Effect of stimulus intensity on short-latency auditory evoked potentials in persons with Down's syndrome

M. Zurrón, A. Amenedo and F. Díaz

Departamento de Psicoloxía Clínica e Psicobioloxia, Facultade de Psicoloxia, Universidade de Santiago de Compostela, Campus Universitario, 15705 Santiago de Compostela, A Coruña, Spain

Keywords: Short-latency auditory evoked potentials (SAEPs), BAEPs, Down's syndrome.

Introduction: Studies of short-latency auditory evoked potentials (SAEPs or BAEPs) in persons with Down's syndrome (DS) [1–10] have shown several differences in comparison with non-retarded subjects. These are: a shortened I-V interval [4, 5, 7, 9], smaller amplitudes in one or more components [2, 3, 10], a smaller increase in the latency of V in response to increased stimulus presentation rate [7–9], and a shorter wave V latency for stimulus intensities above a certain level [1–3, 7, 8, 10]. Levels for the latter effect range from 40 to 75 dB nHL (normal hearing level; dB above the auditory threshold of a sample of healthy subjects with normal hearing) [1, 7, 9, 10].

To investigate these discrepancies, and with a view to furthering knowledge of neurofunctional alterations in general in Down's syndrome, we have studied the effect of stimulus intensity on the latency of wave V in DS subjects, and the differences between DS and non-retarded subjects as regards other SAEP components.

Materials and methods: We studied six males and six females with Down's syndrome, IQs < 50 by the Wechsler Intelligence Scales (WISC or WAIS) and ages ranging from 10 to 17 years (mean 14.17 years). We also studied seven male and seven female non-retarded controls with IQs (WISC or WAIS) of 90–115 and ages ranging from 11 to 19 years (mean 13.64 years).

During each session, the subject sat in a comfortable armchair in a partially sound-proofed, electrically isolated room. At the beginning of each session, the auditory threshold of each of the subject's ears was determined for click stimuli.

Evoked potentials (EPs) were elicited by 100 μs rarefaction clicks presented through headphones to the ear with the lower auditory threshold at a rate of 10 clicks s^{-1}, while a −40 dB white noise was presented to the other ear. Three stimulus levels were employed: 25, 45 and 65 dB SL (sensation level; dB above the individual's auditory threshold).

EEG were recorded with a sensitivity of 25 μV via Ag-AgCl electrodes, with the recording electrode at Cz, the reference electrode on the lobe of the stimulated ear and a ground at Fpz; impedance was always less than 5 kohms. After passage through a 150–3,000 Hz bandpass filter, the EEG signal was digitised at 512 points for 10 ms after stimulus onset. For each stimulus level, 2,000 artifact-free sweeps were averaged.

The variables studied on SAEPs recorded at 65 dB SL were the peak latencies and peak-to-trough amplitudes of waves I, III and V, and the interpeak intervals I–III, III–V and I–V. The variables studied on SAEPs recorded at 45 dB SL were the peak latencies of waves III and V and the peak-to-trough amplitude of wave V. For 25 dB SL SAEPs, only the peak latency and peak-to-trough amplitude of wave V were determined.

Table 1: Data for non-retarded and DS groups at 65 dB SL, with group × sex ANOVA results for group effect (means ± SD). Latencies and inter-peak intervals are in ms and amplitudes in μV.

Variable	Non-retarded	Down's syndrome	ANOVA F	df
Lat. I	1.60 ± 0.15	1.51 ± 0.17	2.90	1, 20
Lat. III	3.85 ± 0.18	3.49 ± 0.15	28.58	1, 22**
Lat. V	5.68 ± 0.28	5.37 ± 0.21	9.13	1, 22*
Int. I–III	2.25 ± 0.20	2.00 ± 0.15	10.46	1, 20*
Int. III–V	1.83 ± 0.28	1.88 ± 0.18	0.27	1, 22
Int. I–V	4.08 ± 0.29	3.86 ± 0.24	3.31	1, 20
Amp. I	0.46 ± 0.21	0.62 ± 0.38	1.59	1, 20
Amp. III	0.41 ± 0.18	0.22 ± 0.08	11.99	1, 22*
Amp. V	0.47 ± 0.20	0.50 ± 0.17	0.14	1, 22

* $p < 0.01$; ** $p < 0.001$.

