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Fasttrack article

Pain perception, hypnosis and 40 Hz oscillations

Rodney J. Croft[a,b], John D. Williams[a,c], Corinna Haenschel[a], John H. Gruzelier[a,*]

[a]*Department of Cognitive Neuroscience and Behaviour, Imperial College London, Medical Faculty, St. Dunstan's Road, London W68 RF, UK*

[b]*Centre for Neuropsychopharmacology, Swinburne University of Technology, Hawthorn 3122, Australia*

[c]*School of Health and Social Sciences, Coventry University, Coventry, UK*

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Abstract

A number of brain regions are associated with the subjective experience of pain. This study adds to our understanding of the neural mechanisms involved in pain by considering the relation between cortical oscillations in response to pain, with and without hypnosis and hypnotic analgesia, and the subjective experience of pain. Thirty-three subjects' neural responses (EEG) were measured during the 40–540 ms period following phasic electrical stimulations to the right hand, under control and hypnosis conditions. Resultant FFT amplitudes for frequencies ranging from 8 to 100 Hz were computed. These were grouped into 7 scalp topographies, and for each frequency, relations between these topographies and pain ratings, performance and stimulus intensity measures were assessed. Gamma activity (32–100 Hz) over prefrontal scalp sites predicted subject pain ratings in the control condition ($r=0.50$, $P=0.004$), and no other frequency/topography combination did. This relation was present in both high and low hypnotisable subjects and was independent of performance and stimulus intensity measures. This relation was unchanged by hypnosis in the low hypnotisable subjects but was not present in the highs during hypnosis, suggesting that hypnosis interferes with this pain/gamma relation. This study provides evidence for the role of gamma oscillations in the subjective experience of pain. Further, it is in keeping with the view that hypnosis involves the dissociation of prefrontal cortex from other neural functions.

Keywords: EEG; Gamma oscillations; 40 Hz; Pain; Subjective experience; Hypnosis

1. Introduction

Although the electroencephalograph (EEG) has proven a powerful tool in the study of pain due to its good temporal resolution, its success in elucidating pain has been largely restricted to evoked potential designs (e.g. Bromm and Lorenz, 1998). With regard to resting EEG the most consistent findings relating to changes in power spectra have been pain-related increases in beta activity (Backonja et al., 1991; Veerasarn and Stohler, 1992; Chen and Rappelsberger, 1994) and pain-related decreases in alpha activity (Backonja et al., 1991; Chen and Rappelsberger, 1994). However, due to the non-specificity of these measures as reported,

*Corresponding author. Tel.: +44-20-8846-7246; fax.: +44-20-8846-1670.

E-mail address: j.gruzelier@ic.ac.uk (J.H. Gruzelier).

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it cannot be determined whether these changes relate to the pain or to other cognitive processes related to the pain protocols. For example, recent research suggests that alpha may relate to inhibitory networks (Klimesch et al., 2000) and/or changes in attention (Cooper et al., in press), which suggests that pain-related alpha changes could represent any one of a number of functional changes related to the pain task, perhaps quite removed from the pain processing itself.

In order to help clarify the relation between pain and spectral power the present study measured EEG activity while subjects engaged in a somatosensory oddball task where the somatosensory stimuli were painful electrical stimulations to the finger. Measures of performance and perceived stimulus intensity were obtained while allowing for control of attention and so overcoming some of the ambiguity in the interpretation of EEG responses. Measures of spectral power were obtained from epochs following sensory processing (500 ms-epochs, beginning 500 ms after phasic pain stimulations). The use of brief epochs though decreasing the accuracy of slow frequencies (such as delta where only 1 cycle fits within the recording window), enables accurate measures of higher frequencies and minimises variance related to other cognitive tasks occurring during the recording. Thus only high frequencies were considered (8–100 Hz). The study also employed a non-pharmacological pain manipulation method (hypnosis) to determine whether this affects any pain-related EEG changes that may be found. This method has been shown to attenuate the subjective experience of pain in highly hypnotisable subjects and avoids many of the non-specific effects of pharmacological agents (Rainville et al., 1997).

This method also allowed us to further investigate the theory that hypnosis involves, inter alia, an alteration of anterior brain functions. This has been the implication of theoretical considerations including a neuropsychological translation of the induction process including the suspension of reality testing and the handing over of the planning of behaviour to the hypnotist (Gruzelier, 1990, 1998), application to the hypnotic process of cognitive models of high order executive and attentional systems (Crawford and Gruzelier, 1992; Woody and Bowers, 1994; Woody and Farvolden, 1998; Kaiser et al., 1997; Gruzelier, 1998; Oakley, 1999) and a range of empirical evidence with measures including neuropsychological tests of ideational fluency, attention and executive functions (Gruzelier and Warren, 1993; Woody and Farvolden, 1998; Kallio et al., 2001), cortical evoked potentials including the N100 difference wave (Gruzelier, 1998), error related positivity (Kaiser et al., 1997), and EEG coherence (Kaiser in Gruzelier, 1998).

2. Materials and methods

2.1. Subjects

One hundred and seventy-five volunteer medical students were initially assessed using the Harvard Group Scale of Hypnotic Susceptibility, Form A (Shor and Orne, 1962). From these, 33 right-handed subjects (17 male, 16 female) aged 17–37 (Mean = 22.03, S.D. = 3.4) were selected on the basis of their hypnotic susceptibility and invited to participate in the main laboratory based experiment. These subjects were paid GBP£10 and consisted of 17 highly susceptible subjects (scores 8–12) and 16 low in hypnotic susceptibility (scores 0–4). The low hypnotically susceptible subjects were included as a control group, as research indicates that they do not engage in the hypnotic condition per se. Two subjects' data were omitted as they were multivariate behavioural outliers ($Z = 2.7$ and -3.7). Subjects gave written informed consent in line with Ethics Committee guidelines and were free to withdraw from the study at any time without penalty.

2.2. Stimuli

Pain stimuli were administered to the right index finger using a Digitimer Constant Current Stimulator, model DS7A, following light abrasion of the finger (cathode—distal phalanx; anode—middle phalanx). Standard stimuli comprised single square-wave electrical pulses of 1.6 ms duration (rise/fall time of 20 μs), and target stimuli comprised three consecutive standard stimuli (i.e. total 4.8 ms duration). Five hundred and fifty stimuli