

Gamma coherence and conscious perception

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Abstract—Background: High-frequency (e.g., gamma 30 to 50 Hz) coherent neural activity has been postulated to underlie binding of independent neural assemblies and thus integrate processing across distributed neuronal networks to achieve a unified conscious experience. Prior studies suggest that gamma activity may play a role in perceptual mechanisms, but design limitations raise concerns. Thus, controversy exists as to the hypothesis that gamma activity is necessary for perceptual awareness. In addition, controversy exists as to whether the primary sensory cortices are involved directly in the mechanisms of conscious perception or just in processes prior to conscious awareness. **Objective:** To investigate the relation of gamma coherence and perception. **Methods:** Digital intracranial electrocorticographic recordings from implanted electrodes were obtained in six patients with intractable epilepsy during a simple somatosensory detection task for near-threshold stimuli applied to the contralateral hand. Signal analyses were then conducted using a quantitative approach that employed two-way Hanning digital bandpass filters to compute running correlations across pairs of channels at various time epochs for each patient and each perception state across multiple bandwidths. **Results:** Gamma coherence occurs in the primary somatosensory cortex approximately 150 to 300 milliseconds after contralateral hand stimuli that are perceived, but not for nonperceived stimuli, which did not differ in character/intensity or early somatosensory evoked potentials. **Conclusion:** The results are consistent with the possible direct involvement of primary sensory cortex in elemental awareness and with a role for gamma coherence in conscious perception.

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The canonical example of the “binding problem” is the question of how the brain associates and integrates one visual feature (e.g., shape) of an object with other visual features (e.g., location).¹ However, the term “binding problem” is actually used for a class of problems representing different phenomena, which concern our capacity to integrate information across time, space, attributes, modalities, perceptions, or ideas.¹ It is likely that binding occurs in many different kinds of brain processes and may represent a diverse set of functions. One proposed solution to the binding problem is the temporal correlation or neural synchronization hypothesis, which posits that binding is achieved through the formation of dynamic cell assemblies based on selective synchronization of distributed neural activities.^{2–6} There is supportive but not incontrovertible evidence for this hypothesis.^{6,7} Perhaps the most challenging binding problem involves understanding the mechanisms underlying conscious awareness. A theory involving synchronous neural activity has been postulated to explain consciousness.⁸

Attention and binding are intimately related.⁹ Focused attention has been found to modulate synchronized neuronal firing in primate somatosensory and visual cortices.^{10,11} Electroencephalographic activity in the gamma range (i.e., 35 to 80 Hz) has been associated with attentional mechanisms for several decades^{12,13} and has been postulated to be critical in information processing.^{4,14,15} Synchronized activity in the gamma range has been hypothesized to underlie conscious awareness by integrating neural activity across different cerebral areas and may form the neural correlate of conscious awareness.^{8,16} Although animal and human studies have provided some support for this hypothesis, the relationship of gamma coherence to cognitive tasks in humans requiring conscious perception remains controversial.^{17,18}

Conscious perception is an intrinsically subjective state. Thus, investigations will ultimately require humans to ensure that conscious awareness has occurred. For example, correct motor responses can be made to unperceived stimuli.¹⁹ Gamma coherence during perceptual tasks has been reported in a few human studies using scalp-recorded EEG or magneto-EEG (MEG).^{20–24} However, the results do not present a uniform pattern. Marked topographic and temporal differences exist that cannot be ex-

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See also pages 800 and 841

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plained by task differences. For example, some MEG studies have reported continuous gamma activity in the awake state involving the entire head with an anterior-to-posterior phase lag,^{20,21} but a similar pattern is absent from scalp EEG studies. In addition, the patterns of gamma coherence in some studies appear at odds with known anatomic behavioral correlates. For example, a recent study reported scalp EEG gamma coherence in central and left cerebral regions for a facial recognition task.²⁴ However, this pattern is not consistent with other studies of face perception, which have implicated bilateral inferior occipitotemporal (right > left) regions, especially the fusiform gyrus.²⁵⁻³⁰ Scalp EEG and MEG recordings may also be contaminated by muscle artifact, which could obscure cortical gamma or be inaccurately attributed to the cortex.

Only a few studies of gamma activity have been conducted in humans using intracranial electrocorticographic (ECoG) recordings. Several of these studies have found gamma differences across behavioral states or gamma responses to various behavioral tasks.^{17,31-35} Gamma activity may dissociate from other bandwidths,³²⁻³³ and the topographic distributions of gamma are more discrete and more consistent with traditional functional anatomy than similar maps for other bandwidths.³² Although gamma coherence has been reported over long distances in some scalp studies, consistent findings have not been found from intracranial studies in humans. Two studies examining ECoG gamma coherence in humans found gamma coherence only over short distances.^{17,33} A third study found phase-lagged coherence in relation to the fusiform gyrus for facial recognition, but the pattern differed from MEG and EEG studies.²⁸

Perhaps most critically, none of the prior scalp or intracranial gamma studies has employed a behavioral task that addresses whether the gamma activity is linked to the conscious component of the perception. Typically, prior studies have involved perceptual tasks requiring discrimination of stimuli into different categories. However, irrespective of which category is perceived, all of the stimuli in these tasks are perceived. Thus, the crucial experiment has not been conducted, which compares ECoG responses for stimuli of identical sensory content, but polar differences in conscious content (i.e., perceived versus nonperceived). Therefore, we proposed to test the gamma coherence hypothesis by comparing ECoG epochs for perceived versus nonperceived stimuli during a simple detection task of near-threshold somatosensory stimuli.

