P1 and Traveling Alpha Waves: Evidence for Evoked Oscillations

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INTRODUCTION

The phenomenon of traveling alpha waves was reported early in electroencephalographic (EEG) research (e.g., Adrian and Yamagiwa 1935; Petsche and Marko 1955) and appeared in line with physiological investigations indicating that from pulsating neurons periodic waves of activity spread over the cortex (Adrian and Matthews 1934; Freeman 2004). Although traveling waves are well documented in the ongoing EEG (for reviews, see e.g., Hughes 1995; Nunez 2000; Nunez et al. 2001), they rarely have been investigated in the context of evoked activity. One of the few studies was performed by Lilly and colleagues, who found that auditory evoked potentials in cats spread in different directions from the focus of activity initiated by acoustical stimulation (Lilly and Cherry 1954). In a recent study, Alexander et al. (2006) documented a close correspondence between traveling waves and ERP latencies.

In this paper, we focus on P1 latency and the hypothesis that the P1 component behaves like an evoked, traveling alpha wave. This hypothesis is based on the following evidence, linking alpha and early evoked activity. First, we have observed that P1 latency is correlated with individual alpha frequency (IAF) in a sense that short latencies are associated with high IAF and long latencies with low IAF (Klimesch et al. 2004). Second, it was found that evoked oscillations—particularly in the alpha and theta frequency range—become synchronized (aligned) in absolute phase during small time windows that coincide with the latencies of the P1-N1 complex (Gruber et al. 2005; Mormann et al. 2005). We were able to demonstrate that the latencies of the P1 and N1 can be predicted at least in part by a phase alignment between different frequencies particularly in the alpha but also theta frequency range. Third, analyzing m:n phase coupling revealed that coupling between alpha and theta was largest during the time window of the P1-N1 complex (Schack et al. 2005). Fourth, growing evidence supports the oscillatory EEG model, suggesting that early evoked potentials (and the P1 in particular) are generated (at least in part) by a reorganization of phase and not by a superposition of an evoked component on ongoing oscillations with random phase (Barry et al. 2000; Basar 1999; Hanslmayr et al. 2007).

These findings suggest that the P1 may be described in terms of an evoked oscillation and raise an interesting question with respect to the topography of P1 latency differences. If the P1 is generated (at least in part) by oscillations in the alpha frequency range, two different mechanisms can be assumed. Either a more global principle is operating in the sense that phase reorganization occurs at the same time at all (relevant) topographies or phase reorganization occurs rather locally and distributed at different locations. Because P1 latency may vary well differ between recording sites, the question is what the functional meaning of latency differences is. Latency differences in the phase of an ongoing oscillation between recording sites are a well-known phenomenon and were described in terms of traveling waves (e.g., Nunez 2000).

By using a Stroop task, we investigate the question, whether P1 latency differences can also be described in terms of a traveling wave. The reason for using a Stroop task was motivated by the fact that this type of task requires a strong top-down strategy that involves ignoring the meaning of a word and focusing on ink color and the hypothesis that the P1 component—considered a manifestation of an evoked alpha wave—reflects the influence of early top-down processes (cf. Klimesch et al. 2007) (see also DISCUSSION).

METHODS

Subjects

After written informed consent, a sample of 21 subjects participated in the present experiment. The sample consisted of 16 females and 5 males (mean age: 24.9; range: 20–33 yr).

Design and stimulus material

A Stroop task with three different conditions (congruent, neutral, and incongruent color words) was performed. In the congruent condition, the color words red, green, or blue were printed in their
respective ink color. The neutral words were PECH (misfortune), SOLL (debit), and LECK (leak) printed in red, green, or blue ink (thus allowing 9 possible word-color pairings). The incongruent condition consisted of six different items (the word red printed in green or blue ink, green printed in blue or red ink, and blue printed in green or red). For each condition, a total of 120 trials was used. Stimulus duration was 1,000 ms. During the interstimulus interval (of 2000 ms), a fixation cross appeared on the screen. The stimuli were presented in four blocks of 90 trials at the location of the fixation cross. The three different conditions were presented in randomized order. Thus subjects did not know whether a congruent, neutral, or incongruent word would appear in the next trial. The stimuli where shown on a 19-in computer screen (70-Hz refresh rate) with a visual angle of 7.5 × 2.9°. Subjects were instructed to respond manually with one of three colored buttons to the ink color of the words and to ignore the meanings of the words. For data analysis, all three conditions were pooled because the P1/N1 component did not differ significantly (with respect to latency and amplitude) between the three conditions.

