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Short Communication

Intracranial EEG power spectra and phase synchrony during consciousness and unconsciousness

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ABSTRACT

Power density spectra and phase synchrony measurements were taken from intracranial electrode grids implanted in epileptic subjects. Comparisons were made between data from the waking state and from the period of unconsciousness immediately following a generalised tonic–clonic seizure. Power spectra in the waking state resembled coloured noise. Power spectra in the unconscious state resembled coloured noise from 1 to about 5 Hz, but at higher frequencies changed in two out of three subjects to resemble white noise. This boosted unconscious gamma power to a higher level than conscious gamma power. For both gamma and beta passbands, synchrony measurements showed more widespread phase synchrony in the unconscious than the conscious state. We conclude that neither gamma activity *per se* nor phase synchrony *per se* are neural correlates of consciousness.

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1. Introduction

EEG practitioners before the introduction of analog to digital converters generally characterised the waking or conscious state as giving rise to what they called desynchronised EEG, in contrast to the synchronised EEG of deep sleep. However, over the last couple of decades this bit of folk-lore has largely been buried by a small avalanche of experimental reports concluding that phase synchrony – especially gamma phase synchrony – increases when particular conscious events occur (e.g. Bressler, 1990; Rodriguez et al., 1999; Roelfsema, Engel, Konig, & Singer, 1997; Srinivasen, Russell, Edelman, & Tononi, 1999; Stopfer, Bhagavan, Smith, & Laurent, 1997; Varela, Lachaux, Rodriguez, & Martinerie, 2001). It has thus become an influential idea that gamma power and long-range neural synchronisation are neural correlates of consciousness.

How does this fit in with the earlier ideas that neural *desynchronisation* is the signature of consciousness? It is, one supposes, possible that neural correlates of the contents of consciousness might be associated with eddies within an overall decreased level of synchrony characterising the state of consciousness (although it must be admitted that this explanation is somewhat confusing). However, since the advent of computerised EEG processing, it has even been reported that phase synchrony decreases with loss of the *state* of consciousness at onset of anesthesia (John et al., 2001). This latter report has recently been quoted in support of a theory specifically associating the neural correlates of consciousness with synchrony (Hameroff, 2009). Thus it seemed to us important to relitigate the early dogma that the state of consciousness is associated with desynchronised EEG, while the state of unconsciousness is associated with synchronised EEG.

Our initial attempts to investigate this question were immediately confronted with one of the major unmentionable problems of consciousness research, which is that the only ways of determining whether or not consciousness is actually present

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in somebody else at any given moment are (a) to observe their responses to external stimuli and/or (b) to ask them, after the moment is over, if they remember being conscious. The default position with respect to these two questions is that consciousness is present when the subject is reactive and not amnesic (i.e. they do remember being conscious). If they are not reactive and they are amnesic, the presumption is that they are not conscious.

But in practice, things are not so simple. Using various drugs commonly employed by anesthetists, it is fairly easy to dissociate reactivity from remembering. For example, benzodiazepines can induce a state in which the subject is reactive but amnesic. Curare in combination with an inadequate dose of various anesthetics can induce a state in which the subject is not reactive, but remembers all too well. Thus it is not easy to be certain that awareness is *not* present at any particular time during general anesthesia.

When studying sleep, the problem is dreaming. Dreaming can reasonably be considered to be a form of consciousness, albeit one involving internally rather than externally generated experiences. During dream consciousness, subjects are usually unreactive to external stimuli (although strong auditory or somatosensory stimuli can be experienced in the context of the dream) and often amnesic (although dreams can sometimes be remembered, particularly if the subject wakes up in the middle of one). EEG-wise, it used to be thought that dreams are present in REM sleep and not present in NREM sleep, but more recent reports indicate that NREM sleep is not dream-free (Hobson, Pace-Schott, & Stickgold, 2000). Thus it is not easy to be certain that no conscious experiences exist during a particular time period in sleep, either.