Another hypothesis concerning the neural correlates of awareness posits that the primary sensory cortex is not directly involved in the activity related to conscious awareness.³⁶ Supporting this notion, it is clear that neural processing can occur in the primary sensory cortex for stimuli that are not perceived.^{37,38} Further, a few patients with severe damage to primary sensory cortex are able to per-

ceive at least crude stimuli in the related sensory modality.^{39,40} In addition, patients with lesions to the primary visual cortex can exhibit residual visual function in the absence of conscious awareness; this phenomenon is called "blindsight."⁴¹ However, it has been argued that blindsight depends on vestiges of geniculostriate function and that patients are unaware of visual stimuli because these islands of neural activity are isolated from the integrated network of neural function.⁴¹

Suppose that two sets of simple sensory stimuli have identical character and intensity, but one set is perceived and the other is not. If the two sets generated identical early components of the ECoG response in the primary sensory cortex, it would demonstrate that both stimuli were conducted to the primary sensory cortex and underwent identical initial processing. If the primary sensory cortex were not involved directly in the mechanisms of conscious perception, then one would not expect that later components of the ECoG in the primary sensory cortex would differ based on whether the stimuli were perceived or not. We proposed to test whether the primary sensory cortex is directly involved in conscious awareness by determining if consistent differences in gamma coherence exist for perceived versus nonperceived stimuli in the primary sensory region during a simple somatosensory detection task of identical stimuli near threshold.

The exact time at which conscious perception occurs is uncertain, but it must be delayed beyond 50 to 100 milliseconds as stimuli are particularly susceptible to masking by a competing stimulus, which is given 50 to 100 milliseconds after the target stimulus.⁴² In fact, masking is greater during this period than if the mask is given simultaneously with the target stimulus. It has been suggested that perception may occur as late as 300 to 500 milliseconds after stimulus because extending the train duration of repetitive stimuli up to 300 to 500 milliseconds lowers perceptual threshold, but longer train durations do not appear to further lower the threshold.^{43,44} As the above studies employed simple somatosensory detection tasks, one might predict that perception in such tasks occurs at approximately 100 to 300 milliseconds after target stimulus. If gamma coherence is involved in perception, then it should occur near this time range for perceived stimuli but be absent for nonperceived stimuli. In summary, we predicted that perceived stimuli would be consistently associated with gamma coherence and that the coherence would exhibit both temporal and topographic specificity.

Methods. *Subjects.* Six patients (five men, one woman; mean age 26 years, range 19 to 31 years) with intractable epilepsy underwent intracranial electrode implantation for strictly clinical indications to localize the seizure focus for possible epilepsy surgery. Prior to intracranial implantation, all patients underwent a detailed clinical evaluation including a thorough review of the history, general physi-

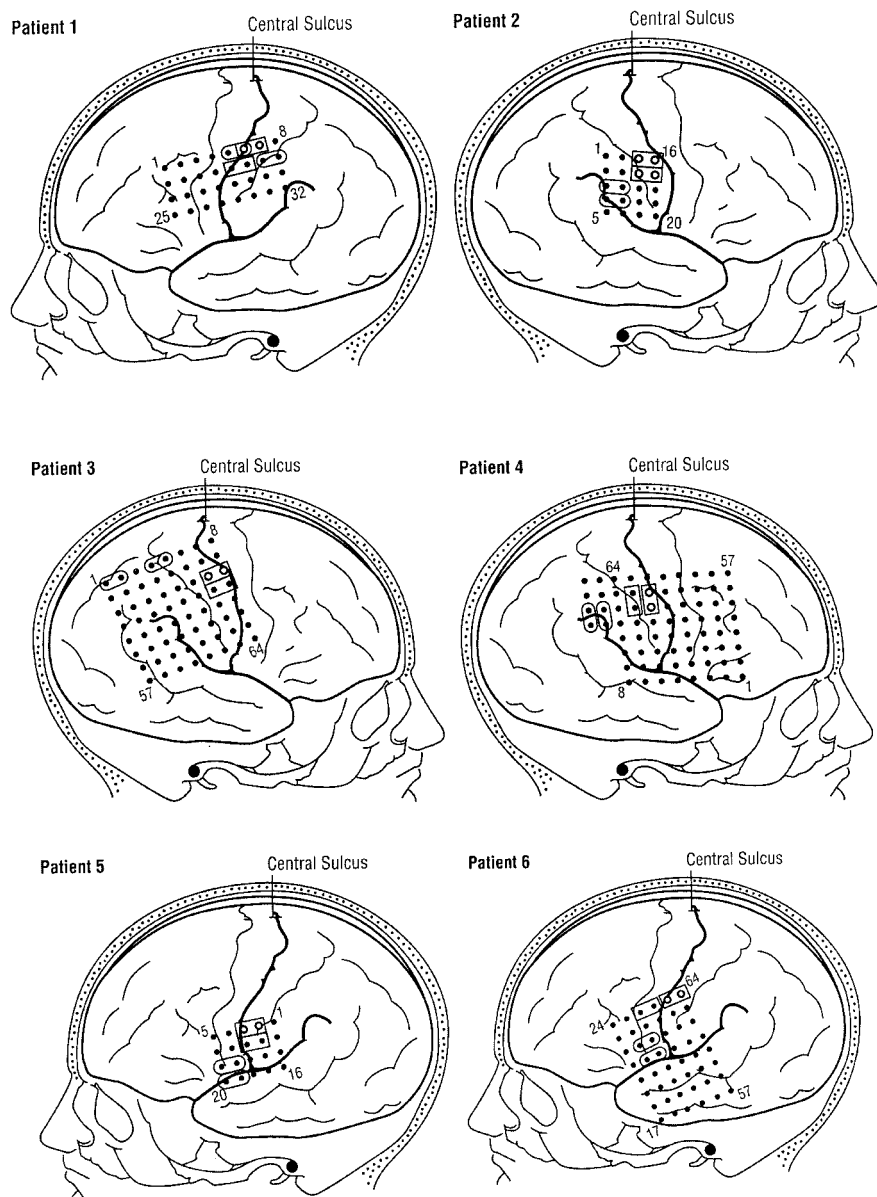


Figure 1. Diagrams for grid electrode locations in the six patients. Open circle electrodes denote location of the primary evoked potentials for the stimulated contralateral hand. Electrode pairs enclosed by rectangular boxes in each patient's grid denote the two pairs of electrodes in the primary somatosensory region, which exhibited gamma coherence to perceived stimuli as depicted in figure 5. (See figures E2 and E3 on the Neurology Web site; go to www.neurology.org.) Electrode pairs enclosed by ovals denote two pairs of electrodes tested outside the primary somatosensory hand region.