Electrophysiological recordings and analyses

Brain electric activity was recorded referentially against a reference placed at the nose tip by a 32-channel amplifier (SynAmps, Neuroscan) with a sampling frequency of 1,000 Hz. Prior to analysis EEG data were arithmetically re-referenced against the average of the two earlobes and down-sampled to 250 Hz. Data were acquired between 0.15 and 70 Hz using 29 Ag-AgCl electrodes at the scalp, placed according to the international 10–20 system. One additional electrooculographic (EOG) channel was used to record vertical eye movements. After reducing ocular artifacts with the Gratton and Coles procedure implemented in Brain Vision (Brain Vision Analyzer Software Brain Products GmBH), remaining artifacts were rejected by visual inspection of the data. Then, data were epoched in 2-s time intervals ranging from 1,000 ms preceding to 1,000 ms after stimulus onset. For all subjects ≥100 artifact-free epochs remained for statistical analysis (average number of analyzed trials per subject was 238).

Analysis of P1, N1, and P3 latencies

Latencies were determined by a semi-automatic peak detection for selected electrodes (P3, P4, P7, P8, Pz, Po3, Po4, O1 and O2). The P1 was defined as the most positive deflection in a time window ranging from 50 to 150 ms. The N1 was defined as the most negative deflection between 150 and 250 ms and the P3 as the most positive deflection between 250 and 350 ms.

Analysis of traveling waves

Two different methods were used to determine whether the P1 behaves as traveling wave. The first method analyses traveling speed between selected electrode sites. Because the shortest P1 latencies were observed for occipital sites (O1, O2) and the longest for Pz, traveling speed was calculated with respect to Pz. As illustrated in Fig. 1, the general idea is that only in the case of a systematic traveling direction, traveling speed will be significantly different from zero (cf. Fig. 1A in comparison to B). By using a correlational analysis, the second method determines whether the topographical pattern of P1 latency differences can be detected in single trials.

Traveling speed

The analysis of traveling speed is based on the following procedure. First, instantaneous phase was calculated for each time point (between −1,000 ms prestimulus and 1,000 ms poststimulus) and frequency bin (between 2 and 20 Hz; width: 0.5 Hz) for each single trial and subject. Within this time window, cumulative phase was calculated for each trial. The calculation of instantaneous phase is based on the Gabor expansion (Gabor 1946; Lachaux et al., 1999; Schack et al., 2001). It transforms a signal \( x(t) \) into a complex time-frequency signal \( \psi_f(t) \). The components of \( \psi_f(t) \)—the Gabor coefficients—form a matrix of size \((m \times n)\) where \( m \) is frequency (with a resolution of 0.5 Hz in the present case) and \( n \) time (with a resolution of 4 ms in the present case). Information about the phase angle for each frequency-time pair can be extracted from the complex coefficients of the matrix (Schack et al. 2001). Second, for eight selected electrodes (P3, P4, P7, P8, Po3, Po4, O1, and O2), the relative phase (i.e., phase difference) was calculated with respect to Pz for each trial (cf. Fig. 1A) and time point. As illustrated in Fig. 1B, the calculation of relative phase was done with respect to Pz as trailing site. Third, phase-differences were transformed to latency differences in ms by using Eq. 1

\[
ms = \frac{1000 \varphi}{360,000} \quad \text{Frequency in Hz: } \varphi \ldots \text{Phase in degrees (1)}
\]

Fourth, the distance (in mm) between each selected electrode site and Pz (as reference site) was divided by the respective latency difference to obtain travel speed in meter per seconds (m/s; cf. Fig. 1A). These data were then averaged over the eight selected electrode pairs to obtain an estimate of traveling speed for each single trial. As illustrated in Fig. 1B, depending on the direction, traveling speed is characterized by positive or negative values. Positive values indicate a posterior to anterior direction whereas negative values indicate an anterior to posterior direction. If a systematic traveling direction is lacking, positive and negative values tend to cancel each other. On the other hand, in the case of a systematic traveling direction, averaging removes unsystematic noise and gives an estimate of travel speed. Travel speed was calculated for each single trial and was then averaged for each subject.

