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Functional Features of Nociceptive-Induced Suppression of Alpha Band Electroencephalographic Oscillations

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Abstract: **Nociceptive stimuli can induce a transient suppression of electroencephalographic oscillations in the alpha frequency band (ie, alpha event-related desynchronization, α-ERD). Here we investigated whether α-ERD could be functionally distinguished in 2 temporally and spatially segregated subcomponents as suggested by previous studies. In addition, we tested whether the degree of dependence of nociceptive-induced α-ERD magnitude on the prestimulus α-power would have been larger than the degree of dependence on the poststimulus α-power. Our findings confirmed the dissociation between a sensory-related α-ERD maximally distributed over contralateral central electrodes, and a task-related α-ERD (possibly affected by motor-related activity), maximally distributed at posterior parietal and occipital electrodes. The cortical sources of these activities were estimated to be located at the level of sensorimotor and bilateral occipital cortices, respectively. Importantly, the time course of the α-ERD revealed that functional segregation emerged only at late latencies (400 to 750 ms) whereas topographic similarity was observed at earlier latencies (250 to 350 ms). Furthermore, the nociceptive-induced α-ERD magnitude was significantly more dependent on prestimulus than poststimulus α-power. Altogether these findings provide direct evidence that the nociceptive-induced α-ERD reflects the summation of sensory-related and task-related cortical processes, and that prestimulus fluctuations can remarkably influence the non-phase-locked nociceptive α-ERD.**

Perspective: ***Present results extend the functional understanding of α-oscillation suppression during pain perception and demonstrate the influence of prestimulus variability on this cortical phenomenon. This work has the potential to guide pain clinicians in a more accurate interpretation on physiological and psychological modulations of α-oscillations.***

Key words: *Pain, intra-epidermal stimulation, event-related desynchronization, α-oscillation, prestimulus α-power, electroencephalography.*

Sensory stimuli can elicit transient changes in ongoing electroencephalography (EEG), which can be distinguished between phase-locked event-related potentials (ERPs) and non-phase-locked modulations of spontaneous EEG oscillations.[45,54] These modulations are characterized by either transient enhancement (event-related synchronization, ERS) or transient suppression (event-related desynchronization, ERD) of the EEG power.[43,56] The functional significance of ERS and ERD differs according to the frequency band within which they occur. For example, ERD in the alpha band (α-ERD, frequencies ranging from 8 to 13 Hz) has been hypothesized to reflect cortical activation or disinhibition,[43,45,54,67] while ERS in the gamma band (frequencies ranging from 30 to 100 Hz) has been

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hypothesized to play a crucial role in cortical integration and perception.[16,38,62,63,68]

Among the modulations of EEG oscillations, the α-ERD has been observed not only during the elaboration of stimuli belonging to different sensory modalities (eg, visual and auditory; ie, sensory-related α-ERD),[9,48,60,67] but also during various mental tasks (eg, working memory processes; ie, task-related α-ERD).[19,28,30,64] In particular, some authors recently hinted to the coexistence of both sensory-related α-ERD and task-related α-ERD during pain.[43,55,56] These scholars reported that nociceptive-induced α-ERD over the contralateral sensorimotor cortex[1,51] may reflect the exogenous activation of the primary somatosensory cortex[55] and its contributions to pain processing.[51,58] In contrast, other authors reported a widespread suppression of α-oscillation at the level of posterior parietal and occipital regions following nociceptive stimulation,[43,46,56] which was hypothesized to reflect endogenous task-related cortical processing.[23] It is important to note that some of the reported modulations[1,4,15,56,60] have been expressed as a function of the poststimulus α-power as compared to the prestimulus α-power (ie, baseline correction procedure). This procedure is classically accepted (in fact, desired) to provide a theoretically noise-free reference cortical signal. Crucially, this assumption does not always hold true, and it has actually been reported that the α-power preceding either sensory stimulation or cognitive operations can dramatically influence the subsequent neural responses (eg, α-ERD),[39,55,60] thus reflecting the fluctuations in subjects' mental states (eg, attention and vigilance).[15,18,33]