cal and neurologic examination, prolonged video-EEG monitoring, brain MRI, neuropsychological assessment, cerebral angiography, and intracarotid amobarbital testing. Patients were implanted with one or a combination of intracranial subdural grid, epidural, or depth electrodes. Electrode location varied across patients based on their specific clinical needs.

Recordings. Studies were conducted between the 3rd and 7th postoperative day. The recordings in the current study were limited to the subdural grids, which consisted of titanium electrodes (5 mm in diameter) with center-to-center distances of 10 mm. Grid locations are depicted in figure 1. Note that only a portion of the electrode contacts were available for analysis in this study owing to underlying lesion, epileptiform activity, artifacts, and limitations in channel collection for computer analysis. Convergent verification of electrode positions was obtained using several techniques. Direct visualization of the grids placed on the surface of the brain was done at the time of implantation and removal. Photographs were taken and graphical notes made concerning the relationship of each electrode

position to identifiable cortical landmarks. Skull radiographs were taken immediately postoperatively, and CT scans were obtained after implantation. In addition, the location of the cortical representation of the stimulated finger was verified by recording somatosensory evoked potentials from the grid electrodes.

Intracranial electrode locations for the six patients are depicted in figure 1. ECoG epochs (750 to 1,000 milliseconds) time-locked to the stimulus presentation were collected. The ECoG was amplified ($5 \times 1,000$), sampled at 256 to 683 Hz, and filtered at 0.01 to 100 Hz with 6 dB/octave and a 60-Hz notch. The ECoG data were stored digitally for off-line review and processing, which included digital resampling at 512 Hz. Electrodes with interictal epileptiform discharges or abnormal background activity were excluded to approximate normal brain as closely as possible. Because of these factors and technical problems, epochs from only a small number of electrodes outside the primary somatosensory hand region were examined.

Procedures. After cleansing the skin surface with alcohol, two 20-mm pregelled disposable stimulating electrodes

were placed 1 cm apart on the medial surface of the index finger contralateral to the implanted grid. The distal electrode was attached to the anode of the stimulator and the proximal electrode to the cathode. A ground electrode was attached to the back of the right shoulder. Impedances were checked at the beginning and end of the experimental session, and resistance was maintained at or below 5 k Ω . The stimulus pulse duration was 100 microseconds. The subject was sitting upright in bed with each arm resting on the ipsilateral side. The investigator was seated in front of the subject, who was asked to remain still and relax with eyes closed. Prior to ECoG collection, the perceptual threshold for the target finger was determined by the method of limits using progressive titration as described elsewhere.⁴² During ECoG collection, stimuli were presented randomly at 5- to 20-second intervals. Approximately 50% of stimuli are perceived when presented in this manner at an intensity about 0.5 mA above the threshold determined by the method of titration.⁴² Stimuli were presented initially at this level and then adjusted as needed to achieve an approximate 50:50 ratio of perceived to nonperceived. The number of ECoG epochs analyzed at any intensity for perceived and nonperceived stimuli were matched. The total number of epochs analyzed was 10 to 50/subject (median = 36), half of which were perceived.

Signal analyses. The digital ECoG data were analyzed to test our experimental hypothesis that perceived stimuli would be consistently associated with gamma coherence. ECoG for perceived stimuli were compared with nonperceived stimuli, which were otherwise identical, assessing the presence, timing, and topography of time-linked gamma coherence using a frequency-adapted running correlation analysis technique. Shifts and drifts were first digitally removed from raw event-related ECoG epochs by detrending (removing best linear fit along each time sweep). Whole-band event-related potentials for each patient, perception state (perceived versus nonperceived), and channel pair designation (primary sensory area versus outside) were obtained by averaging across trials ($n = 14$, 8, 5, 22, 25, 25 perceived for the six patients, with these numbers matched for nonperceived). The event-related potentials were decomposed into four frequency bands (alpha = 8–12, beta1 = 12–25, beta2 = 25–35, and gamma = 35–45 Hz) via digital two-way Hanning bandpass filters. A forward and backward pass through each filter results in zero phase distortion and magnitude response equal to the square of the single-pass filter's response. The main step involved computation of the running correlations (RC) of these decomposed average event-related potentials across pairs of channels across time for each patient and each perception state across the multiple bandwidths (figure 2). (Detailed descriptions of these computations and the statistical analyses are available on the *Neurology* Web site; see the appendix of the online version of this article.)

