation**

The ability of the auditory system to continuously process the acoustic signal is integral for the normal speech and language development in humans. Such an auditory capacity requires integration of multiple systems of acoustic cues, which humans constantly adjust according to the behaviour and environmental needs. Cochlear implants are one of the most advanced forms of hearing restoration currently available for the hearing-impaired individuals. Electrophysiology/evoked potentials (EP) have been constantly used in the diagnosis and monitoring of the cochlear implant outcome in children and adults. One of the recent themes of research using EP is focussed on the maturation of auditory function in children with cochlear implants. Due to the increased demand for evidence based clinical research, cochlear implant outcome measures are majorly utilized with the addition of EPs, mainly cortical auditory evoked potentials (CAEPs). These potentials are generated from the primary auditory cortex in response to an acoustic stimulus, predominantly the speech stimulus. The first peak in CAEP, P1, can be used as the biological marker for cortical auditory processing and maturational aspects as studied by Sharma and Dormon [1]. They have done enormous research in the maturation of P1 in children with hearing impairment and also post CI. Their research states that children who are identified early and done with CI would show greater development of cortical auditory processing compared to those who have done CI after 9 years. These results are again supportive of the much-debated critical age period for children to learn the language through auditory mode.

Cardon et al [19] studied the central auditory maturation in children with ANSD using CI and reported that the children who were implanted before 2 years showed normal P1 latencies compared to the group which was implanted after 2

years. They believed that in children with ANSD, the sensitive period would be much shorter than that of hearing impaired children. Thus, suggesting the clinical importance of CAEP. The varied interest in the application of EPs in CI patients dates back to early 2000. A series of studies have been conducted by various researchers to underpin the developmental course in auditory cortex by monitoring the latency measures of various peaks [17].

Similar line of studies was conducted by Sharma and Colleagues [1,8,20] by comparing the P1 latencies in various groups of children. In fact, they have done large-scale studies on children to prove their basic research question of using P1 as a biological marker for auditory processing in children. Interestingly, these studies also highlighted the CI candidacy, during the process of monitoring the cortical maturation after hearing aid amplification in children. The breakthrough research finding was that some children who were fitted with hearing aids would not show any change in P1 latencies even after 6 months of continuous stimulation. This could eventually lead to the conclusion that acoustic amplification is barely helpful and these children can be considered for CI, if the other parameters are supportive.

The use of CAEPs also helped the clinician to objectively quantify the aural rehabilitation outcomes after cochlear implantation. In some children the development of P1 would go through the natural process [1]. However, the following negative trough would be concordant with the neural strengthening between primary and secondary cortex. This shed light into the possible dent in speech perception in CI children as against speech detection after CI. These changes in the cortical structure are thought to be due to the auditory deprivation. Further, the central auditory plasticity needs to be rewired with continuous stimulation through CI. The underlying mechanisms for the rapid changes in P1 latencies are not clear. Yet, it is believed that the structural resynchronization of granular layers in the auditory cortex with improved neuronal firing could be attributed as a reason for the rapid development. The studies done in animals [2, 3] have reported that, during the early days of stimulation there is rearrangement of cortical functioning occurring in the deaf cats. These evidences when extrapolated into humans, advocate that in young children the cortical stimulation after CI would enhance the cortico cortical structures. Major rewiring would thus occur due to the rapid stimulation in the auditory cortex. However, these measures are interpreted with caution, as it requires in depth analysis with much more research.

Certain sections of researchers believe that the synaptic plasticity is the key in the development of auditory cortical function in the auditory cortex. Kral & Eggermont [18] reported that there is massive increase in synaptic connections during the post-natal period and continues to develop until 4 years of age. This is considered as synaptic overshoot. Therefore,

during this period the development is intrinsically regulated. Any alteration before 4 years of age will thus have a major impact on the potential development. This could be a probable explanation for the sensitive period and cortical maturation in children who were implanted before 4-5 years of age. The same declined in children who were implanted after 5 years. Auditory plasticity is thus age dependent; however, certain sections of researchers still believe that it can be rewired even after the sensitive period.

