## Changes in ERPs

## Complex tones

<u>Before the ADT</u>: General ERP waveforms for complex tones did show that the basic acoustic reactivity of the auditory cortex was normal (Fig. 4A). The exogenous ERP pattern, P100- N250, was equal with the control group of ten healthy children (for reference see Korpilahti & Lang 1994; Korpilahti 1996). An involuntary attentional switch, as reflected in P3a, was recorded in deviant tones. The pre-attentive auditory memory was evaluated with the MMN method. Topographic brain map (Figure 4A) did show two-phasic MMN reactions. The early mismatch negativity (eMMN, related with the frequency difference of the two stimuli) was starting from the left hemisphere (map 150–200 ms) and expanding to the right fronto-temporal area (map 200–250 ms). In healthy children the right hemisphere is more active in frequency difference detection than the left hemisphere. In Peter the late mismatch negativity, IMMN, was stronger than the eMMN and occurred more centrally (maps 500–550 ms, 550–600 ms). The latency of this component was quite slow for the age.

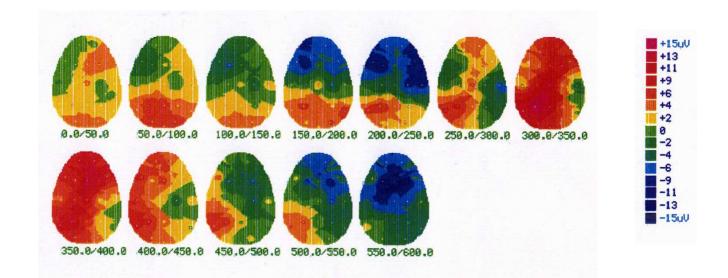


FIGURE 4 A. Before the ADT. An array of twelve brain maps showing the MMN (mismatch negativity; an ERP response reflecting the stimulus difference detection in a passive oddball paradigm) activation for complex tones from the stimulus onset to 600 ms.

Scale + 15  $\mu$ V to – 15  $\mu$ V.

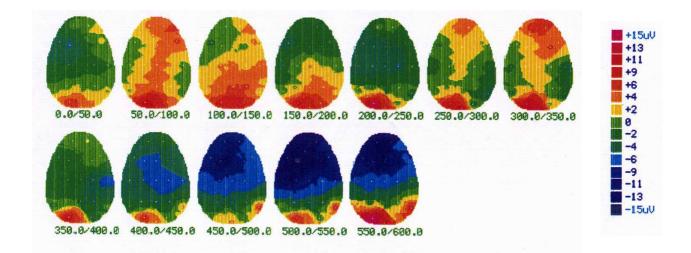


FIGURE 4 B. After the ADT. An array of twelve brain maps showing the MMN (mismatch negativity; an ERP response reflecting the stimulus difference detection in a passive oddball paradigm) activation for complex tones from the stimulus onset to 600 ms. Scale +  $15 \mu$ V to  $- 15 \mu$ V.

<u>After the ADT</u>: In the first research condition the MMN did show that the difference detection of complex tones was no more eliciting a involuntary attentional switch to the tone differences (see the positivity P3a in Figure 4A, 300–400 ms; missing in Figure 4B). After the ADT the late MMN was stronger and begun earlier (maps 350–600 ms) than in the first recording.

## Words

<u>Before the ADT</u>: In the word condition deviant words elicited a negative wave, starting from the left hemisphere. The neural activation was slowly developing over both hemispheres. An involuntary attentional switch, P3a, was recorded also in deviant words. The MMN pattern was two-phasic (Figure 5A) and both peaks had maximum at the centro-frontal area: the eMMN was peaking in normal timing (map 150–200 ms) and was followed by lMMN (map 300–350 ms). The amplitudes of these components were atypically low. In normal children we have reported a integrative time window for the word difference detection. In the case of Peter this summating processing, reflecting the lexical difference detection, was missing. Instead, Peter was processing single acoustical features inside words.