Results. The grand mean whole-band somatosensory evoked potentials for perceived and nonperceived epochs are depicted in figure 3. Note that the early components were similar for perceived and nonperceived epochs, demonstrating that both underwent similar initial processing. Although visual inspection suggests differences in the later components, statistical comparisons of the somatosensory evoked potentials for perceived versus nonperceived epochs revealed no significant differences (see figure 3). Locations

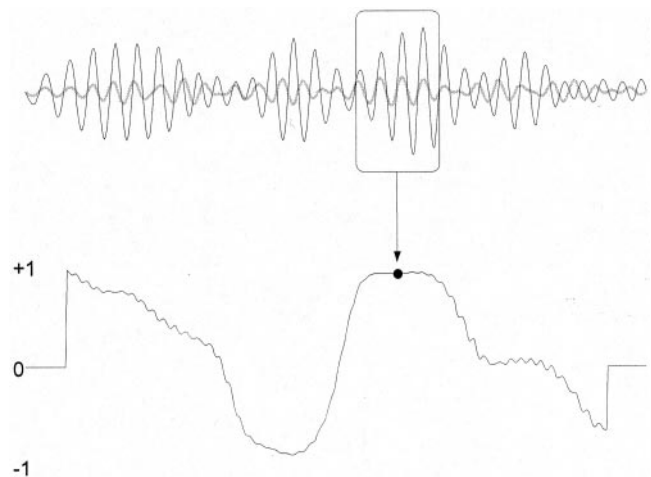


Figure 2. Example of running correlation function over time ($RC[t]$) in the bottom panel for a 1-second epoch of simulated data for a signal pair (top panel; thin and bold traces) at a center frequency of 30 Hz. For a sampling frequency of 512 Hz, the length of the rectangular sliding window is 68 points ($T_w = 133$ ms). This window is shown at an instant during which there is near-perfect synchronization (bottom panel; $RC = 0.93$).

of the early somatosensory evoked potentials in relation to the ECoG experimental recordings are depicted in figure 1. In the first two patients in this study, direct stimulation of the cortex via the implanted electrodes was also conducted for clinical functional mapping. Stimulation of the same electrodes that exhibited the early evoked somatosensory potentials produced paresthesias in the hand that had been stimulated during our ECoG recordings, similar to a prior study.³⁸

For illustrative purposes, the average event-related potentials of ECoG filtered at various frequency windows are depicted for Patient 1 in figure 4. The two channels are from the primary somatosensory region of the stimulated hand. Note the synchrony around 200 milliseconds for the gamma band.

Figure 5 depicts the perceived minus nonperceived difference in grand mean running correlation along with the statistical 95th and 5th percentile CI. Note the significant increase in gamma coherence for the perceived condition at approximately 170 to 270 milliseconds for channel pairs in the primary somatosensory region. (See figure E1 on the *Neurology* Web site; go to www.neurology.org.) This increase in gamma coherence between 100 and 300 milliseconds for perceived stimuli was absent from electrode pairs outside the primary somatosensory region for the stimulated hand. (see figures E2 and E3 on the *Neurology* Web site; go to www.neurology.org.) Note that the apparent increase in gamma coherence around 400 milliseconds is due to a marked negative-phase coherence to nonperceived stimuli.

Discussion. Gamma coherence occurred reliably in the primary somatosensory area when simple somatosensory stimuli were perceived. The absence of this coherence for nonperceived stimuli cannot be explained by differences in the characteristics or intensity of the stimuli. Further, both perceived and

