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Fasttrack article

Pain perception, hypnosis and 40 Hz oscillations

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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,

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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

were presented pseudo-randomly, of which 20% were targets.

2.3. Procedure

Subjects completed a consent form, were fitted with EEG recording apparatus and then were familiarised with the pain inducing stimuli. Sensory threshold and pain tolerance levels were assessed using an ascending method of limits procedure. Having been informed of the necessity to experience stimuli that were painful but bearable, subjects were asked to adjust the electric stimulator to match such a point. This point, divided by the subject's threshold level, is referred to as the 'stimulus intensity'. To minimise variation due to differing strategies of coping with the pain (e.g. diverting attention), a somatosensory oddball task was employed to direct subjects' attention towards the painful stimuli. That is, subjects were instructed to press a trigger as quickly as possible (with the left hand) when a target was detected.

Each pain stimulation session lasted 10 min after which time the subjects were asked to rate the level of pain that they experienced during that session (i.e. an overall rating incorporating both standard and target stimuli). Pain ratings were based on a 10-point Likert scale where '0' represented 'no pain', '5' represented 'moderate pain' and '10' represented 'unbearable pain'. 'Reaction time to correct targets' and 'number of correct targets' were tallied to provide objective measures of performance. These measures allowed the dissociation of relations between pain and the EEG, and performance and the EEG, in order to exclude effects attributable to general cognitive processing.

There were control, hypnosis and hypnotic analgesia conditions which were presented in a random order, counterbalanced across subjects. The control condition involved the somatosensory oddball task only. The hypnosis condition involved the oddball task following a well-established induction procedure (i.e. eye fixation, systematic muscle relaxation, counting down from '20' to '1' and a further 'deepening' technique using guided imagery). The hypnotic-analgesia condition involved the oddball task following a period of guided imagery where it was suggested that they were lying on a warm sandy beach and that they start to bury their right hand deep in the sand, that their hand and arm were becoming increasingly numb and that they were losing sensation in their finger (right index), that this loss of sensation would increase during the course of the following condition and that they would increasingly have difficulty in detecting the target stimuli. Importantly, in all conditions subjects were instructed to focus their attention on their right index finger and press a response key with their left hand if they detected a target stimulus. An Experimental Hypnosis Scale similar to ones used previously (e.g. Gruzelier et al., 1984; Gruzelier and Brow, 1985; McCormack and Gruzelier, 1993) were employed during the hypnosis sessions to validate hypnotic state and group assignment (Gruzelier, 2000).

2.4. Data collection and manipulation

EEG data were collected from 28 scalp sites (Fp1, Fp2, Fz, F3, F4, F7, F8, Cz, C3, C4, Pz, P3, P4, T3, T4, T5, T6, Oz, O1, O2, FTC1, FTC2, TCP1, TCP2, CP1, CP2, PO1, PO2). Data were continuously digitised at 500 Hz, with a 0.01–100 Hz bandpass (24 dB/octave roll-off). The left ear served as reference, and vertical and horizontal electrooculographs (EOG) recorded eye movements. Data were epoched 40–540 ms post stimulus and rejected if either EOG channel had a maximum displacement of greater than 50 μV relative to baseline. The first 40 ms period was excluded in order to remove any early contamination of the EEG signal caused by the electric stimulator. For each of the conditions, mean spectral amplitude was computed from the remaining epochs using fast Fourier transformation (FFT; a composite measure of both phase locked and non-phase locked components of the EEG), for the following frequency ranges-alpha: 8–12 Hz; beta1: 12.5–20 Hz; beta2: 20.5–30 Hz; gamma: 32–100 Hz. Data were transformed to normal using natural log and grouped into prefrontal (FP1, FP2), left fronto-temporal (F7, F3, FTC1, T3), right fronto-temporal (F8, F4, FTC2, T4), midline (Fz, Cz, Pz), left parieto-temporal (C3, P3, CP1, TCP1, T5), right parieto-temporal (C4, P4, CP2, TCP2,