Coma is a notoriously variable state, in which subjects sometimes report on waking that they were aware while completely unreactive. So short of inflicting serious blows to the head just before recording, there initially seems to be no reliable way of obtaining data from a subject who is certainly unconscious.

However, a little thought reveals one ethically accessible condition that does seem to represent a clearcut case of unconsciousness – or at least a case in which the subject is demonstrably both completely unreactive and completely amnesic. This state occurs during the few minutes immediately after the cessation of a generalised tonic–clonic (aka Grand Mal) epileptic seizure. Therefore, in the present study we chose to make within-subject comparisons of phase synchrony during this post-ictal period with phase synchrony during waking.

2. Materials and methods

Data were analysed from three subjects in whom intracranial electrodes had been implanted for the clinical purpose of localising epileptic foci. Subject 1 was a female aged 23 years, Subject 2 was a female aged 20 years and Subject 3 was a male aged 15 years. All were evaluated at the University of Washington Regional Epilepsy Center, based at Harborview Medical Center in Seattle, Washington. All had medically refractory localisation-related epilepsy, manifested by complex partial and secondarily generalised seizures, and all were candidates for epilepsy surgery. The initial noninvasive evaluation included detailed history, clinical examination, standard EEG, magnetic resonance imaging, neuropsychological assessment, and longterm scalp EEG-video monitor studies. After determination that invasive EEG recordings were necessary to localise seizure onsets, each subject underwent longterm intracranial subdural EEG-video monitor recordings that included placement of a subdural, 64 contact, 8×8 “grid” of electrodes over the convexity of the cerebral hemisphere of interest, supplemented by subdural strip electrodes (6–16 electrodes each) implanted over adjacent cortical regions. The total number of intracranial electrodes ranged between 98 and 128 contacts. The interelectrode distance (center to center) within an individual grid or strip array was 10 mm. Fig. 1 is a schematic drawing that shows the approximate locations of the subdural electrodes for each subject. Clinical seizures, typical for each individual, were recorded in all cases, and included both partial seizures and secondarily generalised tonic–clonic convulsions. Continuous audiovisual recordings were time-locked to EEG data, allowing for simultaneous analysis of the clinical and electrographic manifestations of the epileptic seizures. The location of invasive electrodes and length of time deemed necessary for continuous EEG-video recordings were dictated solely by clinical considerations. Each patient (including the guardian in the case of the minor), gave informed consent prior to invasive EEG recordings, and permission for EEG analysis for research purposes was obtained from the University of Washington Human Subjects Review Committee.

Data were recorded at a sampling rate of 500 Hz for Subjects 1 and 2 and a sampling rate of 2000 Hz for Subject 3, with an analog passband of 1–70 Hz for all subjects. Since the subjects were also routinely videotaped, seizure activity was easily identified both from the video and from visual inspection of the intracranial EEG records. One minute selections of waking states were obtained from time periods when the video showed the subject in a state of quiet wakefulness and the intracranial EEG data were relatively artifact-free on visual inspection. Post-ictal recordings were selected from 1 min time periods immediately following a convulsive seizure, when high voltage ictal activity ceases and the EEG appears on visual analysis to be of relatively low amplitude and “quiescent”, with minimal artifact.

2.1. Signal processing

After conversion of the EEG data to Matlab™ format, all analyses were done using purpose written Matlab code. Data from each channel were visually checked and the small number of “bad” channels in each subject’s data were eliminated from further processing. Power density spectra were computed both by constructing a spatial ensemble average (i.e. for each sampling point taking the mean of all available channels) and then applying the Matlab command ‘pmtm’, and by applying