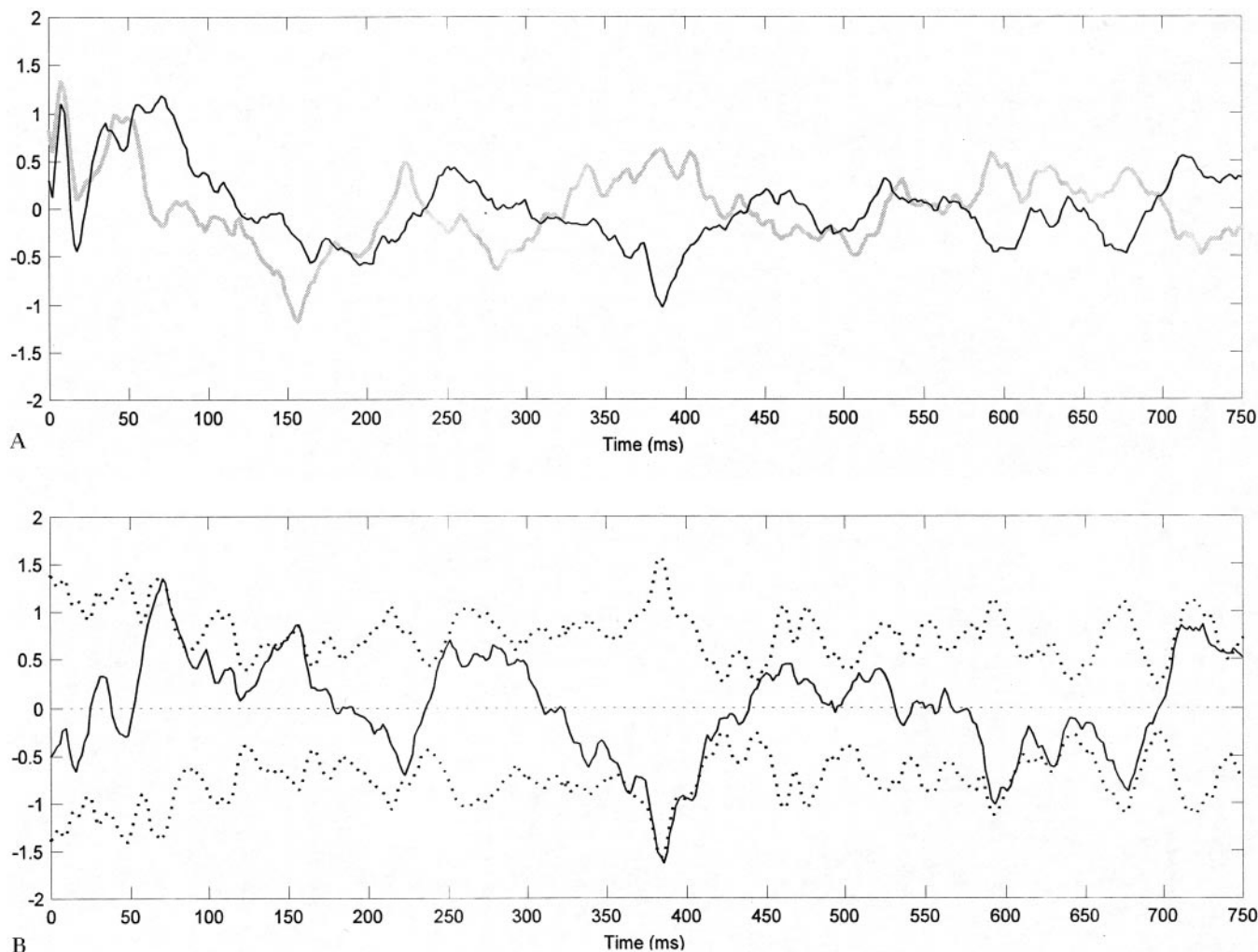


Figure 3. (A) Grand averages across the six patients for normalized somatosensory evoked potentials (SEP) from the primary somatosensory region of the stimulated contralateral hand for perceived (thin black line) and nonperceived (broad gray line) epochs. Normalized Z scores for SEP are shown on the y axis. (B) Grand mean difference SEP (solid line) for perceived minus nonperceived epochs. Upper and lower dotted curves represent latency-dependent statistical threshold levels (i.e., 95th and 5th percentile confidence boundaries) as calculated by Fisher's permutation test. Note that the difference curve for perceived minus nonperceived SEP never surpasses levels significantly different than that expected from normal fluctuations.

nonperceived stimuli appeared to undergo similar initial processing in the primary somatosensory cortex, given that they had similar primary evoked potentials. This finding is consistent with prior studies that have recorded primary somatosensory evoked potentials to stimuli below perceptual threshold.^{37,38}

The observed gamma coherence to perceived stimuli in the current study exhibited both temporal and topographic specificity. The temporal window was consistent with our a priori hypothesis based on prior studies. Topographically, conscious awareness of the elemental form of simple somatosensory stimuli was associated with this specific neurophysiologic activity in the primary somatosensory area. Like other forms of cognitive processing, we expect that the mechanisms of conscious awareness are distributed over a neural network and that the network varies from moment to moment depending on the stimuli and concepts that access consciousness. In

contrast to awareness of simple elemental form, the network for awareness of symbolic content or of more complex stimuli must involve other regions, and the timing of perceptual awareness is probably later and more variable owing to the increased processing demands. Detection of these more time-variable responses would require averaging RC for single trials rather than calculating RC for the average event-related potentials. In addition, the networks for awareness of more complex percepts may not directly involve the primary sensory cortex. For example, a recent ECoG study found face-selective gamma coherence at 160 to 230 milliseconds after stimulus between the fusiform gyrus (which is essential for facial recognition) and multiple brain regions (i.e., temporal, parietal, and frontal cortices).⁴⁵ Phase lag increased with distance from the fusiform gyrus. In our study, we did not find consistent phase lag coherence at distant locations, but this may well be due to