Control analysis to assess the influence of time resolution

The P1 and N1 are rather closely spaced with a typical peak-to-peak latency of ~50 ms. Thus limited time resolution of the filter may cause a mutual contamination of phase estimates between P1 and N1. We have run a control analysis with exactly the same procedure as described above but with a high pass set at 3 Hz and filter parameter gamma set at 25π (instead of gamma = 1 as in the standard analysis). The filter parameter defines the time-frequency resolution of the Gabor filter. If the gamma value is high, good time resolution but poor frequency resolution is achieved (e.g., with gamma = 25π, time resolution is ± 40 ms, but frequency resolution is low). If gamma is set to a low value, the filter reveals good frequency but poor time resolution.

Correlational analysis

Relative phase (i.e., phase difference) was calculated with respect to Pz for each trial (time point and frequency bin) for the selected electrodes (P3, P4, P7, P8, Po3, Po4, O1, and O2) as described for the analysis of travel speed. Then relative phase was transformed to ms. This data vector, obtained for each trial and subject was correlated with the respective data vector of P1 and N1 latency differences. Then, the correlations were Fisher-z transformed and averaged over trials for each subject.

Statistical analysis

To evaluate whether there are constant latency shifts of the P1, N1, and P3 components, one-way ANOVAs were run with factor location (O1, O2, P3, P4,. . ., Pz). To investigate, whether the latency shifts follow a linear trend, polynomial contrasts were calculated.

For the statistical analysis of traveling speed, the obtained values were tested for deviations from zero by using one-sample t-test. This
was done for each frequency and time bin. To compensate for multiple testing, the $P$ level was set to 0.001 (2 tailed).

For the correlational analysis, a two-stage statistical test procedure was carried out. First, the Jarque-Bera test was calculated to test whether the $z$-transformed data are normally distributed. Second, in the case the data were normally distributed, $t$-test was calculated to determine whether the $z$-transformed correlation coefficients differed significantly from zero. To compensate for multiple testing, $P$ level was set to 0.001. The $z$ values, selected by this two-stage procedure, were averaged over subjects and are depicted in Fig. 3, B and C.

Finally, $F$ tests were used to determine whether the variances of the P1 and N1 latencies are different. The idea behind this analysis was that if the P1 and N1 components would be generated by evoked responses, the P1 and N1 can be considered a manifestation of a sequential process with separate processing stages. Consequently, the variance of N1 latency should be larger than those for the P1. If, however, the P1 and N1 component are generated by an evoked oscillation they can be considered a manifestation of a sequential process with a unitary processing characteristic and it is to be expected that latency variances should not differ. The reason for these assumptions are that an increase in the variance of the later component should be observed only if the spread of neuronal activity behaves like a passive process over a long chain of neuronal elements. If on the other hand, the spread of neuronal activity is governed and shaped by a unitary mechanism (e.g., by an oscillation operating in thalamocortical circuits, and/or specific time constants of membrane potentials) different stages of a sequential process should not differ in the variance of latency measures. $F$ tests were calculated for electrodes O1 and O2 only because these electrodes showed the most prominent P1-N1 complex.

R E S U L T S

A pronounced P1 could be observed at occipital and parieto-occipital sites (cf. Fig. 2A). As shown in Table 1, P1 latencies were shortest at lateral parietal and occipital sites (with 108 and 112 ms at PO8 and O2, respectively) and longest at Pz (138 ms; cf. also Fig. 2B). The results of the ANOVA for P1 latencies revealed a significant effect for factor location \( [F(8,160) = 11.09; P < 0.001] \). Polynomial contrast analysis showed that latency differences followed a linear trend \( [F(1,20) = 28.92; P < 0.001] \). The results of the ANOVA for the N1 component did not reach significance. Latency of the P3 was 332 ms at Pz. $F$ tests calculated for the variances of P1 and N1 latencies (for electrodes O1 and O2) did not show significant differences.