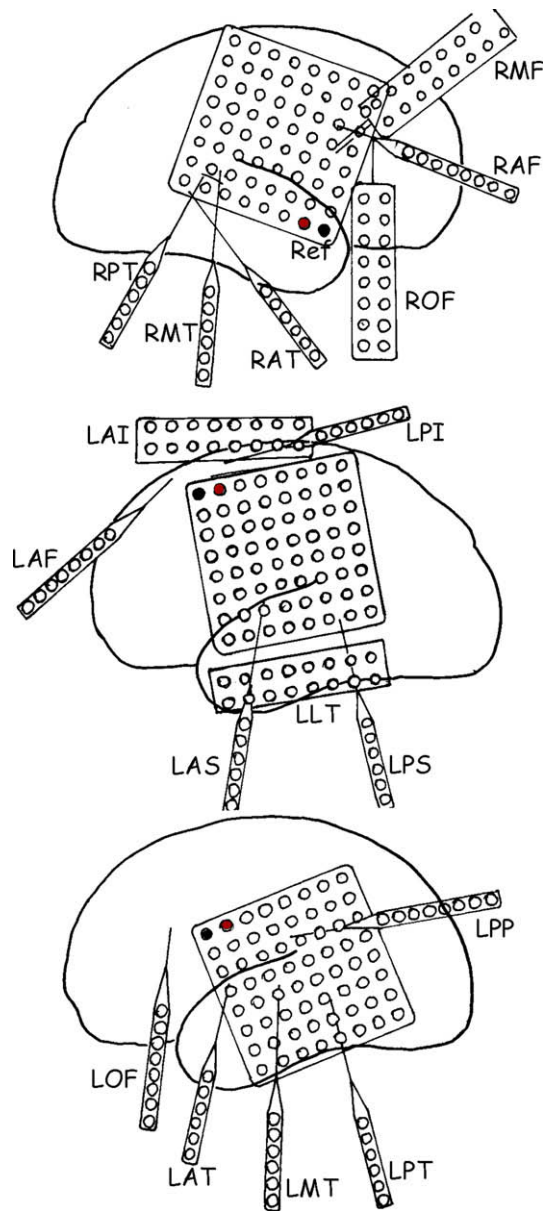


Fig. 1. Electrode positions. Top: Subject 1. Middle: Subject 2. Bottom: Subject 3. Black electrodes: recording reference. Red electrodes: synchrony reference. In all three cases a 64 contact 8×8 subdural grid of electrodes is placed over the convexity of the cerebral hemisphere of interest, encompassing portions of frontal, lateral temporal, and parietal lobes. Supplementary subdural strip electrodes (6–16 contacts) are placed over adjacent areas; the schematic drawing should be interpreted as showing the strips as “wrapping” over the involved cortical regions. RMF = right medial frontal. RAF = right anterior frontal. ROF = right orbital frontal. RAT = right anterior inferior medial temporal. RMT = right inferior medial temporal. RPT = right posterior inferior medial temporal. LAI = left anterior interhemispheric. LPI = left posterior interhemispheric. LAF = left anterior frontal. LAS = left anterior inferior medial temporal. LPS = left posterior inferior medial temporal. LPP = left posterior parietal. LOF = left orbital frontal. LAT = left anterior inferior medial temporal. LMT = left medial inferior temporal. LPT = left posterior inferior medial temporal (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.).

‘pmtm’ to each channel in turn. The resulting spectra were plotted on log–log axes. Synchrony was computed using the SAPD (spatial analytic phase difference) metric of Pockett, Bold, and Freeman (2009). This basically involves detrending the data, band-pass filtering them with a zero-phase-shift FIR filter, then using the Hilbert transform to compute a time series of unwrapped analytic phase for each data channel. At each time point, the instantaneous phase of each data channel is then subtracted from the instantaneous phase of the chosen synchrony reference channel. Instantaneous phase differences of <0.2 radians are taken as indicating synchrony of the data channel with the reference channel. The number of channels in synchrony with the reference channel at each time point is then summed and plotted as a time series, allowing an estimate of

phase synchrony with a time resolution limited only by the sampling rate. Further details and explanation are given by Pockett et al. (2009).

4. Results and discussion

Figs. 2 and 3 show the power density spectra for waking and post-ictal time periods and also the number of channels in synchrony for waking and post-ictal time periods for both beta (12–18 Hz) and gamma (40–46 Hz) filtered data, for each of the three subjects. For Subject 1, samples of raw and filtered data are also shown.