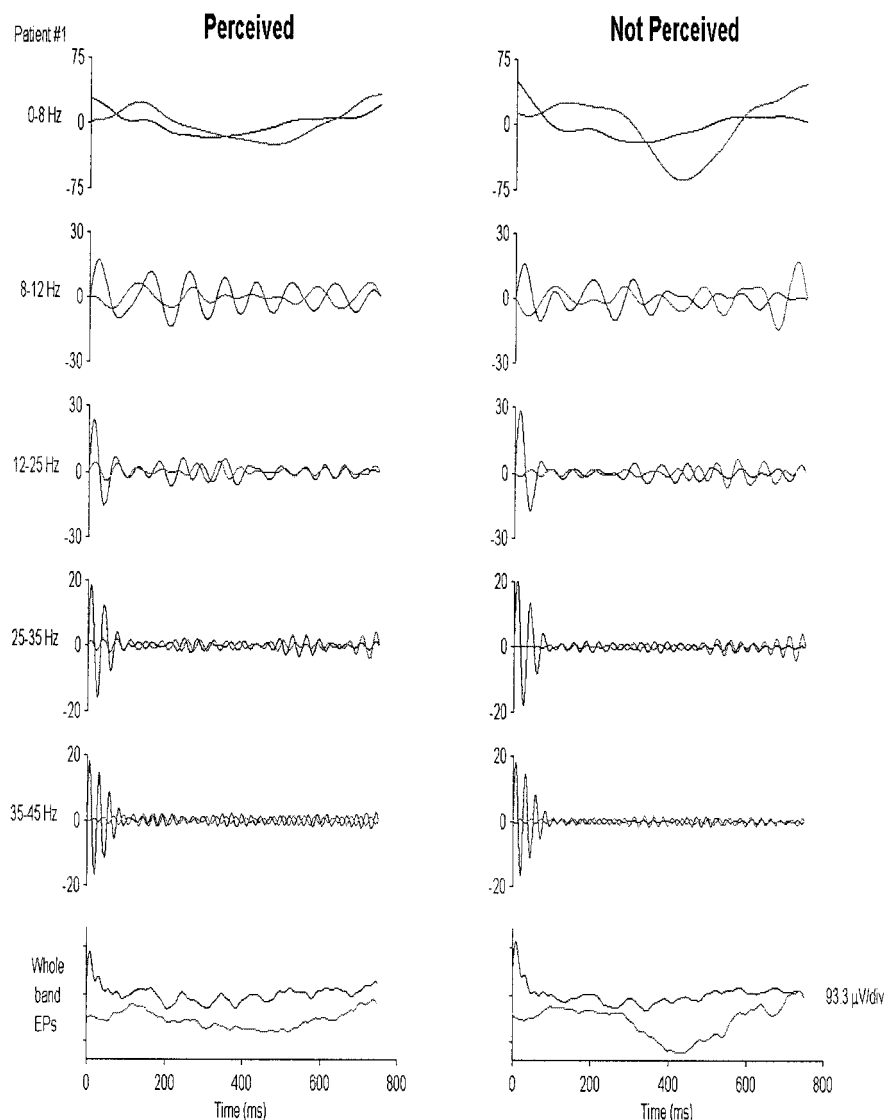


Figure 4. Individual intracranial electrocorticographic epochs were filtered at each of the six indicated bandwidths and the resultant single trials averaged to obtain event-related potentials for perceived and nonperceived stimuli in each bandwidth. This figure depicts the results for Patient 1 from two bipolar channels recorded from the primary somatosensory region of the stimulated hand. Note synchronization in the gamma range for perceived stimuli around 200 milliseconds.

limited and inconsistent areas available for intracranial recordings as electrode locations were determined for individual patients by clinical criteria. Further, the lack of a consistent phase in our study may be related to the use of subdural electrodes that might miss responses from cortex buried in the sulci.

In contrast to our a priori hypothesis for gamma coherence to perceived stimuli in the primary somatosensory hand region, the finding of negative-phase coherence around 400 milliseconds for the nonperceived condition was unexpected, and its significance is uncertain. Further, the limited number of recordings from regions outside the primary somatosensory hand area restricts conclusions as to this finding. However, the phenomenon was present in all six patients. Like our finding of positive gamma coherence to perceived stimuli, this finding will require replication.

Even if the gamma synchrony observed in the somatosensory region for perceived stimuli is necessary for conscious awareness of simple somatosensory stimuli, it is unlikely to be sufficient. We suspect that it is

associated with coherence in other structures such as the thalamus, which has been postulated to be involved in conscious mechanisms.⁴⁶ Consistent with this concept, thalamic stimulation can induce topographic focal gamma activity in the neocortex,⁴⁷ and cortical feedback has been shown to affect synchrony of oscillations in the thalamus.⁴⁸ In addition, we have recently demonstrated that loss of awareness during partial seizures is associated with increase blood flow in the thalamus or midbrain, suggesting that the loss of conscious awareness is the result of seizure spread to or diaschetic effects on these critical central structures.⁴⁹

As a statistical association does not offer causal inference, the gamma coherence associated with perceived stimuli in the current study cannot be conclusively ascribed to conscious perception. Gamma band synchronization may either play a functional role in conscious perception or simply reflect associated neuronal processes. Although the observed association is consistent with a role for gamma coherence in the mechanisms underlying conscious perception, more than simple coherence must be involved be-