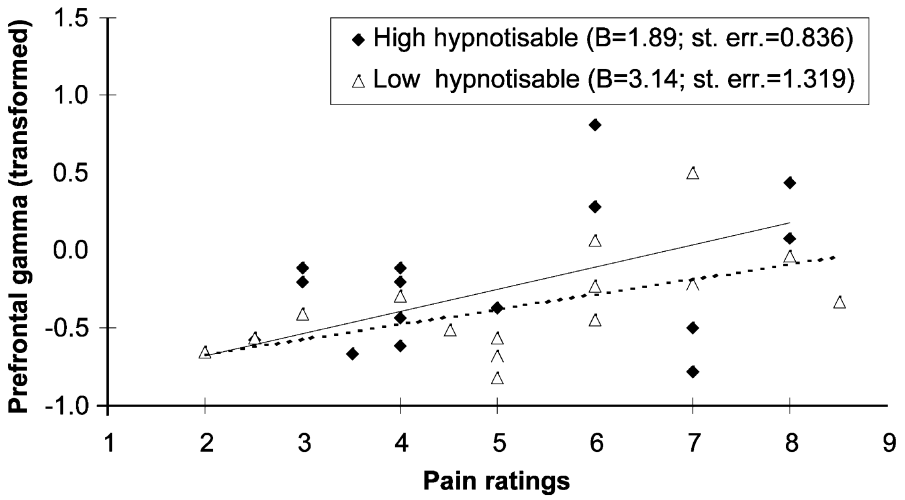

Fig. 1. The relation between subjects' prefrontal gamma activity and their reported pain levels is shown for the control condition, for the high (solid line) and low (dotted line) hypnotisable subjects separately.

T6) and occipital regions (PO1, PO2, O1, O2, Oz).

3. Results

Behavioural and ERP results will be reported elsewhere and will not be discussed. Relevant to the present study, subjects typically reported that they experienced a moderate level of pain (mean = 4.9; S.D. = 2.0).

3.1. Control (non-hypnosis)

1. Stepwise multiple regressions were used to determine if activity in any of the four frequency bands over any of the seven regions was related to pain ratings. This involved one regression for each frequency band, where the 7 scalp regions served as predictor variables and pain ratings the criterion variable (criterion for inclusion = 0.05; criterion for exclusion = 0.10). Prefrontal gamma predicted pain ratings ($B = 2.18$; $t = 3.13$; $P = 0.004$; $r = 0.50$), and no other frequency/region combination did. A non-parametric test found similar results (Spearman's rho = 0.45; $P = 0.011$), demonstrating that the relation between prefrontal gamma and pain was not due to outliers.
2. To determine whether this pain/gamma relation was independent of behavioural results, stimulus setting and threshold, a simultaneous multiple regression was used, where predictor variables were 'reaction time', 'accuracy', 'stimulus setting', 'stimulus threshold' and 'prefrontal gamma', and the criterion variable was 'pain ratings'. Prefrontal gamma predicted pain ratings independently of the other variables ($B = 2.44$; $t = 3.25$; $P = 0.003$), and none of the other variables independently predicted pain ratings ($t < 1.59$; $P > 0.124$).
3. To determine whether this pain/gamma relation was internally consistent, simple regressions were performed for high and low hypnotically susceptible subjects separately, where 'prefrontal gamma' was the predictor and 'pain ratings' the criterion variable. As can be seen in Fig. 1, prefrontal gamma predicted pain ratings in both HIGHs ($B = 1.89$; $t = 2.27$; $P = 0.040$) and LOWs ($B = 3.14$; $t = 2.39$; $P = 0.033$).