4.1. Power spectra

The main remarkable feature of the power density spectra shown in these figures is that conscious and unconscious behavioural states both give rise to spectra reminiscent of pink or brown noise up to a frequency of between 5 and 6 Hz, but at frequencies above that, the spectrum from the conscious state of all subjects continues to look like that of coloured noise, while the spectra from the unconscious state of Subjects 1 and 2 abruptly changes to resemble that of white noise (which has a flat spectrum on a log–log plot). This change from coloured to white noise boosts overall gamma power in the unconscious condition to a higher level than gamma power in the conscious condition.

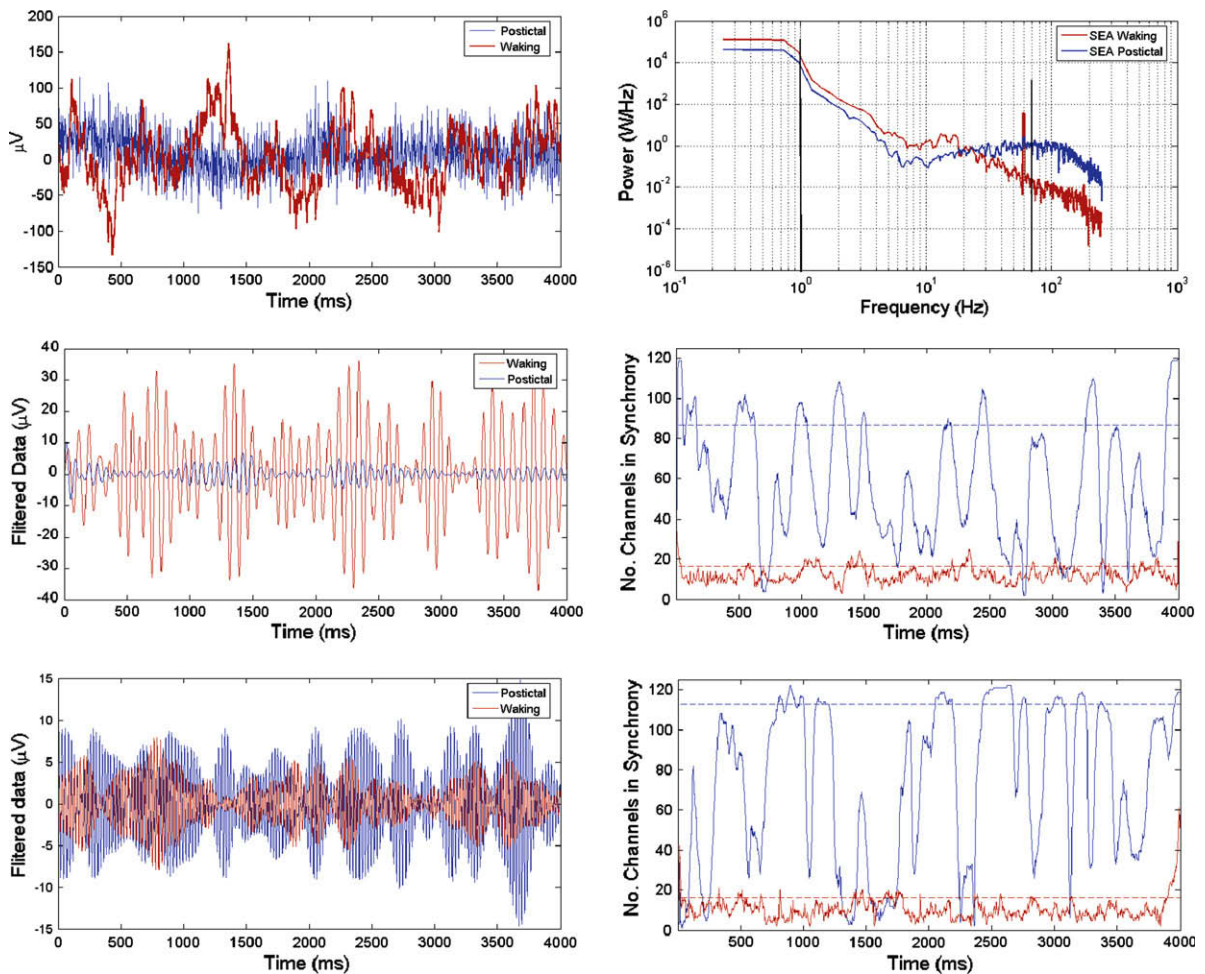


Fig. 2. Raw and filtered data, power density spectra and synchrony in beta and gamma bands for conscious and unconscious states of Subject 1. Top left panel: raw data for conscious (red) and unconscious (blue) states. Middle left panel: beta filtered data (12–18 Hz) for conscious (red) and unconscious (blue) states. Bottom left panel: gamma filtered data (40–46 Hz) for conscious (red) and unconscious (blue) states. Top right panel: power density spectra for conscious (red) and unconscious (blue) states. Vertical black lines indicate nominal cut-off frequencies of hardware band-pass filter. Middle right panel: time series of numbers of channels in synchrony for beta filtered data (conscious – red; unconscious – blue). Dashed horizontal lines show the mean + one standard deviation of number of channels in synchrony for conscious (red) and unconscious (blue) states. Bottom right panel: time series of numbers of channels in synchrony for gamma filtered data (conscious – red; unconscious – blue). Dashed horizontal lines show the mean + one standard deviation of number of channels in synchrony for conscious (red) and unconscious (blue) states (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.).