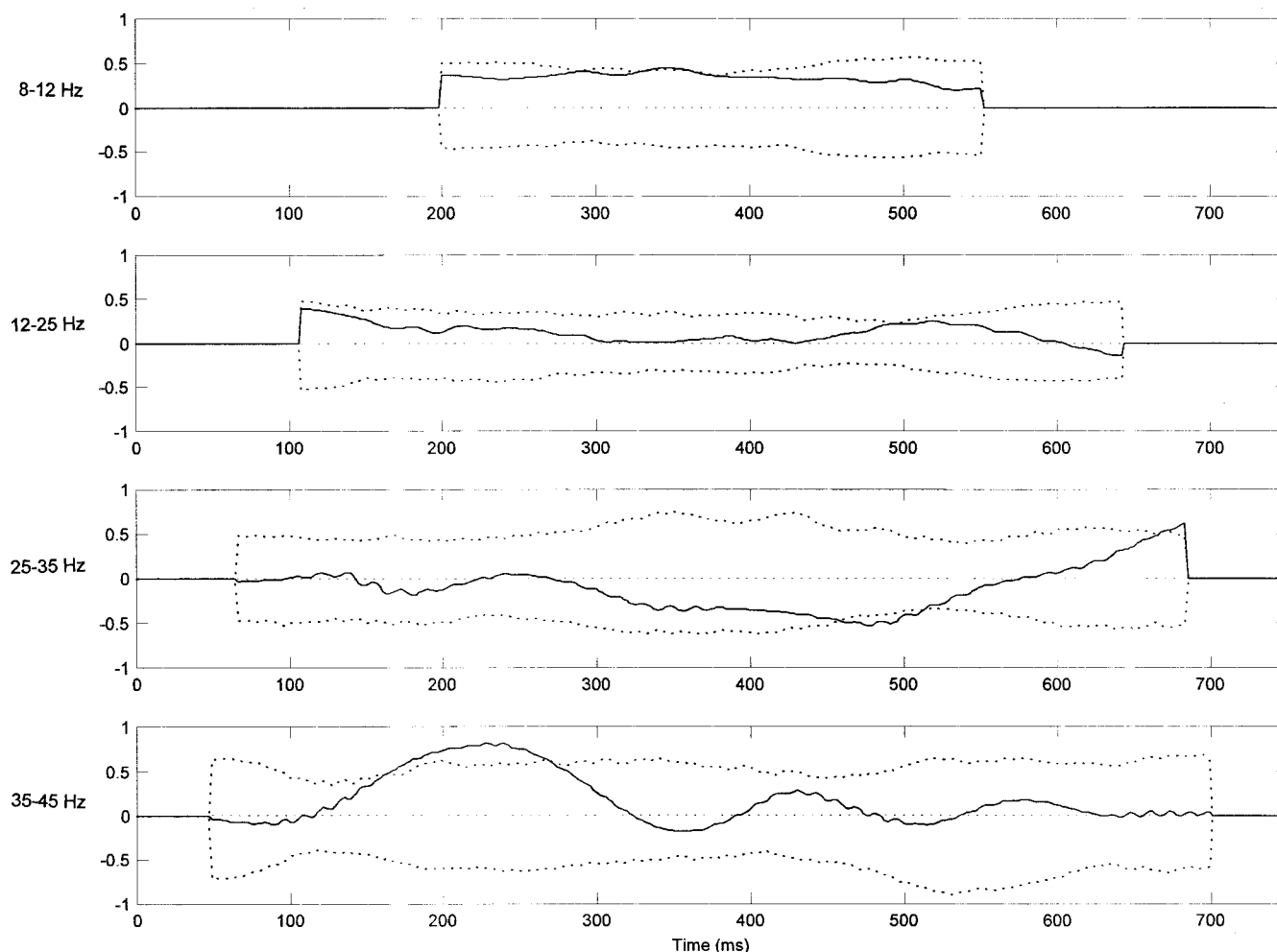


Figure 5. Perceived minus nonperceived grand mean differences for the running correlations across the six patients at four frequency ranges from a channel pair at primary somatosensory region (1° SS) for the stimulated hand. The dotted curves represent the latency-dependent upper and lower statistical threshold levels (i.e., 95th and 5th percentile confidence boundaries) as calculated by Fisher's permutation test. The running correlation curve is significant when it exceeds the dotted curve. Note significant gamma coherence for perceived stimuli at approximately 170 to 270 milliseconds.

cause patients may exhibit EEG coherence during seizures that impair consciousness. Another example that simple coherence is not adequate for consciousness is the observation that gamma coherence has been demonstrated between the brain (EEG) and muscle (electromyography) during movements.⁵⁰

Nevertheless, our findings may offer insight into the neural mechanisms associated with conscious perception. The consistent association of spatially and temporally specific gamma coherence to perceived stimuli (and its absence for nonperceived stimuli) suggests that it does play a role. Further, the findings suggest that the primary sensory cortex may be directly involved in the mechanisms of conscious perception when detection of simple elemental form is required. The implication from prior studies that the primary sensory cortex is not involved in conscious awareness may be due to the fact that these studies involved more complex stimuli or concepts, which would be expected to involve other brain regions at higher processing levels.

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References

1. Roskies AL. The binding problem. *Neuron* 1999;24:7–9.
2. Milner P. A model for visual shape recognition. *Psychol Rev* 1974;81:521–535.
3. Von der Malsburg C. The correlation theory of brain function. MPI biophysical chemistry, internal report 81-2. Reprinted in Domany E, Van Hemmen JL, Schulten K, eds. *Models of neural networks II*. Berlin: Springer, 1994.
4. Engel AK, König P, Kreiter AK, Schillen TB, Singer W. Temporal coding in the visual cortex: new vistas on integration in the nervous system. *Trends Neurosci* 1992;15: 218–226.
5. Bressler SL. Large-scale cortical networks and cognition. *Brain Res Rev* 1995;20:288–304.
6. Gray CM. The temporal correlation hypothesis of visual feature integration: still alive and well. *Neuron* 1999;24:31–47.
7. Shadlen MN, Movshon JA. Synchrony unbound: a critical evaluation of the temporal binding hypothesis. *Neuron* 1999; 24:67–77.
8. Crick F, Koch C. Towards a neurobiological theory of consciousness. *Neuroscience* 1990;2:263–275.

