Article *NIMHANS Journal*

# Auditory Brainstem Responses in Autistic Children .

These papers form a part of the proceedings of the symposium on "Childhood Language Disorders - Autism" held from 6-8 February 1985 at NIMHANS, Bangalore. Six papers of this proceedings have appeared in the January 1989 issue.

**Volume: 07 Issue: 02 July 1989 Page: 159-162**

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The faulty modulation of sensory input, which is a key feature of the autistic syndrome has drawn the attention of many researchers and as a result, has been put into close scrutiny to evaluate the physiological dysfunctions. The studies include electroencephalographic responses [1], [2], [3], [4], autonomic responses [2], [5], [6], [7] vestibular responses [8], [9], [10] and auditory brainstem responses [11], [12], [13], [14], [15], [16], [17]. In almost all the cases the results have demonstrated brainstem dysfunctions giving way to propose a brainstem model for the abnormal behaviour so evident in these children.

However, these symptom complex argue for a central rather than a brainstem defect [18], which we also endorsed. The role of intricate neural activity of the subcortical structure in the development of higher cortical functions cannot be overlooked.

With the growing interest in studying the auditory brainstem dysfunctions in autistic children and because of the fact that auditory brainstem responses are very sensitive, stable, unaffected by the arousal of the subjects, easily reproducible and provides a reliable measure of the functioning of brainstem in various neurological conditions even disclosing the subtle lesions affecting the normal functioning of the brainstem, we carried out a study to record the auditory brainstem responses in autistic children.

## Methodo1ogy

### Subjects

Seven autistic children (5 males and 2 females) age ranging from 1 to 11 years (mean age 6 years) constituted the experimental group. These children were referred from the child guidance clinic of the Department of Psychiatry, NIMHANS, Bangalore, India, after the complete assessment. The diagnosis was made based on the ICD-9 classification.

Twenty normal children (10 males and 10 females) age ranging from I to 15 years (mean age 8 years) constituted the control group.

Speech and language evaluation was made in all the seven children at the Department of Speech Pathology, NIMHANS. Of the seven children, four had limited communicative speech and three had

absolutely no communicative skills. There was no history of prenatal, natal or postnatal complications in all these children except for one child who had an attack of generalized epilepsy at the age of ten. None of these children. were on any kind of medication at the time of ABR study.

## Procedure

Standard set procedures were adopted for recording the auditory brainstem responses in both the groups, and wherever necessary under sedation, in subjects who were non-cooperative. The auditory brainstem responses were recorded in a sound proof and electrically shielded room at the Department of Neurology, NIMHANS. DISA 15CO1 multichannel recording system was used for the ABR measurements. The electrodes were placed as per the International Standard of 10-20 system and accordingly Cz-vertex as the positive with reference electrode over the mastoid and ground electrode over the forehead. The electrical impedance was kept below 3-5 kilo ohms. Rarefaction doom shaped clicks were presented through TDH-39 earphones. A total of 1024 clicks with a stimulus rate of 10 clicks per second were presented monaurally at an intensity level of 60 dB SL with contralateral masking noise of 60 dB. The responses were displayed and measured on the oscilloscope and further recorded on an aluminium foil sheet.

The experimental group were initially examined otoscopically to rule out any external or middle ear pathologies. Incidentally, all the seven subjects had good record of aural hygiene. The auditor brainstem responses were successfully recorded in all the seven subjects in both ears, wherever necessary under sedation for those who were non-cooperative. In all the subjects, the measurements were recorded twice for cross validation.

The interwave latencies between I-III, I-V and III-V were measured to determine the functional integrity of the auditor pathways in the brainstem. The mean values of the interwave latencies in the control group ± 2.5 SD were considered as normal.

# Results

Table I shows the interwave latencies in the experimental group of each subject in each ear as against the normal mean latencies with ± 2.5 SD. Of the seven subjects studied, four subjects (subjects P M, S and D) had interwave latencies within normal limits, and the remaining three subjects exhibited prolongation of inter wave latencies. The subjects K and B had prolongation of interwave latencies of III-V suggesting involvement across the rostral portion of the brainstem and the subject I, had prolongation of interwave latencies of I-III and I-V suggesting involvement across the caudal portion of the brainstem.

***Table Ia - Mean interwave latencies of each subject in each ear in the experimental group as against the mean interwave latencies of the control group***

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***Table Ib - Mean interwave latencies of each subject in each ear in the experimental group as against the mean interwave latencies of the control group***

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***Table Ic - Mean interwave latencies of each subject in each ear in the experimental group as against the mean interwave latencies of the control group***

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## Discussion

The present study reveals pathological ABR in three subjects (42.8%) pointing towards the dysfunction at the level of brainstem favouring the brainstem model. The results also show that there exist two groups of autistic children, one with completely normal ABR and the other with pathological ABR. The results obtained in this study are quite similar to that of Rosenblum et al [14] Skoff et a1 [15], Tanguay and Edwards [16] and Gilberg et a1 [17].

The results of the present study together with the earlier reports on ABR in autistic children, indicated pathological ABR in as much as 50% of the autistic population and argues for the faulty modulation of the auditory input thus leading to failure in the development of complex cognitive skills. It would be more so if such distortions occur in the early post natal conditions.

The fact that there exists two sub-groups in autistic population, one with pathological ABR and the other with normal ABR, needs further exploration and the present study does not confirm the observation that there exists a tendency for autistic children with pathological ABR results to be without language more frequently than other autistic children [17].

It would be of interest if one can track both the groups for a longer period to assess the language attainment after sufficient training and to correlate with the ABR results, to see whether ABR can be used a prognostic indicator of the future development of the cognitive skills in autistic children.

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