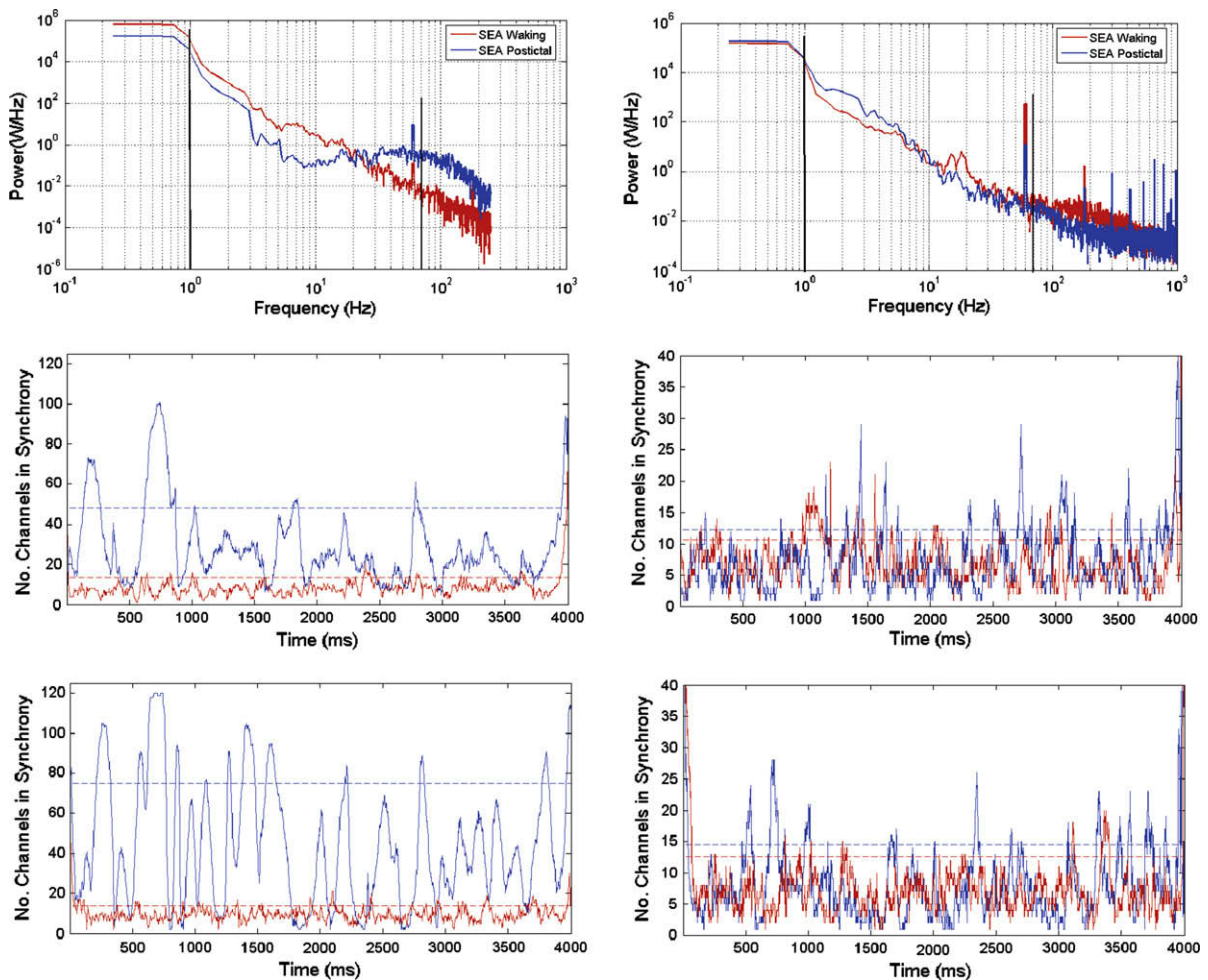


Fig. 3. Power density spectra and synchrony in beta and gamma bands for conscious and unconscious states of subjects FW and VD. Top left panel: power density spectra for conscious (red) and unconscious (blue) states of Subject 2. Vertical black lines indicate nominal cut-off frequencies of hardware band-pass filter. Middle left panel: time series of numbers of channels in synchrony for beta filtered data from Subject 2 (conscious – red; unconscious – blue). Dashed horizontal lines show the mean + one standard deviation of number of channels in synchrony for conscious (red) and unconscious (blue) states. Bottom left panel: time series of numbers of channels in synchrony with channel 2 for gamma filtered data from Subject 2 (conscious – red; unconscious – blue). Dashed horizontal lines show the mean + one standard deviation of number of channels in synchrony for conscious (red) and unconscious (blue) states. Top right panel: power density spectra for conscious (red) and unconscious (blue) states of Subject 3. Middle right panel: time series of numbers of channels in synchrony with channel 2 for beta filtered data from Subject 3 (conscious – red; unconscious – blue). Dashed horizontal lines show the mean + one standard deviation of number of channels in synchrony for conscious (red) and unconscious (blue) states. Bottom right panel: time series of numbers of channels in synchrony for gamma filtered data from Subject 3 (conscious – red; unconscious – blue). Dashed horizontal lines show the mean + one standard deviation of number of channels in synchrony for conscious (red) and unconscious (blue) states (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

In post-ictal data from Subject 1, the bipartite nature of the PSD slope seen in the spatial ensemble average was also strongly evident in the PSDs of all individual channels. In post-ictal data from Subject 2, only 101 of the 121 channels studied showed a bipartite slope like that seen in the spatial ensemble average. The remaining 20 channels had PSD slopes that were either straight lines, or showed only a slight flattening at higher frequencies. These 20 channels were clustered in several small areas, none of which showed any obvious spatial relationship with the epileptic focus.

In contrast to Subjects 1 and 2, white noise did not appear at high frequencies post-ictally in Subject 3. The post-ictal PSD of Subject 3's spatial ensemble average (Fig. 3 top right panel) shows only a slight flattening in slope, starting at about 20 Hz. Individually, only 32 of the 92 channels studied showed even this slight slope change, while the other 60 were essentially straight lines, broken in six of the channels by significant "bumps" similar to that in the spatial ensemble average of this subject's waking PSD but centred at various frequencies (15 Hz, 20 Hz, 25 Hz, 30 Hz, 40 Hz), in different combinations for different channels.