The data were subjected to various 2 × 2 (group × sex) analyses of variance. In addition, 2 × 3 (group × intensity) repeated measures ANOVA was performed on the latencies and amplitudes of wave V. All statistical processing was carried out using the SPSS/PC+ package. Results are expressed as means ± SD.

Results: The auditory thresholds of the non-retarded and DS subjects were respectively 46 ± 4.01 and 61 ± 8.56 dB SPL (sound pressure level, by physical measurement of sound-wave pressure).

For stimuli of 65 dB SL (Table 1), the latencies of III and V were significantly shorter in DS than in non-retarded subjects ($F(1, 22) = 28.581$, $p < 0.001$ for III; $F(1, 22) = 9.131$, $p < 0.01$ for V), the I–III interval was significantly shorter ($F(1, 20) = 10.458$, $p < 0.01$), and the amplitude of III was significantly smaller ($F(1, 22) = 11.99$, $p < 0.01$). The I–V interval was also shorter in the DS group, but without meeting the $p < 0.05$ significance criterion ($F(1, 20) = 3.312$, $p = 0.084$).

Sex had no influence on any variable, either alone or as a group × sex interaction. For stimuli of 45 dB SL (Table 2), the latencies of III and V were again significantly shorter in DS than in non-retarded subjects ($F(1, 20) = 15.643$, $p < 0.01$ for III; $F(1, 22) = 4.761$, $p < 0.05$ for V), and again sex and group × sex interaction had no significant effect.

For stimuli of 25 dB SL (Table 3), the latency of V was significantly shorter for females than males ($F(1, 22) = 4.410$, $p < 0.05$). But there was no significant effect on the latency or amplitude of V due to group or the group × sex interaction.

Repeated Measures ANOVA for the wave V data showed no statistically significant effect of group on either latency ($F(1, 24) = 3.25$, $p = 0.084$) or amplitude ($F(1, 24) = 0.33$, $p = 0.573$). The within-subject effect of stimulus intensity was significant for both latency ($F(2, 48) = 151.62$, $p < 0.001$) and amplitude ($F(2, 48) = 4.87$, $p < 0.05$), latency decreasing and amplitude increasing with increasing stimulus intensity. No effects of the group × intensity interaction were significant.

Table 2: Data for non-retarded and DS groups at 45 dB SL, with group × sex ANOVA results for group effect (means ± SD). Latencies and inter-peak intervals are in ms and amplitudes in μV.

Variable	Non-retarded	Down's syndrome	ANOVA F	df
Lat. III	4.36 ± 0.34	3.87 ± 0.21	15.64	1, 20**
Lat. V	6.14 ± 0.32	5.83 ± 0.42	4.76	1, 22*
Amp. V	0.42 ± 0.18	0.39 ± 0.12	0.27	1, 22

* $p < 0.05$; ** $p < 0.01$.

Table 3: Data for non-retarded and DS groups at 25 dB SL, with group × sex ANOVA results for group effect (means ± SD). Latencies and inter-peak intervals are in ms and amplitudes in μV.

Variable	Non-retarded	Down's syndrome	ANOVA F	df
Lat. V	6.78 ± 0.36	6.72 ± 0.61	0.13	1, 22
Amp. V	0.38 ± 0.15	0.31 ± 0.20	1.04	1, 22

Discussion: Folsom *et al.* [1] and Squires *et al.* [7] reported that subjects with Down's syndrome had shorter wave V latencies than non-retarded subjects for stimuli louder than 40 dB nHL. Widen *et al.* [10] reported a level of 60 dB nHL for this difference, and in data shown by Squires *et al.* [9] (specifically in their Figure 3), we can see that subjects with Down's syndrome had shorter wave V latencies than non-retarded subjects only at 75 dB nHL.

We believe that the fundamental cause of these discrepancies is that DS subjects frequently suffer auditory deficiencies [1, 5, 10]. Most papers in this field either do not state the auditory thresholds of the DS subjects studied [2, 3, 7, 8], or mention deficits differing in kind or intensity from one study to another [5, 9, 10]. It seems likely, therefore, that the difference between the auditory thresholds of DS and non-retarded groups has varied from study to study. This would naturally lead to discrepancies among the results of studies measuring stimulus intensity on the dB nHL scale, which does not adequately reflect the intensity perceived by the subject with hearing deficit.

We suggest that when SAEPs are recorded as a means of investigating neurofunctional deficits among groups prone to hearing deficiencies, the auditory threshold of each subject should be determined and the dB SL scale used. Adherence to this practice would allow meaningful inter-study comparison in terms of perceived intensity.

