

# Influences of syllabic compression on speech evoked potentials in Cochlear Implant users.

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## Background

Fitting of Cochlear Implants is usually based upon subjective statements of the patients about their auditory perception. Cortical Auditory Evoked Potentials (CAEP) are a well known method to get information about auditory perception without any cooperation of the subject. (Agung et al., 2006; Hyde, 1997; Jones et al., 1998; Korczak et al., 2005; Näätänen and Picton, 1987) In the past several studies have shown, that it is possible to elicit CAEP in cochlear implant users (Hoppe et al., 2001; Hoth 1998; Groenen et al., 2001). This paper describes speech evoked CAEP in cochlear implant listeners in association with a used acoustic pre-processing, the syllabic compression. (McDermott 2002) In particular, the influence of syllabic compression was investigated for two consonant-vowel-syllables differing in the voicing of the consonant.

## Methods

CAEP were recorded from ten cochlear implant listeners provided with a nucleus freedom implant. A sine burst and the natural syllables /ta/ and /da/ were used as stimuli for the CI implanted group, without and with activated syllabic compression. Additionally, CAEP to the same speech and tone stimuli were derived in five normal hearing subjects serving as a reference group. All stimuli were presented at 60 dB SPL.

## Results

The following significances are listed in Table 1. Figure 1 and 2 illustrate the findings as box-whisker charts. Therefore the left side shows the results for the CI implanted group and the right side shows the results for the normal hearing group. The middle line of each box shows the median of the pooled data and the lower and upper lines indicates the lower and upper quartile. Figure 1 shows the latencies of the N1 waves (upper plot) and of the P2 waves (lower plot). Figure 2 displays the belonging amplitudes.

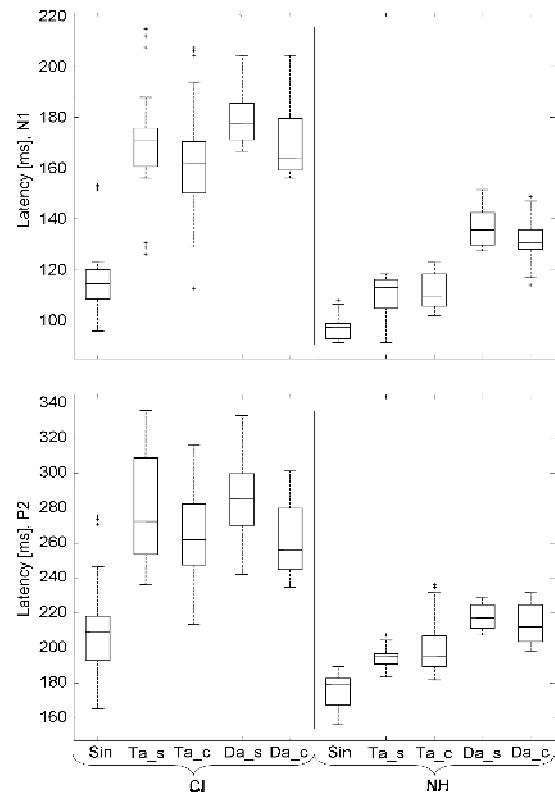


Figure 1. Shown are the N1 latencies (upper plot) and the P2 latencies (lower plot) of the potentials obtained from CI implanted listeners and from normal hearing subjects (NH).

Result 1) In all subjects CAEP could be reliably recorded and a clear N1-P2 complex was observed. In the CI group N1 and P2 latencies are significantly longer than those of the normal hearing group. While N1 amplitudes differ not significantly between the two groups, P2 amplitude is decreased in the cochlear implant group.

Result 2) In all subjects the sine burst elicits earlier N1 and P2 components and larger N1 amplitudes than the spoken syllables.

Result 3) The speech stimulus /ta/ elicits earlier N1 and P2 waves than the stimulus /da/ in both groups.

Result 4) When a syllabic compression is used latencies of both N1 and P2 and N1 – P2 interpeak amplitude decrease.

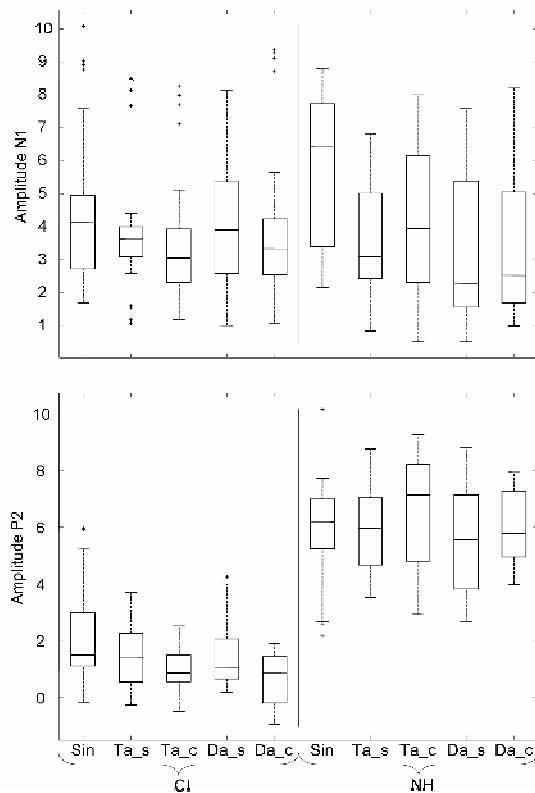


Figure 2. Shown are the N1 amplitudes (upper plot) and the P2 amplitudes (lower plot) of the potentials obtained from CI implanted listeners and from normal hearing subjects (NH).