The possibility that this major difference between the PSD of Subject 3 and that of the other two subjects is the result of a processing error stemming from the different sampling rate of Subject 3's data is excluded by the observation that the

expected 60 Hz line noise spike is present, in the right place, in Subject 3's power spectra. A more likely explanation for the inter-subject difference is that the appearance or non-appearance of white noise at higher frequencies may be related to duration of illness. Subject 1, in whom white noise was seen at higher frequencies in all 122 channels studied, was 23 years old at the time the data were recorded. Subject 2, in whom white noise was seen in 101/121 channels, was 20 years old at the time of the study. Subject 3, in whom no white noise at all but only a slightly bipartite PSD slope was seen, and that in only 30/92 individual channels, was 15 years old at the time of the study. Clearly no firm conclusions can be drawn on the basis of so few subjects, but the data are suggestive that the longer a brain is subjected to regular seizures, the more likely it is that each seizure will be followed by a period during which high frequency brain activity becomes completely random, with none of the power law characteristics of normal brain activity. More work is needed to investigate this possibility further.

In any case, given that Subject 3 was just as unreactive and amnesic as the other two subjects during the post-ictal period, but unlike the other two subjects did not show higher gamma power post-ictally than during waking, it cannot be said that high gamma power is a universal correlate of either consciousness or unconsciousness. The present finding that gamma power can be higher in the unconscious than the conscious state conflicts with the current folklore that gamma activity correlates with consciousness, but not with the findings that (a) there can be more gamma in anesthetised than waking rats (Vanderwolf, 2000) and (b) gamma activity can be present during slow wave sleep in cats (Steriade, Amzica, & Contreras, 1996; Steriade, Contreras, & Amzica, 1996). However, the latter observations are perhaps less convincing than those in the present report in terms of showing a lack of correlation between gamma activity and consciousness, owing to the difficulties mentioned in the Introduction about being sure that consciousness is really absent at any particular moment during either anesthesia or sleep.

4.2. Phase synchrony

The main obvious feature of the synchrony plots in the present report is that in all cases there is more widespread synchrony in data from the unconscious state than in data from the conscious state. This is true for both beta and gamma passbands. Fig. 2 shows that the effect is not just a consequence of increased volume conduction in higher power data, since it occurs not only in the gamma passband, where unconscious data have higher power than conscious data, but also in the beta passband, where the opposite is true. The passbands shown in Figs. 2 and 3 for beta and gamma are 12–18 Hz (beta) and 40–46 Hz (gamma). However very similar results were also found for data filtered at 18–25 Hz (beta) and 38–48 Hz (gamma). In Subject 3, where conscious power is higher than unconscious power in both beta and (marginally) gamma bands, both beta and gamma synchrony are still higher in the unconscious state, although this effect is not so marked as in the other subjects.

Again this finding is in marked contrast with the general folklore that consciousness is associated with gamma synchrony. However it is in general accord with various papers that:

- (1) Report problems with attempts to replicate the original experimental reports claiming such an association (Trujillo, Peterson, Kaszniak, & Allen, 2005).
- (2) Point out theoretical reasons for doubting that synchrony *per se* can be a sufficient correlate of consciousness (Canales, Gomez, & Maffet, 2007; Sowards & Sowards, 2001; Shadlen & Movshon, 1999).
- (3) Show that short periods of synchrony appear upon recognition of an external stimulus only in some groups of subjects – not, for example, in adolescents (Uhlhaas et al., 2009).

It does seem that intermittent periods of high beta and/or gamma power which is transiently synchronised between distant sites have something to do with the ability to report conscious events (Gaillard et al., 2009; Tallon-Baudry, 2009; Wyatt & Tallon-Baudry, 2008). However, our findings that (a) overall gamma power can be higher during unconsciousness than during consciousness and (b) widespread phase synchrony can be more evident during unconsciousness than during consciousness, lead us to conclude that neither gamma power *per se* nor synchrony *per se* can be called a neural correlate of consciousness.

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