#### **d) Auditory decoupling hypothesis**

In congenital hearing loss subjects, a long-standing perception remains that after the sensitive period, the cortical activation would be degraded. In a breakthrough research, Kral et al [5] reported that in congenitally deaf white cats, the sensitive period is approximately first 4 months. During this period acoustic stimulation would show some amount of activation in the auditory cortical region. However, after the sensitive period, the auditory stimulation shows degraded responses in supragranular and infragranular layers in the auditory cortex. The delay in the activation seen at the supragranular layer level and absence of infragranular layer indicates the incomplete developmental process. This affects the free flow of signal to top down and bottom up region, which could be attributed to the lack of synaptogenesis during the post sensitive period. As a result, they reported that there would be functional decoupling of the primary auditory cortex from that of the higher order cortical regions for acoustic processing. It is also believed that this functional decline in processing would have a detrimental effect in strengthening of the sub cortical auditory pathways. If these results could be extrapolated to humans, then it is imperative that the children who are implanted post sensitive period would show functional decline in the various cortico-cortical structures in the auditory cortex. Also, its effects could be seen in the sub cortical development after the implantation. All these results eventually point at the functional outcomes of CI in children.

It was also reported that the human auditory cortex would maintain a rudimentary capacity in processing the incoming acoustic signal, even in the absence of primary auditory stimulation[18]. However, the delay or lack of acoustic stimulation would lead to the under development of the higher order cortical structures that are responsible for the primary auditory processing. It will also reduce the speed at which signals are corresponded to the auditory areas in the brain. Overall, these functional declines in the system would deteriorate the rapid development of the combined bottom up and top down processing. As a result of the decoupling, the other sensory areas in the brain would strengthen their territory, eventually rendering it difficult to rewire after the sensitive period. This could be a prime reason to justify the poor outcomes and poor auditory processing seen in children who are implanted post sensitive period. The functional coupling



of all these areas is integral for linguistic learning. The decoupling of higher order cortical areas would result in poor speech processing skills in young children after the sensitive period [21]. Thus, substantiating that the auditory plasticity is intrinsic to the time period of learning, especially in congenital hearing loss children.

### e) Cortical reorganization and cross modal plasticity

It is a proven theory that the sensitive period is critical in normal development of auditory processing in humans. Late implanted children after sensitive period would show delayed representation of the acoustic image in the CANS and auditory cortical areas. This could be due to the lack of early stimulation in the auditory cortex and consequent reorganization of cortical function. To prove this significant phenomenon of cortical reorganization in children, Gilley et al [15] studied the EEG responses for the stimulus /ba/ in normal hearing, early and late implanted children. They found that in normal hearing children, there was normal activation of superior temporal sulcus and inferior temporal gyrus. Similar near normal activation was seen in children who were implanted before 3.5 years of age, which is considered as cut off duration for the sensitive period. Interestingly, post sensitive period implanted children showed certain activation areas in the anterior temporal cortex, insular areas etc., that are considered as the multi modal areas in the brain. In addition, activation was seen in visual cortical regions justifying the notion that during the post sensitive period, cortical areas undergo various changes functionally and reorganization of the areas takes place [20].

The impact of cortical reorganization in CI children was an interesting area of research for many researchers. It will be interesting to figure how the already re organized areas in the brain would accommodate the new incoming auditory stimulation. The speculation is that the auditory areas in the brain would have already been wired to other senses predominantly visual function [22]. If such a takeover has occurred, then the new input signal would face competition to find its place in the cortical regions. Such an action would enhance the already reorganized brain to adopt new forms of plasticity to accommodate the new signal. There are various studies done in these areas, to assess the effect of auditory mode of stimulation versus combined auditory –visual stimulation in late implanted children. Bergerson [23] reported that, children who are implanted before the age of 4 years showed better responses for the auditory mode alone experiment and those who were implanted after 4 years relied more on the combined auditory visual mode. These results are in conjunction with the earlier theory that cortical reorganization hampers the learning period for the late implanted children. Also, it was reported that in cases with congenital hearing loss, the auditory cortex was activated for visual input signal for the

location of the objects in space. Thus, underlining the cross modal plasticity that occurs during the post sensitive period [4].

## CONCLUSION

To conclude, auditory plasticity and its related functions are critical in influencing the outcome after the CI in children. Various studies in literature have pin pointed the impact of sensitive period in auditory and linguistic learning in congenital hearing loss children. The advent of electrophysiology into clinical research has paved a path breaking journey in understanding the complex processing of auditory signals in the primary and secondary cortical areas. Lack of auditory stimulation in the early years would lead to the cortical decoupling and the cortical re organization. Taken together, auditory plasticity serves a unique form of function that would allow the brain to rewire the incoming signal after the implantation. This subsequently allows them to learn the new world of sounds. Research in this area of unravelling the mystery behind the cortical plasticity and sensitive period is still naive. The unprecedented increase in the CI in children in recent years has paved way for more research in coming years for better understanding of the sensitive period. Further research is also warranted to study how auditory plasticity could be detrimental to obtain good outcomes after CI in children.

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