The ERPs, filtered in the lower alpha range (7–10 Hz) exhibit the typical P1 – N1 waveform with a traveling direction from O1, O2 to Pz (cf. Fig. 2C). Topographical maps (Fig. 2D) show that the P1 emerges at around 100 ms at lateral parietal and occipital regions and then spreads to more anterior sites.
arriving at Pz at ~140 ms. At about this time, the N1 emerges at occipital and lateral parietal sites and exhibits a traveling pattern very similar to that of the P1. It should be mentioned, that an early negative component, termed N60 (with a latency of 66 ms at O1), precedes the P1 (cf. Fig. 2B) and exhibits a very similar traveling pattern as the P1 and N1. The interpeak latency of the N60 with respect to the P1 (of ~110 ms) suggests a frequency of ~77 Hz.

The findings about traveling speed are summarized in Fig. 3A, which depicts only those values that are significantly different from zero with a posterior to anterior traveling direction. As shown within the time window of the N60 and the P1-N1 complex significant values were obtained almost exclusively in the alpha frequency range (marginally including fast theta with ~6–7 Hz and slow beta with ~14–15 Hz). The results of the correlational analysis are similar but show a slightly different pattern for the P1 and N1. In the latency window of the P1, two different clusters of correlations were obtained, one in the upper alpha and slow beta and another in the lower alpha and theta frequency range. In contrast, the N1 exhibits only one cluster in the lower alpha and theta frequency range. (Fig. 3, B and C).

As depicted in Fig. 4, the control analysis (with high time but low-frequency resolution) yielded very similar results. The main differences to the standard analysis are that the time window of a systematic traveling speed ~3 m/s is concentrated at around 100–160 ms, which largely overlaps with the P1-N1 complex and that slow frequencies appear to play an important role. The latter effect, however, is due to the low-frequency resolution of the control analysis, which can be estimated by comparing the results of Fig. 4 with those of Fig. 3A.

**DISCUSSION**

The obtained findings clearly confirm the hypothesis that the P1 component behaves like a traveling alpha wave. It is important to emphasize that this conclusion is not only drawn from analyzing ERP components but from analyzing phase relationships on a single-trial basis. Two different analyzing methods, one focusing on traveling speed, the other on a correlation with ERP latencies, yielded very similar results. Analyzing topographical phase relationships expressed in terms of traveling speed revealed that a systematic posterior to anterior traveling pattern (between the selected electrodes O1, O2, P3, P4, Po3, Po4, P7, and P8 with respect to Pz) appeared only in the broad time window of the P1-N1 complex and in the extended alpha frequency range. The correlational analysis reflecting the similarity of the single-trial phase relationships with respect to the topography of P1 and N1 latencies underlines also the significance of the alpha frequency range and the time window of the P1-N1 complex (cf. Fig. 3).
It is interesting to note that not only the P1, but also the N1 and an early negative component, behave as traveling waves. The frequency characteristics of all of these three components lie in the alpha range. Thus the P1 can be considered part of an evoked traveling alpha wave complex which emerges in a time window of ~50–250 ms (cf. Fig. 2C).

The obtained findings are consistent with the oscillatory ERP model and imply that the phase of ongoing alpha waves becomes reorganized at occipital and lateral parietal sites in a time window coinciding with the N60 component. Then, from occipital and lateral parietal sites evoked alpha waves spread to more anterior regions with a focus on Pz. The interpretation of our findings on the basis of the oscillatory model raises several questions, which we will address in the following text: what are the mechanisms for the generation of traveling waves? For how many cycles traveling waves are expected? Why do both, the P1 and N1 behave as a traveling alpha wave? And what is the functional meaning of evoked traveling alpha waves with respect to the P1 component?

Alpha oscillations are not a unitary phenomenon and cannot be simply dismissed as ‘idling’ (cf. Klimesch et al. 2007 for a recent review). With respect to the generation of alpha waves, Nunez and Srinivasan (2006) presented evidence that ongoing alpha band activity appears to consist not only of traveling (and standing) waves but also of local patches of activity that may be associated with thalamocortical networks. Thus scalp recordings of either ongoing EEG or P1, N1 are likely to reflect some—generally unknown—combination of wave and network activity (cf. Nunez and Srinivasan 2006) that may underlie the event-related reorganization of phase.