9. Wolfe JM, Cave KR. The psychophysical evidence for a binding problem in human vision. *Neuron* 1999;24:11–17.
10. Steinmetz PN, Roy A, Fitzgerald PJ, Hsian SS, Johnson KO, Neibur E. Attention modulates synchronized neuronal firing in primate somatosensory cortex. *Nature* 2000;404:187–190.
11. Fries P, Reynolds JH, Rorie AE, Desimone R. Modulation of oscillatory neuronal synchronization by selective visual attention. *Science* 2001;291:1560–1563.
12. Sheer DE. Focused arousal and 40 Hz EEG. In: Knight RM, Bakker DJ, eds. *The neuropsychology of learning disabilities*. Baltimore: University Park Press, 1976:71–87.
13. Sheer DE. Focused arousal and 40 Hz EEG. In: Rockstroh ET, Lutzenberg W, Birbaumer N, eds. *Self-regulation of the brain and behavior*. Berlin: Springer, 1984:64–84.
14. Bressler SL. The gamma wave: a cortical information carrier? *Trends Neurosci* 1990;13:161–162.
15. Singer W. Synchronization of cortical activity and its putative role in information processing and learning. *Annu Rev Physiol* 1993;55:349–374.
16. Crick F, Koch C. Some reflections on visual awareness. *Cold Spring Harbor Symp Quant Biol* 1990;55:953–962.
17. Menon V, Freeman WJ, Cuttillo BA, et al. Spatio-temporal correlations in human gamma band electrocorticograms. *Electroencephalogr Clin Neurophysiol* 1996;98:89–102.
18. Herrmann CS, Mecklinger A, Pfeifer E. Gamma responses and ERPs in a visual classification task. *Clin Neurophysiol* 1999;110:636–642.
19. Taylor JL, McCloskey DI. Selection of motor responses on the basis of unperceived stimuli. *Exp Brain Res* 1996;110:62–66.
20. Ribary U, Ionnides AA, Singh KD, et al. Magnetic field tomography of coherent thalamocortical 40-Hz oscillations in humans. *Proc Natl Acad Sci USA* 1991;88:11037–11041.
21. Joliot M, Ribary U, Llinàs R. Human oscillatory brain activity near 40 Hz coexists with cognitive temporal binding. *Proc Natl Acad Sci USA* 1994;91:11748–11751.
22. Desmedt JE, Tomberg C. Transient phase-locking of 40 Hz electrical oscillations in prefrontal and parietal human cortex reflects the process of conscious somatic perception. *Neurosci Lett* 1994;168:126–129.
23. Tomberg C, Desmedt JE. Human perceptual processing: inhibition of transient prefrontal-parietal 40 Hz binding at P300 onset documented in non-averaged cognitive brain potentials. *Brain Res* 1998;255:163–166.
24. Rodriguez E, George N, Lachaux JP, Martinerie J, Renault B, Varela FJ. Perception's shadow: long-distance synchronization of human brain activity. *Nature* 1999;397:430–433.
25. Haxby JV, Ungerleider LG, Clark VP, Schouten JL, Hoffman EA, Martin A. The effect of face inversion on activity in human neural systems for face and object perception. *Neuron* 1999;22:189–199.
26. DeRenzi E, Perani D, Carlesimo GA, Silveri MC, Fazio F. Prosopagnosia can be associated with damage confined to the right hemisphere—an MRI and PET study and a review of the literature. *Neuropsychologia* 1994;32:893–902.
27. Clark VP, Maisog JM, Haxby JV. fMRI study of face perception and memory using random stimulus sequences. *J Neurophysiol* 1998;79:3257–3265.
28. Halgren E, Baudena P, Heit G, Clarke JM, Marinkovic K. Spatio-temporal stages in face and word processing. I. Depth-recorded potentials in the human occipital, temporal and parietal lobes. *J Physiol (Paris)* 1994;88:1–50.
29. Watanabe S, Kakigi R, Koyama S, Kirino E. Human face perception traced by magneto- and electro-encephalography. *Brain Res Cogn Brain Res* 1999;8:125–142.
30. Klopp J, Halgren E, Marinkovic K, Nenov V. Face-selective spectral changes in the human fusiform gyrus. *Clin Neurophysiol* 1999;110:676–682.
31. Perez-Borja C, Tyce FA, McDonald C, Uihlein A. Depth electrographic studies of a focal fast response to sensory stimulation in the human. *Electroencephalogr Clin Neurophysiol* 1961;13:695–702.
32. Crone NE, Miglioretti DL, Gordon B, Lesser RP. Functional mapping of human sensorimotor cortex with electrocorticographic spectral analysis II. Event-related synchronization in the gamma band. *Brain* 1998;121:2301–2315.
33. Gross DW, Gotman J. Correlation of high-frequency oscillations with the sleep–wake cycle and cognitive activity in humans. *Neuroscience* 1999;94:1005–1018.
34. Aoki F, Fetz EE, Shupe I, Lettich E, Ojeman GA. Increased gamma-range activity in human sensorimotor cortex during performance of visuomotor tasks. *Clin Neurophysiol* 1999;110:524–537.
35. Lachaux JP, Rodriguez E, Martinerie J, Adam C, Hasboun D, Varela FJ. A quantitative study of gamma-band activity in human intracranial recordings triggered by visual stimuli. *Eur J Neurosci* 2000;12:2608–2622.
36. Crick F, Koch C. Are we aware of neural activity in primary visual cortex? *Nature* 1995;375:121–123.
37. Libet B, Alberts WW, Wright EW, Feinstein B. Responses of human somatosensory cortex to stimuli below threshold for conscious sensation. *Science* 1967;158:1597–1600.
38. Ray PG, Meador KJ, Smith JR, Wheless JW, Sittenfeld M, Clifton GL. Physiology of perception: cortical stimulation and recording in humans. *Neurology* 1999;52:1044–1049.
39. Bottini G, Paulesu E, Sterzi R, et al. Modulation of conscious experience by peripheral sensory stimuli. *Nature* 1995;376:778–781.
40. Engelien A, Huber W, Silbersweig D, et al. The neural correlates of “deaf-hearing” in man. Conscious sensory awareness enabled by attentional modulation. *Brain* 2000;123:532–545.
41. Fendrich R, Wessiger CM, Gazzangia MS. Speculations on the neural basis of islands blindsight. *Prog Brain Res* 2001;134:353–366.
42. Meador KJ, Ray PG, Day L, Ghelani H, Loring DW. Physiology of somatosensory perception: cerebral lateralization and extinction. *Neurology* 1998;51:721–727.
43. Libet B, Alberts WW, Wright EW, Delattre LD, Levin G, Feinstein B. Production of threshold levels of conscious sensation by electrical stimulation of human somatosensory cortex. *J Neurophysiol* 1964;27:546–578.
44. Libet B, Pearl DK, Moreledge DE, Gleason CA, Hosobuchi Y, Barbaro NM. Control of the transition from sensory detection to sensory awareness in man by the duration of thalamic stimulus. *Brain* 1991;114:1731–1757.
45. Klopp J, Marinkovic K, Chauvel P, Nenov V, Halgren E. Early widespread cortical distribution of coherent fusiform face selective activity. *Hum Brain Map* 2000;11:286–293.
46. Newman J. Thalamic contributions to attention and consciousness. *Conscious Cogn* 1995;4:172–193.
47. MacDonald KD, Fifkova E, Jones MS, Barth DS. Focal stimulation of the thalamic reticular nucleus induces focal gamma waves in cortex. *J Neurophysiol* 1998;79:474–477.
48. Bal T, Debay D, Destexhe A. Cortical feedback controls the frequency and synchrony of oscillations in the visual thalamus. *J Neurosci* 2000;20:7478–7488.
49. Lee KH, Meador KJ, Park YD, et al. Pathophysiology of altered consciousness during seizures: subtraction SPECT study. *Neurology* 2002;59:841–846.
50. Mima T, Hallett M. Corticomuscular coherence: a review. *J Clin Neurophysiol* 1999;16:501–511.