3.2. Hypnosis/hypnotic analgesia

1. To determine whether the pain/gamma relation held during hypnosis and hypnotic analgesia, simple regressions were performed for the two hypnotic susceptibility groups, for each condi-

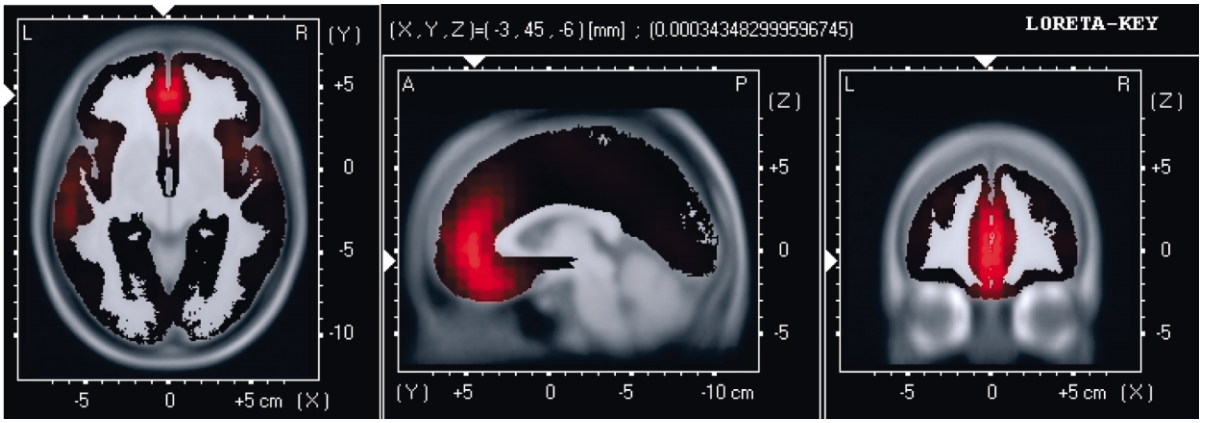

Fig. 2. The LORETA-estimated source-field of the gamma activity is shown to be centred about bilateral anterior cingulum [Brodmann Area 32; Talairach co-ordinates (± 3, 45, -6)].

tion separately, where 'prefrontal gamma' was the predictor and 'pain ratings' the criterion variable. In accordance with the results from the control condition, tests were directional, predicting more pain with more prefrontal gamma. Prefrontal gamma predicted pain ratings in the low susceptibility group during hypnosis ($B=3.47$; $t=1.77$; $P=0.050$) and hypnotic analgesia ($B=1.96$; $t=2.13$; $P=0.027$). Importantly prefrontal gamma did not predict pain ratings in the highly susceptible group during either hypnosis ($B=-2.24$; $t=-0.70$; $P=0.249$) or hypnotic analgesia ($B=-0.27$; $t=-0.24$; $P=0.409$).

2. To determine whether the lack of gamma/pain relation in highly susceptible subjects during either of the hypnosis conditions was due to prefrontal gamma decreasing during the hypnosis conditions, repeated measures contrasts compared the two susceptibility groups, and the control condition with the combination of the hypnosis and hypnotic analgesia conditions. Prefrontal gamma was the same in the control and hypnosis conditions ($F_{1,29}=0.02$; $P=0.892$), and this did not interact with group ($F_{1,29}=1.56$; $P=0.222$).

3.3. Source estimation

The source of the prefrontal gamma was estimated using LORETA (Pascual-Marqui et al., 1994). To separate gamma related to pain from other gamma, principal component analysis was used (Varimax rotation; correlation matrix; eigenvalues >0.6; 5 factors extracted; 94% variance explained), and the factor loadings used in place of scalp site amplitude values. The third factor corresponded to prefrontal gamma, correlated with pain ratings ($r_{\text{Spearman}}=0.47$; $P=0.01$) and was thus used to represent prefrontal gamma. As can be seen in Fig. 2, this factor was best estimated as a field centred about bilateral anterior cingulum [Brodmann Area 32; Talairach co-ordinates (± 3, 45, -6)].