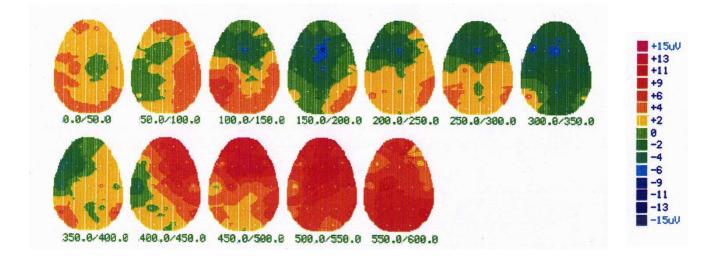


FIGURE 5 A) Before the ADT. An array of twelve brain maps showing the MMN activation for naturally spoken words from the stimulus onset to 600 ms.

<u>After the ADT:</u> In the word condition the change in MMN component was quite clear (Figure 5B). The integrative time-window was seen in the latencies of 300–600 ms after the stimulus onset. The maximum IMMN was recorded at the brain map of 400–450 ms, reminding the results of the control group. The left hemisphere was leading the difference detection of words. The auditory processing was no more based on fragmented acoustic information. This result is equal with the reference values in normal population of the same age.

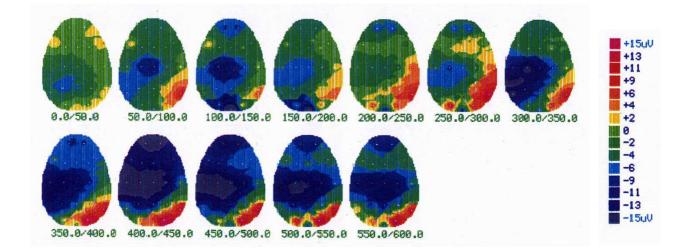


FIGURE 5 B) After the ADT. An array of twelve brain maps showing the MMN activation for naturally spoken words from the stimulus onset to 600 ms.

## Pseudo-words

<u>Before the ADT:</u> During the first recording Peter reacted to the auditory difference in pseudo-words (Figure 6A) in a more active way that to the word differences. In control group the MMN amplitudes for words and pseudo-words has been opposite to the results of Peter. In our subject the maximum MMN was elicited at the time window of 350–600 ms, and reached a very large distribution over the scalp. In normal children this component occurred at the latency of  $454 \pm 43$  ms and was much weaker  $-6.0 \pm 3.8 \,\mu\text{V}$  than in Peter's MMN recording.

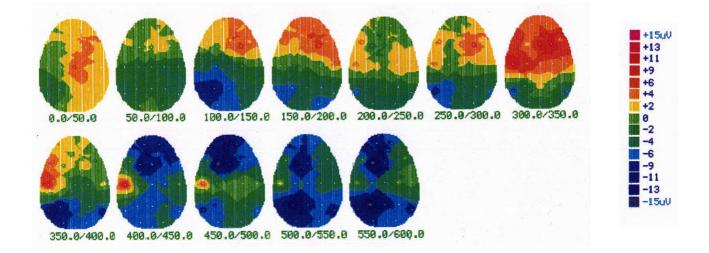


FIGURE 6 A. Before the ADT. An array of twelve brain maps showing the MMN activation for naturally spoken pseudo-words from the stimulus onset to 600 ms.

<u>After the ADT:</u> The neural activity for the auditory difference detection in pseudo-words (Figure 6B) was greatly faded after the ADT. There was a weak eMMN (map 250–300 ms) at the right hemisphere. The cerebellum activity was recorded and might be connected with the articulatory loop of the auditory memory. After the ADT rehabilitation Peter was pre-attentively reacting in a different way if the stimuli were words or pseudo-words. This change made the ERP results to remind more the reference group of healthy children.

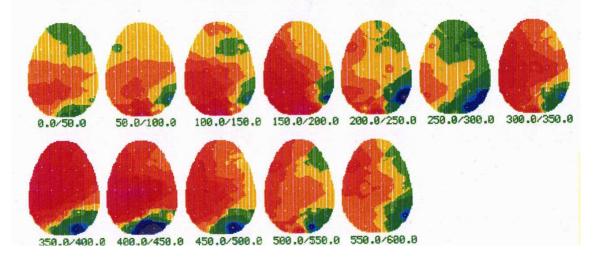


FIGURE 6 B. After the ADT. An array of twelve brain maps showing the MMN activation for naturally spoken pseudo-words from the stimulus onset to 600 ms.