In contrast to the ongoing EEG, only a brief period of traveling alpha waves is expected for the event-related EEG. The main reason is that after a brief period of evoked activity, alpha desynchronizes in response to a stimulus and/or event. Another potential reason is that according to the oscillatory model oscillations with different frequencies become aligned in phase during a certain time window (most likely within the latency window of the P1 and N1). Thus evoked traveling waves that are generated by the superposition of different frequencies (e.g., within the broad alpha and/or theta range) necessarily are a transient phenomenon.

The results of the control analysis (cf. Fig. 4) suggest that phase estimates obtained for the P1 are not contaminated by filter artifacts induced by the N1 (and vice versa). Because a consistent travel speed is obtained in a time window of ~100–160 ms, which nicely overlaps with the P1-N1 complex, we conclude that both components behave like a traveling alpha wave. This conclusion is supported by the fact that N1 latency shows a very similar topographical latency shift as the P1 does (cf. Table 1). The high time resolution of the control analysis (with filter parameter gamma set at 25 π, allowing for a time resolution of ±40 ms) can be judged by comparing Fig. 4 with Fig. 3A. Whereas in Fig. 4, a consistent travel speed ~3 m/s is limited to the time window of ~100–160 ms, the respective findings shown in Fig. 3 appear in a broad time window of ~0–250 ms. On the other hand, the low-frequency resolution

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**TABLE 1. P1 and N1 latencies**

<table>
<thead>
<tr>
<th>Component</th>
<th>Pz</th>
<th>P3</th>
<th>P4</th>
<th>PO3</th>
<th>PO4</th>
<th>P7</th>
<th>P8</th>
<th>O1</th>
<th>O2</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>138</td>
<td>128</td>
<td>132</td>
<td>120</td>
<td>122</td>
<td>116</td>
<td>108</td>
<td>113</td>
<td>112</td>
</tr>
<tr>
<td>N1</td>
<td>183</td>
<td>170</td>
<td>175</td>
<td>166</td>
<td>163</td>
<td>162</td>
<td>159</td>
<td>162</td>
<td>163</td>
</tr>
<tr>
<td>Difference: N1-P1</td>
<td>45</td>
<td>42</td>
<td>43</td>
<td>46</td>
<td>41</td>
<td>46</td>
<td>51</td>
<td>49</td>
<td>51</td>
</tr>
</tbody>
</table>

Values are in milliseconds.
of the control analysis shows up as a large frequency smear with an artificially large contribution of low frequencies. This is largely due to the fact that slow frequencies have much larger amplitudes than higher frequencies.

Well-established findings implying that the P1 and N1 reflect processing in separate cortical processing streams (e.g., Mangun 1995) may at first glance appear contradictory with our conclusion that both the P1 and N1 behave like a traveling alpha wave. But the oscillatory ERP model makes clear assumptions about the functional differentiation of the P1 and N1. The basic idea is that—depending on the type of task—oscillations with different frequencies contribute to the generation of the ERP. Consequently, different oscillations must become aligned in absolute phase within a certain time window poststimulus to generate a prominent ERP component such as the P1 or N1. We have shown that—depending on the type of task—the frequency characteristics of the P1 and N1 may be different. Whereas the P1 has a frequency characteristic primarily in the alpha range, the N1 has a comparatively stronger focus on theta frequencies (cf. Fig. 3, B and C) (Gruber et al. 2005; Klimesch et al. 2004). Thus the N1 is—depending on the contribution of other frequencies—a different component with respect to function, latency, and topography. It is very important to emphasize that the frequency composition (and thus degree of “heterogeneity” with respect to alpha) is very much task dependent. In complex memory tasks, the N1 is very much influenced by slower frequencies (e.g., Klimesch et al. 2004). In simple perceptual tasks, however, the N1 appears as “pure” (evoked) alpha component, very much like the P1 (Hanslmayr et al. 2007). Thus the differential aspect of the N1 as well as the possibility that the frequency characteristics of the P1 and N1 overlap to a large extent (as was found in the present study) can very well be explained by the oscillatory ERP model.