4. Discussion

This study has found that increased gamma activity (32–100 Hz) over prefrontal scalp sites is related to the subjective experience of pain. This relation was shown to be internally consistent in the control condition (present in both high and low hypnotically susceptible subjects) and consistent across conditions in that it remained through the hypnosis and hypnotic analgesia conditions in subjects low in hypnotic susceptibility (that the relation did not occur in subjects high in susceptibility during the hypnosis conditions will be discussed later). Importantly, this activity was independent of the activity related to both performance and stimulus parameter measures, suggesting

that the relation was not due to increased general processing following more painful stimuli nor merely due to stimulus intensity change. Furthermore, as gamma did not decrease with pain ratings during hypnosis for highly susceptible subjects, and as lower frequencies (particularly beta 2) did not correlate with pain ratings, muscle activity was unlikely to be responsible for this relation (Davidson, 1988).

That gamma activity accounted for 25% of the variance in subject ratings is particularly noteworthy given the following issues. Subjective and oscillatory aspects of pain were only measured once per subject which increases gamma variance due to the different physical properties of the subjects' heads (e.g. skull and brain densities, volumes and morphologies). Second, error variance will occur because subjects' reports of their pain do not necessarily correspond to the level of pain that they experience due to the inherent difficulties in communicating such concepts. Third, pain ratings related to a 10-min period. This increased the signal to noise ratio, but would have introduced gamma variance due to habituation. Finally, gamma was calculated from a 500-ms time window. This window was chosen to avoid invoking unjustifiable a priori rationale, but refinement to this window would remove some gamma 'unrelated to pain' and would increase the proportion of pain ratings explained by gamma.

The origin of the gamma was best estimated by a field centred about bilateral anterior cingulum (ACC; Brodmann area 32). This estimate is necessarily low in resolution, and, as co-registration was not available in the present study, we can only make tentative conclusions about source and shall treat this as the anterior cingulate region (ACCr). Such an interpretation of the source of the gamma is consistent with a number of findings. For example ACC metabolism has been found to vary with within-subject pain report (Rainville et al., 1997; Porro et al., 1998), lesions to ACC produce antinociceptive effects in mice (Lee et al., 1999) and dedicated pain cells have been isolated in ACC (Hutchison et al., 1999).

Further, that the frequency related to pain was gamma is consistent with research implicating gamma with subjective experience. For instance, gamma is enhanced when people subjectively distinguish a pattern from a seemingly random array of visual stimuli (Keil et al., 1999), gamma increases during dreaming (Llinas and Ribary, 1993) and hallucinating (Baldeweg et al., 1998), and of particular relevance to pain, gamma is reduced when anaethetised (Kulli and Koch, 1991). In contrast to other research (Backonja et al., 1991; Veerasarn and Stohler, 1992; Chen and Rappelsberger, 1994), pain did not relate to lower frequencies in the present sample. This may suggest that the reported effects in the lower frequencies were due to non-pain-related processes such as attentional modulation that were controlled in the present study, however as this study was not designed to replicate those results specifically, it remains that the differences may be due to protocol differences.

That the relation between pain report and gamma was not present in highly hypnotically susceptible individuals during either hypnosis conditions sheds light on the neural mechanisms involved with the hypnosis process. In particular, this supports the view that hypnosis involves the suspension of a high order attention system (Crawford and Gruzelier, 1992; Woody and Bowers, 1994) and other anterior executive functions (Gruzelier, 1998). This is because gamma did not decrease in the highly susceptible subjects during hypnosis, but rather it was no longer related to their subjective experience—it would seem that the gamma activity was no longer important to the highly susceptible subjects. This is consistent with research demonstrating that error-related negativity, a preconscious anterior cingulate-generated response, is not affected by hypnosis, whereas the later error-related positivity is (Kaiser et al., 1997). It is also consistent with a number of studies reporting hypnosis-induced impairment on frontal lobe tasks (Gruzelier and Warren, 1993; Woody and Farvolden, 1998; Nordby et al., 1999; Kallio et al., 2001).

In conclusion, the present study has found gamma oscillations recorded over prefrontal sites to be related to the subjective experience of pain, independent of stimulus intensity and performance measures. As this relation was between subjects suggests that with further clarification, this may

allow an objective measure of the subjective experience of pain. Hypnosis was found to interfere with this gamma/pain relation, suggesting that consistent with previous research, hypnosis interferes with normal anterior brain function involving a high order attention system.

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