


Pfurtscheller, G., Spatiotemporal analysis of alpha frequency components with the ERD technique, Brain Topogr., 2 (1989b) 3–8.


References


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Response to M. Backonja and W. Howland

Electroencephalography has been used to determine whether the acquired data demonstrate temporal (time and frequency domain) and/or spatial (topographic) specificity of pain. Three studies have examined the encephalogram during painful tonic stimulation produced by the immersion of an arm in ice-cold water (Chen et al. 1989; Backonja et al. 1991), or the infusion of an algesic substance into muscle (Veerasarn and Stohler 1992).

The reported effects of tonic experimental pain on cortical power densities of various frequency bands can be summarized as follows:

There is agreement among all 3 studies with respect to the observed significant increase in cortical beta power density in pain which was explained to a large part by muscular effects according to our work. These effects are likely due to specific pain-related expressions produced by facial and scalp muscles in the close vicinity of the recording electrodes. Interestingly, these reactions are not limited to pain. In fact, remembering a previous painful episode causes similar effects in the cortical beta power density and the electromyogram. The particular spatial arrangement of the involved facial and scalp muscles, located directly under the recording electrodes favors the contamination of the electroencephalographic record.

The exciting findings of our colleagues of increased cortical delta, particularly in pain-sensitive subjects (Chen et al. 1989), or alpha power (Backonja et al. 1991) density during the immersion of a hand in ice-cold water could not be replicated in our study. How about a re-analysis to consolidate these findings?

Given our repeated-measures design, we estimated the required sample size for (a) a given significance level (P = 0.01), (b) power of the test (90%), (c) magnitude of the effect of pain (20%, 2-tailed) with (d) the measurement parameter having an either average (coefficient of variation: 0.23) or large variability (0.32) to be 13 or 19 subjects, respectively. Because we did not observe any significant increase in the delta power in pain in our total group of 19 subjects, we did not examine whether the subjects which needed higher infusion rates differed from the others. In addition, we considered the comparison of the outcome of the cold pressor test with the result of a given subject’s required infusion rate to maintain pain at a level of greater than 5 on a 10-point scale as questionable. For this very reason, we did not investigate the response in a subgroup of pain-sensitive or pain-tolerant subjects.

Pooling of the theta and alpha bands raises a valid point. In our experiment, it was necessitated by the equipment/software constraints of the topographic brain mapping system, CADWELL S32 (Cadwell Laboratories, Kennewick, WA 99336). Because our main focus was to examine the extent to which the beta power density is
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**Stimulus** | Cold pressor test (upper extremity) | Cold pressor test (upper extremity) | Muscle pain and sham pain (face)
**EEG delta power** | Significant increase, particularly in pain-sensitive subjects | Not significant | Not significant
**EEG theta power** | Not significant | Significant increase in ipsilateral frontal electrodes | Not significant with alpha and theta bands (3.5–13 Hz) pooled
**EEG alpha power** | Not significant | Specific temporal low (8–10 Hz) and high (10–12 Hz) alpha response to pain | Not significant with alpha and theta bands (3.5–13 Hz) pooled
**EEG beta power** | Significant increase bilaterally in frontal and parietal regions | Significant increase bilaterally in frontal and parietal regions | Significant increase bilaterally in frontal and parietal regions
**EMG** | Response in high beta (26–30 Hz) band suggested contamination | Significant relationship between beta (13–35 Hz) and EMG signals (35–100 Hz) demonstrated by Pearson’s product moment correlation

dependent of the contamination of muscle effects, pooling of the *alpha* and *theta* bands was needed and regarded as the most acceptable compromise. Instead, we added the frequency band of 35–100 Hz to our data collection protocol.

Because uneven pain intensity scores might affect the variables under examination, we chose to maintain pain by means of the continuous infusion of saline into muscle (Stohler et al. 1992). Artifact-free epochs were selected during times when the subject rated the pain intensity greater than 5 on the 10-point scale, starting at 90–150 sec following the onset of stimulus delivery and as soon as a relatively steady-state condition was reached. As far as the work of the colleagues from Wisconsin-Madison is concerned, our sampling would favor the discovery of their finding of *alpha* augmentation because we did not consider the initial phase following pain initiation during which Backonja et al. (1991) reported *alpha*-blocking. However, we could not observe an increase in the combined cortical *alpha* and *theta* power density. Specifically, such an increase was not observed in the ipsilateral frontal leads for which both significant *alpha* and *theta* increases were reported (Backonja et al. 1991).

Finally, the cerebral representation of pain of the upper extremity and the face may account for possible differences between our studies as well.

References


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Comments on P.D. Wall, PAIN, 51 (1992) 1–3

Dr. Wall’s recent editorial comments on the placebo effect (Pain, 51 (1992) 1–3) are to be praised for bringing further attention to this misunderstood and somewhat notorious topic. However, I wish to highlight some ways in which his comments may foster the undeservedly notorious reputation of this ubiquitous process.

The negative connotations associated with the placebo effect arise largely because the effect appears to contradict a fundamental tenet of the biomedical model of reality – namely that psychological events cannot cause physical and anatomical changes. As he implies in his 2nd and 3rd reasons (if I may overstate the case somewhat), the placebo effect seems implausible because it appears unrelated to the ‘true’ effects of any medical therapy. In other words, only objective, organic, material reality is ‘real’, and any subjective process is inherently less valid or worthy of our attention–placebo is a nuisance variable, and the whole realm of psychological processes creates only nuisance artefacts.

However, when we use a biopsychosocial model to understand medical phenomena, rather than a reductionistic biomedical model, then placebo response becomes not only more understandable, but also more desirable. In other words, all great medical healers possessed the ability to elicit, whether by conscious design or not, the