Delays in brainstem auditory evoked potentials at ages 7 and 14 years due to prenatal methylmercury exposure

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Introduction and Objectives Delayed evoked potential latencies have been documented in methylmercury poisoning, and latencies of brainstem auditory evoked potentials (BAEP) have shown significant associations with developmental methylmercury exposure in two different fishing populations. This neurophysiological response requires only minimal cooperation by the subject and is unlikely to be affected by socioeconomic or cultural differences. The long-term validity of this indicator of methylmercury neurotoxicity in children was therefore studied.
Methods We examined a birth cohort of 1022 Faroese children born in 1986-1987 at ages 7 years and 14 years using a 4-channel electromyograph (Medelec Saphire-4ME) for assessment of BAEP latencies. Click signals were presented to the right ear through earphones at a rate of first 20 Hz, then 40 Hz. At age 7 years, the responses were averaged 1,024 times after amplification and filtration, with one replication at each condition. Twice as many responses were used at age 14 years to obtain better precision. The peak I latency showed greater imprecision than peaks III and V. Thus, although interpeak latencies I-III and I-V would be preferable to avoid interference from inner ear disease, we also used the total peak III and peak V latencies as outcome parameters in the present study. Regression coefficients and partial correlation coefficients were calculated using the cord-blood mercury concentration as the dose parameter. The distribution of the mercury results was skewed, and an improved fit of the dose-effect association was obtained using logarithmically transformed mercury data. Sex and age were included as potential confounders, although they had only a small effect on the results.
Results A total of 818 children participated in both examinations. Between the ages of 7 and 14 years, BAEP latencies decreased by 0.21-0.25 ms. Although almost entirely due to the greater delays in peak I at age 7 years, the difference did not disappear after exclusion of children with middle ear infection at that age. Correlation coefficients between the results at the two occasions were 0.39-0.55 for peaks III and V, but only 0.20-0.30 for peak I at the two frequencies. For all BAEP outcomes, girls had shorter latencies than boys at both examinations. Methylmercury exposure showed very similar associations with the latencies recorded at age 14 years as had already been reported for the results at age 7, peaks I and V at both 20 and 40 Hz being significantly associated with mercury. However, mercury showed associations in the same direction also with peak I latencies, and the correlations with interpeak latencies I-III and I-V, though in the same direction, were no longer statistically significant.
Conclusions These results support the notion that developmental methylmercury neurotoxicity causes permanent changes and that methylmercury may affect nerve transmission throughout the auditory system.