Here we investigated these multiple features of the nociceptive-induced α-ERD (sensory-related, task-related, and state-dependent) in an attempt to provide a functional characterization of their distinct electrophysiological features. A 64-channel EEG set-up was used to record event-related brain responses during a classical oddball task, whereby subjects were required to detect and respond to low-probability nociceptive target events intermingled in a series of high-probability nociceptive nontarget events. This task allowed us to explore whether the posterior parietal and occipital α-ERD would have been preferentially associated with the top-down endogenous voluntary orienting of attention toward task-relevant nociceptive stimuli (ie, targets) while the sensorimotor α-ERD would have been rather associated with the bottom-up exogenous involuntary orienting of attention to salient nociceptive stimuli, regardless of their task relevance (ie, nontarget). In addition, we tested the hypothesis that the degree of dependence of nociceptive-induced α-ERD magnitude on the prestimulus α-power would have been larger than the degree of dependence on the poststimulus α-power, regardless of the experimental condition.

Methods

Subjects

Eighteen healthy right-handed volunteers (9 females) with a mean age of 22 years (range, 19–29 years) participated in the study. All subjects gave written informed consent before participation, and the study was approved by the Institutional Review Board of the University of Hong Kong/Hospital Authority Hong Kong West Cluster.

Nociceptive Stimulation

Intra-epidermal electrical stimuli were constant-current square electric pulses of .5-ms duration delivered through a stainless steel concentric bipolar needle electrode consisting of a needle cathode (length: .1 mm; diameter: .2 mm) surrounded by a cylindrical anode (diameter: 1.4 mm).[25,26] The stimuli were delivered to the medial and lateral side of the left hand dorsum, and the stimulus intensity was twice the individual perceptual threshold, previously proved to be able to preferentially activate the Aδ nonciceptive fibers without coactivation of the fast-conducting Aβ fibers.[44]

Experimental Procedure

Subjects were seated in a comfortable chair in a lighted, shielded room and were instructed to relax and equally attend all the sensory stimuli prior to experiment start. Nociceptive stimuli were administered in 2 separated blocks. In 1 block, target and nontarget stimuli were randomly delivered to the medial and lateral side of the left hand dorsum according to a probability rate of 1:4. In the other block, the stimulus sites of target and nontarget stimuli were reversed, and they were randomly presented with the same probability rate used in the previous block (1:4). Each block consisted of 200 stimuli, and interstimulus intervals were randomly varied between 2,500 and 3,000 ms. The subjects were required to respond as fast and accurately as possible to the predefined target stimuli by pressing the response button upon their detection, using their right index finger. The order of the blocks was counterbalanced across subjects. Prior to data collection in each block, the subjects were repeatedly presented with 20 stimuli to familiarize them with the task.

Behavioral Data Analysis

In the target condition, trials without any response and those with reaction times shorter than 200 ms or longer than 1,000 ms were considered as error trials,[7] while in the nontarget condition, trials with any response were considered as error trials. The percentage of error trials across all trials (both target and nontarget conditions) was defined as error rate. As a result, 71 ± 3 (out of 80) and 304 ± 9 (out of 320) trials were obtained for the target and nontarget conditions, respectively. To rule out any statistical bias due to different size of trials in the 2 conditions, trials with the same number as in the target condition were randomly selected from the nontarget condition in each subject. Results are reported as mean ± standard error of mean (SEM). Statistical differences were considered significant at $P < .05$.

EEG Recording

The EEG data were recorded using a 64-channel Brain Products system (Brain Products GmbH, Munich, Germany; pass band, .01–100 Hz; sampling rate, 500 Hz) using a standard EEG cap based on the extended 10–20 system. The left mastoid was used as the reference channel, and all channel impedances were kept lower than 10 kΩ. To monitor ocular movements and eye blinks, electro-oculographic signals were simultaneously recorded from 4 surface electrodes: