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Transient Phase-Locking and Dynamic Correlations: Are They the Same Thing?

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Abstract: This work represents an attempt to bring together two important themes in neuronal dynamics. The first is the characterization of *dynamic correlations* in multiunit recordings of spike activity using joint-peri-stimulus time histograms (J-PSTHs) [Aertsen and Preissl, 1991: Non Linear Dynamics and Neural Networks]. The second is transient *phase-locking* at high (gamma) frequencies, either in terms of spiking in separable spike trains [e.g., Eckhorn et al., 1988: Biol Cybern 60:121–130, Gray and Singer, 1989 Proc Natl Acad Sci USA 86:1698–1702], or using continuous electrical or biomagnetic signals [e.g., Desmedt and Tomberg, 1994 Neurosci Lett 168:126–129]. In this paper we suggest that transient phase-locking is necessary for frequency-specific, dynamic event-related correlations. This point is demonstrated using the gamma-frequency (36 Hz) component of neuromagnetic signals measured in the prefrontal and partial regions of a subject during self-paced movements. A J-PSTH analysis revealed dynamic changes in prefronto-parietal correlations in relation to movement onset. These frequency-specific dynamic correlations were associated with changes in the degree of phase-locking, of the sort reported by Desmedt and Tomberg [1994 Neurosci Lett 168:126–129]. *Hum. Brain Mapping 5:48–57, 1997.* © 1997 Wiley-Liss, Inc.

Key words: neural dynamics; phase-locking; dynamic correlations; MEG; self-paced movement; joint-PSTH

INTRODUCTION

This paper is about fast dynamic interactions in the brain, as measured with magneto-encephalography (MEG). Its principal aim is to suggest that transient phase-locking, of high-frequency neuronal oscillations, can result in dynamic or time-dependent changes in the correlation between two neuronal processes, at the frequency in question, following a salient sensory or behavioral event. If true, this would establish a relationship between two important aspects of neuronal dynamics. The first is the characterization of *dynamic correlations* in multiunit recordings of spike activity using joint-peri-stimulus time histograms (J-PSTHs) [Aertsen and Preissl, 1991] as exemplified by the recent paper of Vaadia et al. [1995]. The second is *phase-locking* at high (gamma) frequencies, either in terms of the probability of spiking in separable spike trains [e.g., Eckhorn et al., 1988; Gray and Singer, 1989] or using continuous electrical and biomagnetic signals [e.g., Desmedt and Tomberg, 1994]. (See also Thatcher et al. [1994] for a summary of related work at lower (theta)

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frequencies.) The contention here is that event-related phase-locking is necessary for the emergence of frequency-specific, event-related correlations in neuronal activity.

A fundamental phenomenon observed by Vaadia et al. [1995] is that, following behaviorally salient events, the degree of coherent firing between two neurons can change profoundly and systematically over the ensuing second or so. Furthermore, the mean firing rate (averaged over epochs) does not, necessarily, change. One implication is that a "better" metric of neuronal interactions could be framed in terms of dynamic changes in correlations, modulated on time scales of 100–1,000 msec. Dynamic correlations are measured, not *over time*, but over trials or epochs as a *function of time* after an event. These correlations can be characterized using J-PSTHs, which are effectively crosscorrelation matrices referred to an event of interest [Aertsen and Preissl, 1991]. J-PSTHs can reveal the emergence and subsequent decay of "excess" correlations following an event. Recently, it was noted that these dynamic correlations could result from the correlated expression of stereotyped and transient changes in the propensity to fire [Friston, 1995], where these transients have the same time course as the dynamic correlations they produce. Desmedt and Tomberg [1994] demonstrated recently a transient phase-locking of gamma waves (35–45 Hz) in the prefrontal and parietal cortex using electroencephalography. This transient phase-locking was observed during selective attention and enabled the authors to make some interesting inferences about the role of electrophysiological synchronization and the integration of perceptual features into the behaviorial domain.

In this paper, J-PSTH analysis was applied to MEG data obtained during self-paced movements in man. To discount dynamic correlations due to the correlated expression of slow transients [Friston, 1995], the data were filtered to leave only a high (gamma) frequency. The analysis revealed dynamic changes in prefronto-parietal correlations in relation to movement onset. These frequency-specific dynamic correlations were taken to imply changes in the degree of phase-locking of the sort reported by Desmedt and Tomberg [1994]. We were able to find evidence for modulation of phase-locking using a post hoc analysis of phase differences.

This paper is divided into two sections. The first section provides a brief mathematical description of the relationship between phase-locking and dynamic correlations. The second section deals with the empirical (MEG) data used and the results of a J-PSTH analysis demonstrating frequency-specific dynamic correlations. The section concludes with a demonstration of phase-locking using the distribution of prefronto-parietal phase differences, estimated over trials or epochs.

PHASE-LOCKING AND FREQUENCY-SPECIFIC DYNAMIC CORRELATIONS

This section provides a mathematical discussion of the intuitively obvious idea that if two neuronal processes show phase-locking, at some specific time after an event, then the activities, at this time, measured over repeated trials or epochs, will be correlated. In other words, consider two sets of signals, of the same frequency, that are observed at a particular point in time. The two values observed will only correlate if there is some systematic phase relationship between the two sets of signals. If the phase relationships are random, then the observed values will not be correlated. More specifically, it is shown that a systematic phase relationship is *necessary* for the emergence of dynamic correlations, measured at a given frequency. This is important because the demonstration of these frequency-specific correlations is an implicit demonstration of phase-locking.

Let the activities of two neuronal processes (e.g., discharge rates in two separable spike-trains or signals in two MEG channels), at time t in the post-event period, at a particular frequency (ω), be modelled by x_1 and x_2, where:

$$x_1(t) = \alpha_1 \sin(\omega t + \tau + \delta)$$
$$x_2(t) = \alpha_2 \sin(\omega t + \tau) \qquad (1)$$

where, for a given epoch, α_1 and α_2 represent the amplitudes of this frequency component, τ is the phase at which sampling occurred, and δ is the phase difference between the two processes. It should be noted that Equation (1) holds for any signals in the Fourier domain. α_1, α_2, τ, and δ are stochastic variables that pertain to a series of epochs, observed at time t. α_1 and α_2 are independent of δ and $\alpha_1, \alpha_2 > 0$. τ has a uniform distribution in the range $(-\pi, \pi)$ and is independent of δ (i.e., the sampling has no systematic phase relationship to the processes). The phase relationship between the two processes is described by the probability density function $P(\delta)$ again in the range

$(-\pi, \pi)$. By direct calculation, the covariance between $x_1(t)$ and $x_2(t)$ is given by

$$\gamma(t) = \langle x_1(t) \cdot x_2(t) \rangle$$
$$= \langle \alpha_1 \cdot \alpha_2 \cos(\delta) \rangle$$
$$\text{(by independence)} = \langle \alpha_1 \cdot \alpha_2 \rangle \int P(\delta) \cos(\delta)\, d\delta \quad (2)$$

where $\langle \cdot \rangle$ denotes expectation . and the integral is from $-\pi$ to π. Equation (2) implies that for a dynamic covariance $\gamma(t)$ to exist, the integral involving δ must be nonzero. This integral will only be nonzero if there is some systematic phase relationship between the two processes. In other words, phase-locking is a necessary condition for the expression of frequency-specific dynamic correlations. Note that phase-locking is not a sufficient condition (e.g., the phase difference could be $\pi/2$). Note also that covariance in the amplitude [cov $\{\alpha_1, \alpha_2\}$] [e.g., see Pfurtscheller and Aranibar, 1979] over epochs can affect the observed dynamic covariance. This is because $\langle \alpha_1 \cdot \alpha_2 \rangle = \langle \alpha_1 \rangle \langle \alpha_2 \rangle + \text{cov}\{\alpha_1, \alpha_2\}$. However, this effect can only be realized in the presence of phase-locking. In short, if one observes frequency-specific dynamic correlations, one can infer a degree of phase-locking. The argument presented above holds even if the measured brain signals are attenuated (or even phase-delayed) differentially by the measurement system. It is assumed, however, that the measurements are independent in the sense that the first signal does not contribute to the measurement of the second and vice versa.

Frequency-specific correlations and coherence

It is important to make a distinction between frequency-specific correlations and coherence. Although they are very similar, coherence does not change with δ, the phase difference. The Appendix includes an expression for coherence in terms of $P(\delta)$. Compare this equation [Eq. (A3)] with Equation (2). The difference can be seen most clearly if we assume exact phase-locking at some phase ∂. $P(\delta)$ is then a delta-function centered on ∂. The integral in Equation (2) then reduces to $\cos(\partial)$, whereas the integrals in Equation (A3) sum to unity. In other words, in the context of phase-locking, frequency-specific correlations are periodic functions of the phase-difference ∂, whereas coherence is not. This is of practical importance because the phase-difference can include differences due to "lag" λ, when computing the crosscovariance between $x_1(t)$ and $x_2(t + \lambda)$ in the J-PSTHs below. These crosscovariance (and crosscorrelation) functions are consequently periodic functions of λ at the frequency being examined.

ANALYSIS OF MEG DATA

In this section, MEG data were subject to J-PSTH analysis to demonstrate frequency-specific dynamic correlations in the gamma range and their event-related modulation. On the basis of the results obtained we then estimated the distribution of phases (cf. $P(\delta)$ in the previous section) when dynamic correlations were expressed to the greatest and least extents. As predicted theoretically, these distributions did indeed suggest prefronto-parietal phase-locking.

MEG data

MEG data were obtained from a normal subject during self-paced movements of a joystick using a Siemens KRENIKON® 37-channel machine. The subject was trained to perform the movement, with the right hand, every 2 sec or so. The data were acquired every millisecond for 72 movements. ECG artifacts were removed using linear regression. In order to enhance the spatial resolution of the multichannel data we used a V_3 transformation [Ioannides et al., 1990] and selected two time-series from a prefrontal (anterior cingulate/SMA) and a parietal (left superior parietal/somatosensory) region (the exact locations are shown in Fig. 1, top right). The V_3 transformation uses spatial derivatives to effect something like an "edge-enhancement" and attenuates spurious correlations between regions that could be attributed to the low spatial resolution of MEG.

The prefrontal and parietal MEG time-series were sorted into 72 epochs of 2,000 msec, time-locked to the onset of electromyographic (EMG) activity at 1,000 msec. EMG onset was defined whenever the activity exceed 20% of its maximum, after the EMG data were squared and smoothed with a Gaussian kernel (256 msec wide). The average (square) of EMG activity over epochs is shown in Figure 1 (top left).

A frequency component at $\omega = 36$ Hz was extracted from the MEG data $x_i(t)$ using the following device:

$$f_i(t) = x_i(t) \otimes \{h(t) \cdot \exp(-j2\pi\omega t)\}$$
$$x_i^*(t) = \text{real}\{f_i(t)\} \quad (3)$$

where $i = 1$ or 2. Here $\otimes$ denotes convolution and $h(t)$ is some suitable windowing function. A 512-msec

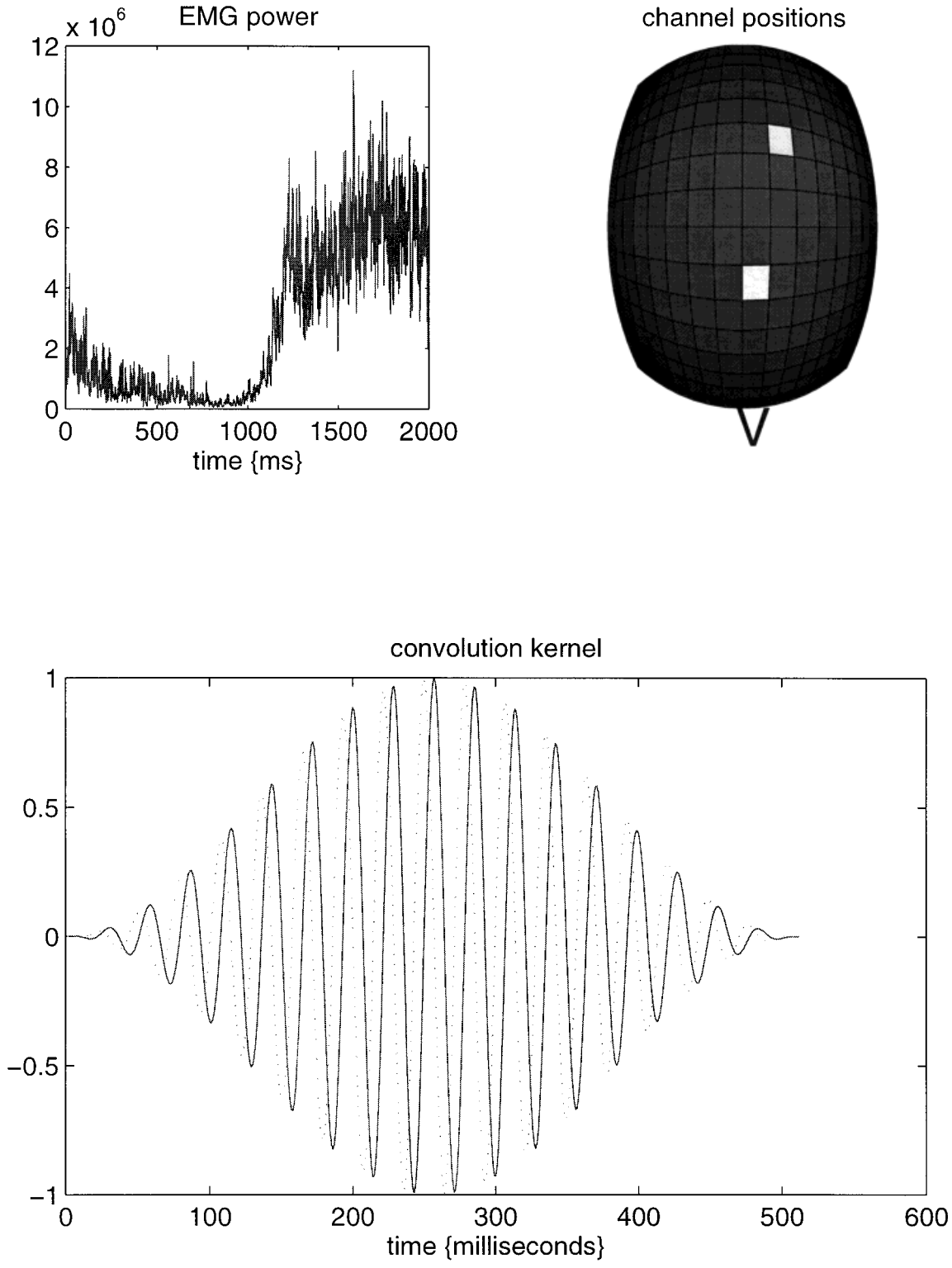


Figure 1.

Top right: Location of the two MEG time-series chosen for the analysis. These data were selected after a V_3 transformation of 37-channel MEG data obtained during self-paced movements. The 37 channels were located symmetrically, over both hemispheres, in 5 rows. Rows were arranged in equally-spaced coronal planes. V denotes the nose, and left corresponds to right. Lower square is over the anterior cingulate and SMA region. Upper square is over the left superior parietal region. **Top left:** Average of the square of EMG activity over all epochs used in the analysis. **Below:** The (complex) convolution kernel used to extract a gamma (36 Hz) frequency component from the data and to assess the phase relationships (solid line, real; dotted line, imaginary).

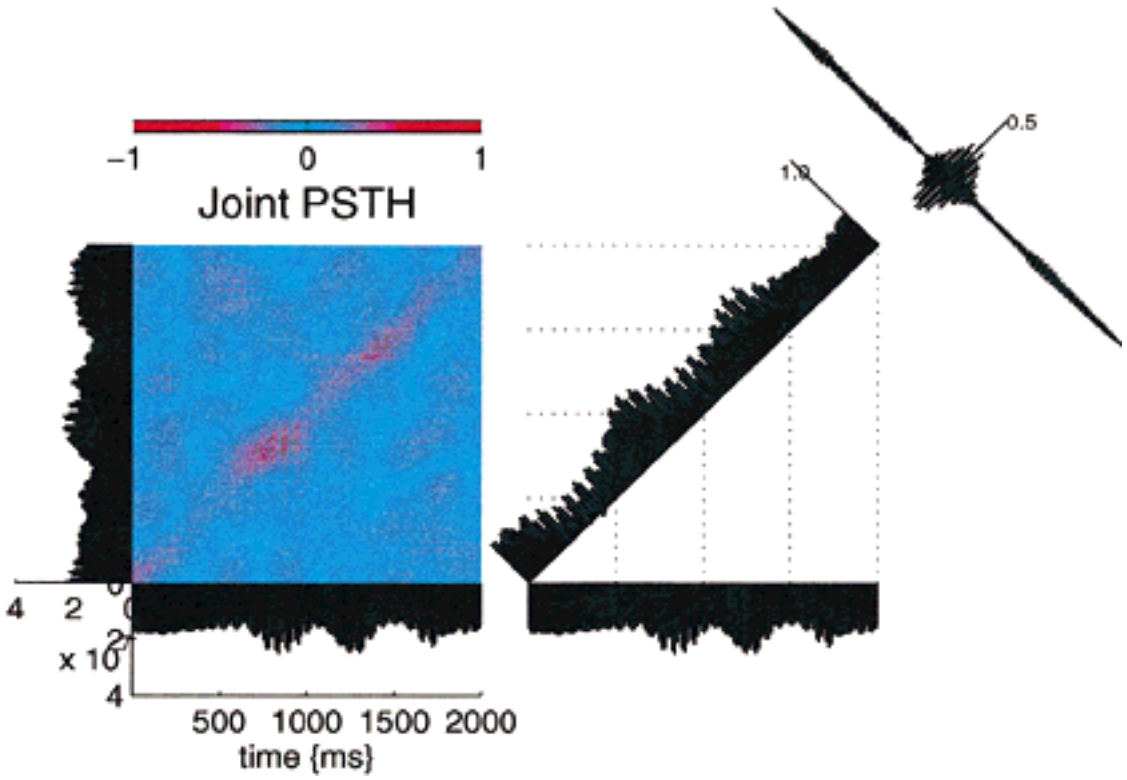


Figure 2.

Joint peri-stimulus time histogram (J-PSTH) based on MEG data. **Left:** Signal variance over epochs (side panels). These data replace the conventional peri-stimulus time histogram (PSTH) usually depicted in this format. Main panel is an image representation of the crosscorrelation matrix (referred to the stimulus event at 1,000 msec). The horizontal axis corresponds to the prefrontal channel, and the vertical axis to the parietal channel. The color scale employed does not differentiate between positive and negative correlations. This is because the sign of the correlation is not important: as discussed in the text, the crosscorrelation function is a periodic function of lag (with the same periodicity as the frequency at which the correlations are measured). The important aspect of this function is its amplitude. Note the initial reduction and then excess of correlations just before and after EMG onset. **Right:** Correlations as a function of time during the epoch (cf. a coincidence-time histogram). This is the main diagonal of the crosscorrelation matrix, showing the time-dependent nature of the correlations (referred to the stimulus event). The conventional time-averaged crosscorrelogram is shown at upper right.

Hanning function was used in this paper. Figure 1 (bottom) shows the real and imaginary parts of the convolution kernel $|h(t) \cdot \exp(-j2\pi\omega t)|$. $x_i^*(t)$ can be thought of as a filtered version of $x_i(t)$ with similar power and phase relationships but retaining only the frequency component ω.

Joint-PSTH analysis

The two sets of epochs (x_1^* and x_2^*) were subject to J-PSTH analysis as described in Vaadia et al. [1995]. For this analysis the data were reduced by resampling every 8 msec. The results are seen in Figure 2 and could be compared with Figure 2 in Vaadia et al. [1995]. The side panels correspond to the variance of the two time-series over epochs, and the main panel is an image representation of the crosscorrelation matrix (referred to the stimulus event at 1,000 msec). The crosscorrelation matrix shows an "excess" of correlations (200 msec) before and (400 msec) after EMG onset. These increases are preceded by a (200-msec) period of profoundly reduced correlations around 600 msec before EMG onset. The event-related profile of correlations is shown along the diagonal on the right, and more clearly reveals their time-dependent modulation. The crosscorrelogram (conventional crosscorrelation function) is shown at upper right. Remember that because we are dealing with frequency-specific correla-

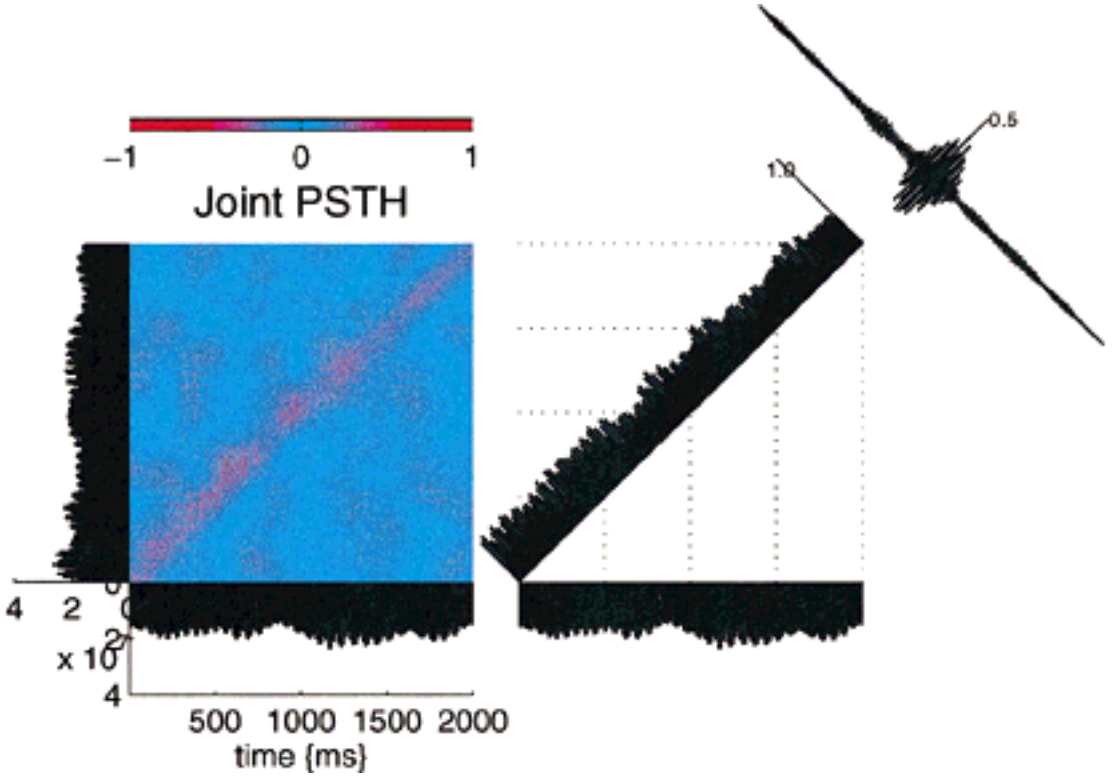


Figure 3.
J-PSTH analysis of null data. As for Figure 2, only using epochs that were not time-locked to EMG onset. Note that the leading diagonal of the crosscorrelation matrix shows little modulation over the epoch.

tions, the crosscorrelation functions are themselves periodic.

To demonstrate that this dynamic modulation is event-specific we repeated an identical analysis but chose (nonoverlapping) epochs at random (i.e., using epochs with no relationship to EMG onset. The corresponding J-PSTH is seen in Figure 3 and shows that the correlations are extant throughout the epoch with little obvious modulation.

Statistical inference

To ensure that the event-related modulation of dynamic correlations was significant, we normalized the leading diagonal of the J-PSTH in Figure 2, after smoothing with a 32-msec Gaussian kernel. This normalization used the estimates of its mean and standard deviation under the null hypothesis of no event-related modulation (i.e., using the observed mean and variance of the smoothed leading diagonal in Fig. 3). The resulting process Z(t) was then treated as a stochastic Gaussian process of the Z statistic, and extreme values were characterized using standard results from statistical parametric mapping [see Friston et al., 1995]. These expressions allow one to determine a *P* value for each time-bin that reflects the significance of very high or low correlations. This *P* value *P*(t) is the probability that the observed value of Z(t) or higher would have been found by chance over the entire epoch. In essense this *P* value is corrected for the length of the epoch and the autocorrelations in Z(t):

$$P(t) \leq S(2\pi W)^{-1} \exp\left(-Z(t)^2/2\right) \qquad (4)$$

where

$$W = \mathrm{Var}\left[\partial Z(t)/\partial t\right]^{-1/2}.$$

S is the length of the epoch and W is a parameter related to the smoothness or the autocorrelations in Z(t). In brief, this approach uses the theory of stochastic processes to calculate corrected *P* values based on the Z scores that comprise the process Z(t). This correction takes account of the fact that the Z scores are not independent [i.e., smoothness in Z(t)]. The assumptions implicit in this analysis are the same for statistical parametric maps of the Z statistic that show autocorrelations, namely (1) that the process Z(t) is a reasonable point-representation of an underlying continuous random Gaussian process under the null hypothesis; (2) that this process is stationary (the multivariate probability density functions, over many realizations, are not a function of t); and (3) the Z scores one makes inferences about are relatively high (this is because the distributional approximations used are asymptotically true at very high thresholds).

Figure 4, top, shows the two smoothed leading diagonals (or correlations at zero lag) from the epochs time-locked to EMG onset at 1,000 msec (solid line) and the epochs that were not (broken line). Figure 4, bottom, plots *P*(t) and shows significant early decreases ($P < 0.0001$) followed by increases ($P < 0.01$) in correlations immediately before EMG onset. Immediately after movement onset, there is a further decrease ($P < 0.01$). In conclusion, prefronto-parietal MEG gamma oscillations show nonspecific correlations. Self-paced movements are associated with significant modulations that include a profound attenuation of these correlations, at about 600 msec, which reverses to give an excess of correlations about 200 msec before EMG onset.

Distribution of phase relationships

To demonstrate that the dynamic correlations shown in Figure 1 were associated with changes in the systematic phase relationship, we computed the phase differences between the prefrontal and parietal signals at 760 msec (maximum correlation) and 352 msec (minimal correlation), for all the epochs. These differences (estimates of δ) were computed using

$$\text{angle}\{f_1(t_k)\} - \text{angle}\{f_2(t_k)\} \qquad (5)$$

where t_k was either 760 msec or 352 msec. The distributions of phase differences are shown in Figure 5 and can be thought of as estimates of the form of $P(\delta)$. Clearly, when dynamic correlations are more pronounced (Fig. 5, top) there is a tendency towards a zero phase difference, with the distribution being fairly tight (i.e., more phase-locking). Around 352 msec the distribution is much flatter and shifted to a positive nonzero phase relationship.

DISCUSSION

In this paper we suggest that the emergence of event-related transient phase-locking at a particular frequency is necessary for, and a likely concomitant of, dynamic correlations at that frequency. We demonstrated this point using a gamma-frequency component of MEG data taken from the prefrontal and parietal regions of a subject performing self-paced movements. A J-PSTH analysis of dynamic correlations showed attenuated and augmented correlations in relation to EMG onset that were associated with changes in phase-locking.

Qualifications

We have not presented an exhaustive study of dynamic correlations in relation to self-paced movements. The small set of data presented here serves to illustrate an idea. Anecdotally it appears that the modulation of dynamic correlations varies with the regions selected and with minor variations in the experimental design (e.g., joystick movement to the left or in random directions) (data not shown). It is possible that the background correlations, e.g., those seen in Figure 3, could reflect artifactual phase-locking due to the low spatial resolution of MEG. We made efforts to minimize this effect by using the V_3 transformation; however, it cannot be completely discounted. Event-related *modulation* of correlations could conceivably be attributed to transient bursts of power in the same neuronal generator, as picked up by both channels. However, this is an unlikely explanation for the event-related modulation of correlations seen in Figure 2 because the phase-relationships, during maximal correlations, are skewed to the right of 0 (Fig. 5). The modulation is therefore more likely to reflect real changes in long-range neuronal interactions.

As noted in an earlier section, phase-locking is not a sufficient condition for correlations to emerge (e.g., the mean phase difference could be $\pi/2$). This means that the observed crosscorrelations at any given lag will be a function of the relative phase difference. In terms of characterizing transient phase-locking, or frequency-

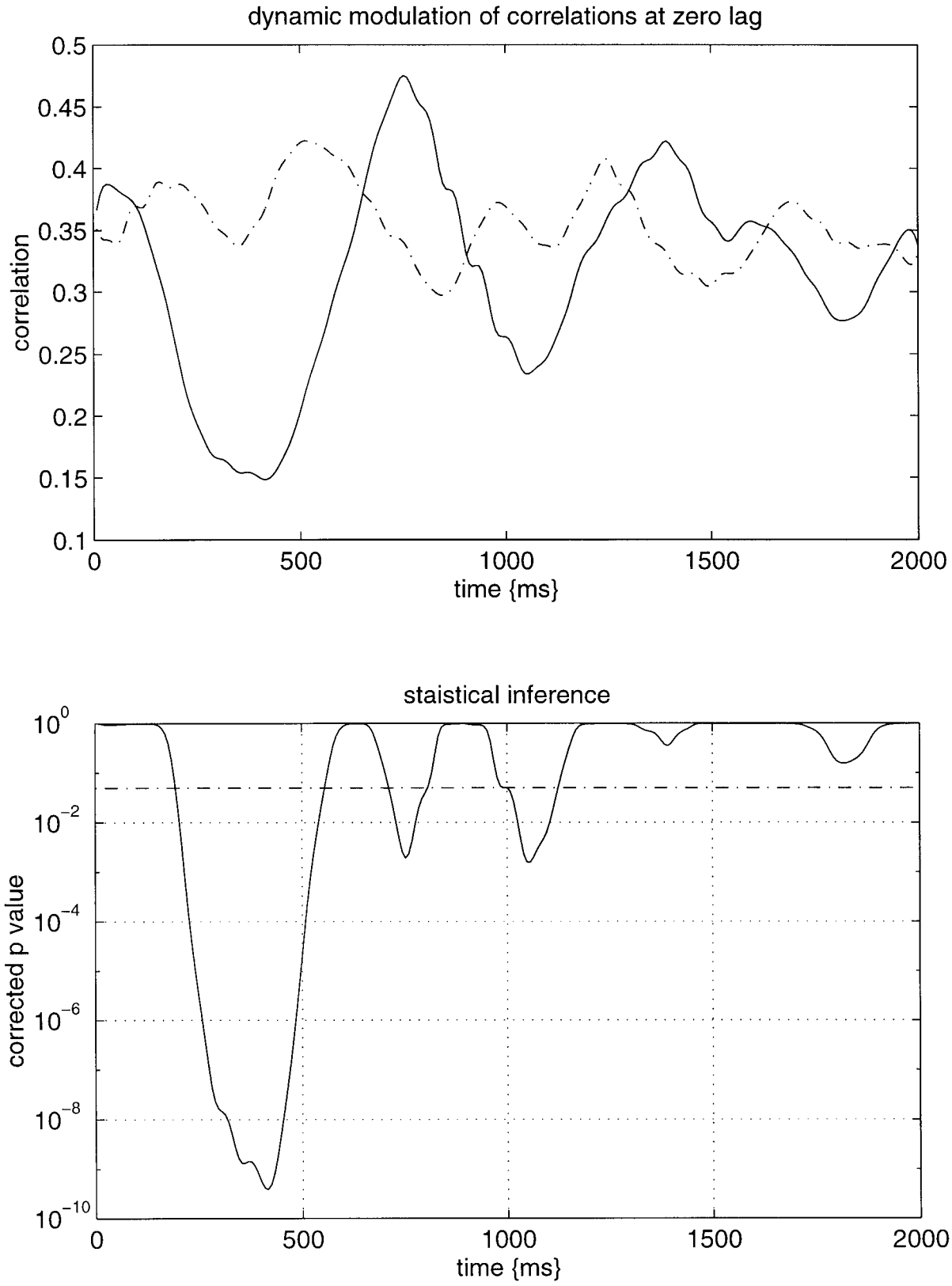
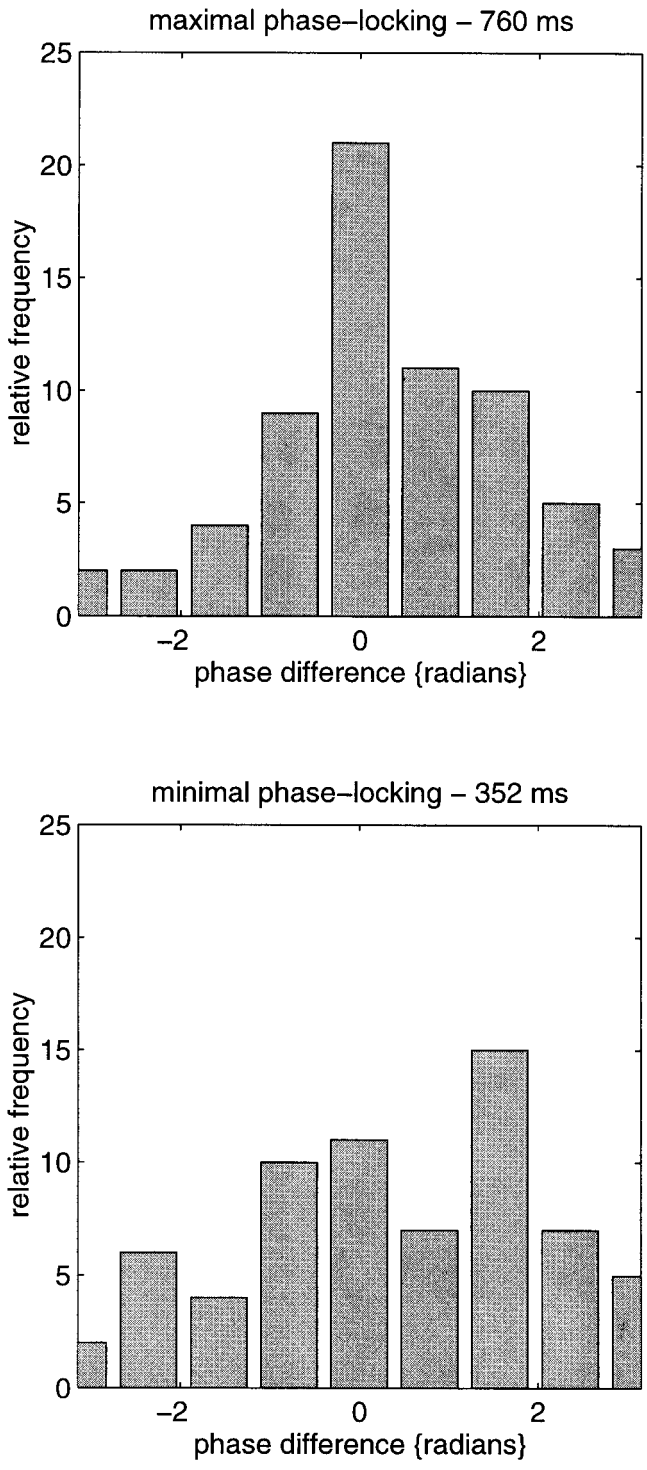


Figure 4.

Statistical inference. **Above:** Smoothed leading diagonals of the crosscorrelation matrices for epochs that were (solid line) and were not (broken line) time-locked to EMG onset. **Below:** Corrected *P* values testing the alternative hypothesis that the normalized correlations (solid line, above) were different from those predicted under the null hypothesis of no modulation. This is a plot of p(t) in the text. The broken horizontal line corresponds to $P < 0.05$.

specific dynamic correlations, this highlights the usefulness of the J-PSTH. The crosscorrelation matrix comprising the J-PSTH includes the crosscorrelations at all lags and will therefore provide evidence for correlations, irrespective of the underlying phase difference.



Spike trains vs. MEG

The answer to the question, "Are phase-locking and dynamic correlations the same thing?" is a qualified yes. The qualification is that the *dynamic correlations have to be frequency-specific*. It should be noted that the J-PSTH analyses applied to multiunit recording data [e.g., Vaadia et al., 1995] used all the frequency components of binned firing rate data. The fact that these data represent sparse point processes may preclude the sort of frequency-specific J-PSTH presented here. The possibility that the sorts of dynamic correlations presented in Vaadia et al. [1995] can be attributed to phase-locking is unlikely, because the crosscorrelograms and crosscorrelation matrices do not show any periodic structure. Such periodicity would be expected if the correlations were a result of systematic phase relationships at a particular frequency. This periodic structure is expected, because at a certain lag between the two signals they must be $\pi/2$ out of phase and the correlation must be zero. With increasing lag, the phase difference will fall and rise periodically with concomitant increases and decreases in the correlation (compare this to coherence that is not sensitive to the phase difference). However, when it comes to continuous electrical or biomagnetic data, we hope to have illustrated an important link between frequency-specific dynamic correlations and transient phase-locking.

CONCLUSIONS

In conclusion, we can assert that frequency-specific dynamic correlations and transient phase-locking are the same, using a purely theoretical analysis; and that this phenomenon can be demonstrated in human biomagnetic signals. Dynamic correlations that do not show frequency specificity (no periodic modulation

Figure 5.
Distribution of phase differences over epochs. **Top:** Distribution of phase differences between the prefrontal and parietal time-series at 760 msec (i.e., when correlations were expressed the most). This distribution was based on the data used in Figure 2. **Bottom:** Equivalent distribution at 352 msec. Note that both distributions suggest a tendency to phase-locking, with the distribution associated with greater correlations (top) being slighter "tighter" (i.e., more phase-locking).

of the crosscorrelation functions) may or may not be associated with transient phase-locking at high frequencies and can be more generally framed in terms of the conjoint expression of neuronal transients with a wide range of frequency components [Friston, 1995].

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APPENDIX

Relationship between distribution of phase differences and coherence

Let the activity of neuronal processes i (e.g., discharge rates in a separable spike-train or biomagnetic signal in a MEG channel), at a particular time in the postevent period, be $x_i(t)$, where the Fourier transform pair

$$x_i(t) = \frac{1}{2\pi}\int s_i(\omega)\cdot e^{-i\omega t}\,d\omega \qquad \text{(A1a)}$$

$$s_i(\omega) = \int x_i(t)\cdot e^{i\omega t}\,dt \qquad \text{(A1b)}$$

provides representations of the activities in time t and frequency space ω. These equations are generally applicable to any time-series. Equation (A1a) says that the time-series can be represented as the superposition of many frequency components distributed according to the complex vector $s_i(\omega)$ that is the Fourier transform of $x_i(t)$. $s_i(\omega)$ can be equivalently expressed in terms of its absolute value α_i and phase ϕ_i where $s_i(\omega) = \alpha_i \exp(i\phi_i)$. α_i corresponds to the magnitude of the frequency component at ω and ϕ_i its phase. We are interested in characterizing coherence in terms of the phase relationship. Let the two processes have spectral representations:

$$\begin{aligned} s_i(\omega) &= \alpha_i \exp(i\phi_i) \\ s_j(\omega) &= \alpha_j \exp(i\phi_i + \delta) \end{aligned} \qquad \text{(A2)}$$

where δ is the phase-difference between processes i and j. Coherence is based on the crossspectral density $g_{ij}(\omega) = \langle s_i(\omega)s_j(\omega)^*\rangle = \langle \alpha_i\alpha_j \exp(-i\delta)\rangle$. Here $\langle\rangle$ denotes expectation over epochs or realizations of the processes and * the complex conjugate. The coherence is given by:

$$C = \frac{|g_{ij}(\omega)|^2}{g_{ii}(\omega)g_{jj}(\omega)} = \frac{\langle\alpha_i\alpha_j\rangle^2}{\langle\alpha_i^2\rangle\langle\alpha_j^2\rangle}\left[\left(\int_{-\pi}^{\pi} P(\delta)\cos(\delta)\,d\delta\right)^2 + \left(\int_{-\pi}^{\pi} P(\delta)\sin(\delta)\,d\delta\right)^2\right]. \qquad \text{(A3)}$$