## Discussion

Result 1) An observation of occurring activation artefacts showed that the delay caused by the processors processing time lies in the range between 11ms to 13 ms. This delay correlates with the longer latencies observed by the responses evoked by the sine burst, but it can't explain the much bigger differences found when the syllabic stimuli were presented. Furthermore the reason for this could be an effect of different perceived loudness, caused by the difference of how the nerve is stimulated. Maybe the presented stimuli were perceived on different gages even though the presented level was the same. But because of the small differences in N1 amplitude between the two groups, this is an unlikely explanation. Furthermore the reason can be a missing activation during the consonant part of the syllables, resulting from a stimulus level, which is lower than activation thresholds defined as map parameters. This can be examined by simulating the implant activation in respect to specific parameter of the map, used by the subject during stimulation. The results of such simulations are electrodiagrams, which for one subject are shown in figures 3, the simulation without syllabic compression and figure 4, the simulation with used syllabic compression. In these figures the whole activation for all 22 electrodes of the electrode array is shown. Electrode 22 is the electrode which represents the lowest frequency band and electrode 1 is the electrode for the frequency band includ-

ing the highest possible frequencies. The length of each line indicates the energy of activation.

In both figures it can be seen, that the consonant yields to an activation, so the differences in N1 and P2 latency between the two groups can not be an effect of missing stimulation.

We assume that cortical processes are responsible for the found delay.

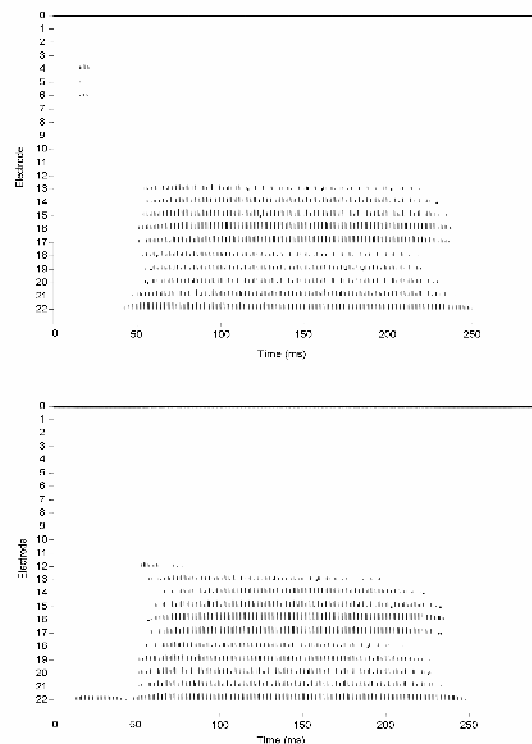


Figure 3. Shown are the electrodiagrams of subject S4 evoked by the syllable /ta/ (upper plot) and by the syllable /da/ (lower plot) while the standard setting was used.

Result 2) The sine burst was on the onset and offset manipulated with a cosine ramp, to avoid spectral splatter. But anyhow the electrodiagrams showed spread activation over eleven electrodes, while after a few milliseconds only three electrodes were active. This spread activation yields to the earlier waves and the bigger amplitudes.

Result 3) The shorter N1 and P2 latencies in this case can be explained by the different time-frequency properties of the voiced and unvoiced consonants of the two stimuli, respectively. These differences yield to different CI activation patterns, shown in figure 3. The consonant part of the stimuli /da/ only activates one electrode nearest the apex. The consonant of the syllable /ta/ activates 3 electrodes more basal instead. The amount of activated electrodes and the place of activation yields to the observed shorter latencies.

Result 4) This finding can again be explained with the electrodiagrams in figure 4. A syllabic compression yields to an activation of additional electrodes and it raises the energy of each activated electrode. This

again yields to summation effects and to shorter N1 and P2 latencies.

## Conclusions

In summary it can be said that CAEP can be reliably recorded on CI users and a syllabic compression yields to significant changes in CAEP. These changes

can be explained by a more spread activation along the electrode array as a result of the enhanced perceived level. The study demonstrates that speech evoked CAEP are a good candidate for objective evaluation of acoustic pre-processing algorithms. Furthermore, the study indicates different speech processing in CI listeners and in normal hearing listeners.