ANOVA showed that the latency of wave V (group × sex) was significantly shorter in DS than in non-retarded subjects at intensities of 45 and 65 dB SL, and practically the same in the two groups at 25 dB SL. The fact that both groups exhibited the same wave V latency at 25 dB SL is probably responsible for the intergroup difference failing to rank as statistically significant in the repeated measures analysis (group × intensity).

The DS group also exhibited a shorter wave III latency than the non-retarded group at 45 and 65 dB SL. Since the two groups did not differ significantly as regards the latency of wave I at 65 dB SL, the I–III and I–V interpeak intervals at this intensity were shorter among DS than non-retarded subjects. The only amplitude to differ significantly between the two groups was that of wave III, which for stimuli of 65 dB SL was smaller among DS than non-retarded subjects. This result is in keeping with those of other authors [2, 3, 10].

The suggestion [2, 7] that the abnormal SAEPs of DS subjects may be due to the smaller size of their brainstem [11] seems extremely unlikely. Normal children develop the same latencies as adults at a stage when they still have a smaller brainstem [2], and brainstem size is not correlated with the I–V interval [7]. Besides, brainstem size differences cannot explain why latencies are only shorter in DS subjects for stimulus intensities above a certain level.

Another suggestion, that the abnormalities are due to the high-frequency hearing loss of DS subjects [10], seems to be ruled out by the fact that the I–V interpeak interval is shorter in these individuals regardless of whether they have normal or deficient hearing [9]. In view of the above, we believe that the shorter latencies of waves III and V for stimulus intensities of 45 dB SL or greater, and the smaller wave III amplitude and I–III and I–V intervals at 65 dB SL, constitute a set of neurofunctional abnormalities inherent to DS.

As a working hypothesis, it seems possible that this abnormality may be due to the balance between excitation and inhibition being tilted towards greater net excitation. Perhaps this is a result of neurochemical and neuroanatomical alterations in the pons and the mesencephalon, where the chief neural generators of waves III and V are thought to be located [12].

1. Folsom, R.C., Widen, J.E. and Wilson, W.R. 1983. *Arch. Otolaryngol.*, **109**, 607–610
2. Galbraith, G.C., Squires, N., Altair, D. *et al.* 1977. In: Begleiter, H. (ed.). *Evoked Brain Potential and Behavior*, pp. 229–245. Plenum Press, New York
3. Galbraith, G., Aine, C., Squires, N. *et al.* 1983. *Am. J. Ment. Def.*, **87**, 551–557
4. Hattori, H., Murata, R., Matsuoka, O. *et al.* 1986. *No to Hattatsu*, **18**, 21–28
5. Kaga, K. and Marsh, R.R. 1986. *Int. J. Pediatr. Otorhinol.*, **11**, 29–38
6. Miyanomae, Y., Yoshida, A., Nomoto, N. *et al.* 1987. *No to Hattatsu*, **19**, 22–28
7. Squires, N., Aine, C., Buchwald, J. *et al.* 1980. *Electroenceph. Clin. Neurophysiol.*, **50**, 172–185
8. Squires, N., Buchwald, J., Liley, F. *et al.* 1982. In: Courjon, J., Mauguiere, F. and Revol, M. (eds), *Clinical Applications of Evoked Potentials in Neurology*, pp. 233–240. Raven Press, New York
9. Squires, N., Ollo, C. and Jordan, R. 1986. *Ear and Hearing*, **7**, 83–92
10. Widen, J.E., Folsom, R.C., Thompson, G. *et al.* 1987. *Am. J. Ment. Def. Res.*, **91**, 472–479
11. Crome, L., Cowie, V. and Slater, E. 1966. *J. Ment. Def. Res.*, **10**, 69–72
12. Legatt, A.D., Arezzo, J.C. and Vaughan, H.G. 1988. *Neurol. Clin.*, **6**, 681–705

We thank the Special Education School (La Barcia) and the Clinical Electroencephalography Service of the Hospital Xeral de Galica (Santiago de Compostela) for facilities and assistance.

Reprint requests to: Dr Montserrat Zurron, Departamento de Psicoloxía Clinica e Psicobioloxía, Universidade de Santiago de Compostela, Campus Universitario, 15705 Santiago de Compostela, A Coruña, Spain.

Paper received: 27th June, 1992.