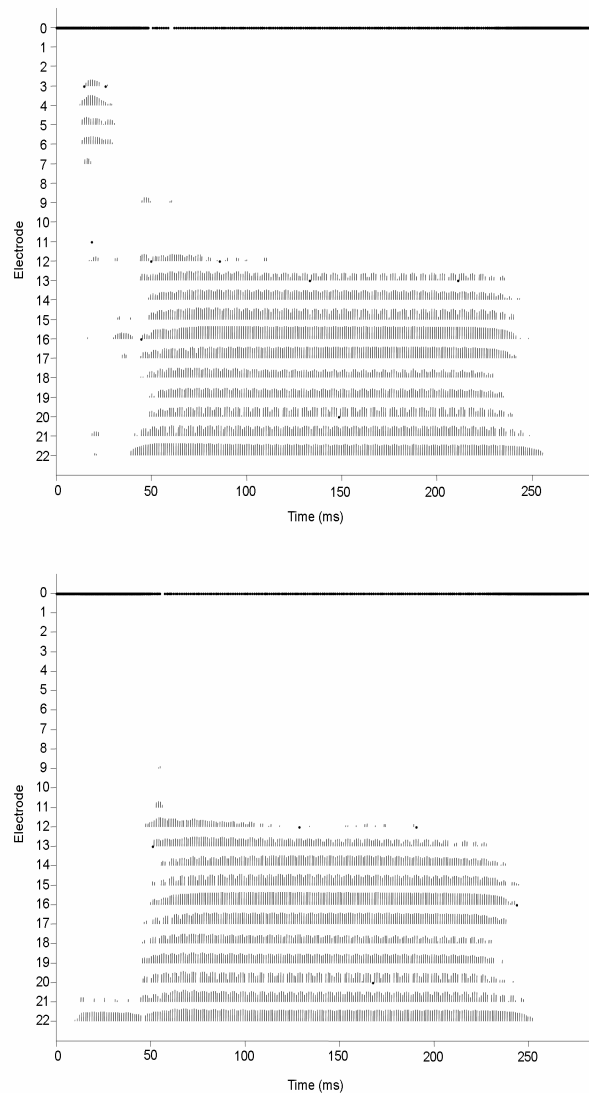


Figure 4. Shown are the electroencephalograms of subject S4 evoked by the stimulus /ta/ (upper plot) and by the stimulus /da/ (lower plot) while the syllabic compression was active

Significance values for differences in latencies and amplitudes inside of the four tested stimulus pairs

Stimulus pair	CI implanted subjects						Normal hearing subjects					
	N1 tency	la-N1 tude	ampli-P2 latency	P2 tude	ampli-N1 amplitude	– P2 amplitude	N1 tency	la-N1 tude	ampli-P2 tency	la-P2 tude	ampli-N1 amplitude	– P2 amplitude
Sin / Ta <sub>s</sub>	p < 1.7e-7	p < 4.5e-4	p < 8e-7	p < 7.6e-4	p < 8.6e-5		p < 2.9e-4	p < 1.9e-4	p < 8.8e-5	p < 0.88	p < 0.021	
Sin / Da <sub>s</sub>	p < 1.7e-7	p < 1.5e-4	p < 1.7e-7	p < 4.7e-5	p < 1.e-6		p < 8.8e-5	p < 1e-4	p < 8.8e-5	p < 0.85	p < 0.002	
Ta <sub>c</sub> / Ta <sub>s</sub>	p < 0.007	p < 0.002	p < 0.018	p < 0.0015	p < 9.9e-4		p < 0.41	p < 9e-3	p < 0.17	p < 0.041	p < 0.019	
Da <sub>c</sub> / Da <sub>s</sub>	p < 3.6e-7	p < 0.17	p < 1.7e-5	p < 0.003	p < 1.9e-5		p < 3.9e-4	p < 0.3	p < 2.9e-3	p < 0.12	p < 0.12	
Ta <sub>s</sub> / Da <sub>s</sub>	p < 0.003	p < 0.38	p < 0.78	p < 0.37	p < 0.39		p < 8.8e-5	p < 0.66	p < 8.9e-5	p < 0.52	p < 0.83	
Ta <sub>c</sub> / Da <sub>c</sub>	p < 0.046	p < 0.0042	p < 0.865	p < 0.28	p < 0.295		p < 8.6e-5	p < 0.08	p < 2.2e-4	p < 0.12	p < 0.044	

Table 1, shown are the significance values obtained via the paired both sided wilcoxon signed-rank test to show the differences in N1 and P2 latency and in N1, P2 and N1 – P2 inter peak amplitude, for both between the sine burst and the stimulus Ta<sub>s</sub> and between the sine burst and the stimulus Da<sub>s</sub>. Further for the pairs of stimuli Ta<sub>c</sub> and Ta<sub>s</sub>, Da<sub>c</sub> and Da<sub>s</sub>, Ta<sub>s</sub> and Da<sub>s</sub> and between Ta<sub>c</sub> and Da<sub>c</sub>

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