In general, the assumption is that the spread of alpha reflects the direction of neuronal communication as was proposed by a variety of other studies (cf. Schack et al. 2003). With respect to the link between alpha and the P1, we have suggested that the P1 is the earliest manifestation of a top-down process during early sensory processing (Klimesch et al. 2007). The idea is that under conditions where sensory processing is guided by a specific expectancy, e.g., about the spatial location and/or type of stimulus, the P1 amplitude will be larger than under condi-

**FIG. 3.** A: summary of findings about traveling speed. Only those values with a posterior to anterior traveling direction are shown that are significantly different from 0. Within the time window of the N60-P1-N1 complex significant values were obtained almost exclusively in the alpha frequency range. Color coding reflects traveling speed. B: very similar results were obtained for the correlational analysis. Color coding reflects significant correlations (transformed to z values) between relative phases with respect to Pz (expressed as latencies) calculated for each single trial and P1 latency differences with respect to Pz (see METHODS for details).
tions where specific expectancies are lacking. In the present task, subjects had to build up a very specific expectancy for stimulus processing that was to ignore the meaning of the word and focus on the ink color only. We assume that this type of expectancy triggers a top-down process during early stimulus processing in a sense that traveling alpha waves control or gate the direction of information processing in the brain. Because congruent, incongruent, and neutral words were presented in random order, subjects had to use the same strategy for all trials. This may be the reason why we did not find differences in the P1/N1 complex (with respect to latency and amplitude) among the three conditions. If congruent, incongruent, and neutral words would have been presented in different blocks, we would predict differences between conditions.

Within the theoretical framework of the traditional evoked model the obtained latencies of the P1-N1 complex may be interpreted to reflect a spread of single evoked components. But if we would adopt this view the following question arises. Two consecutive components (such as e.g., the P1 and N1) would imply a sequential process of separate processing stages in a sense that the later component (e.g., the N1) receives (or is a manifestation of) output from the earlier component (e.g., the P1). Thus in this case, the spread of neuronal activity behaves like a passive process over a long chain of neuronal elements. Such a process should lead to a gradual increase in jitter with time. But analyzing P1 and N1 latencies did not show an increase in the variance of the N1 latency. Thus the tight coupling of both components suggests the influence of some other process. For the evoked model, one might assume that a nonoscillatory top-down activation process operating on both the P1 and N1 might reduce the jitter of the latter component. On the other hand, for the oscillatory model the tight coupling of both components appears as the consequence of a single process—a traveling evoked oscillation—generating the P1 and N1. In theory, a combination of both models, spreading evoked components that are tuned by traveling waves, may also be considered. But regardless of the theoretical interpretation, the obtained findings are hardly consistent with a local, sequential processing mechanism. Evidence for this view also comes from the fact the P3 does not exhibit topographical latency differences and thus is not affected by latency differences of the P1-N1 complex.

Alpha phase velocities vary between ~6 and 14 m/s (cf. Nunez et al. 2001). Burkitt et al. (2000) analyzed phases of steady-state visual-evoked potentials and observed evoked traveling waves with a velocity ranging from 7 to 11. These estimates, however, were obtained after correcting for cortical folding by using a folding factor of ~2. Thus, multiplying travel speed of 3 m/s obtained in the present study gives an estimate of ~6 m/s, which is well in line with those reported by Nunez et al. (2001) and Burkitt et al. (2000). It should be emphasized that estimates for phase velocity depend on a variety of different factors. Nunez (1995) has brought attention to the fact that bipolar recordings give lower estimates than reference methods (as used in our study). Furthermore, measurements using MEG technology give even lower estimates. The disparity between these estimates is due in part to reference electrode and volume conduction effects. Another important factor is frequency. As an example, phase velocity for lower alpha (with a frequency of ~8 Hz) is ~6.4 m/s, whereas for upper alpha (~12 Hz) the estimate is ~8.4 m/s (cf. Nunez 1995; p. 568). As demonstrated e.g., by Schack et al. (2003), the type of cognitive demands also have a strong influence on phase velocity.

It is interesting to note that the obtained phase velocity estimate of ~6 m/s is well in line with propagation speed in corticocortical fibers. This type of fibers leave and reenter the thin cortical layer and are myelinated. In contrast, intracortical fibers that connect cortical neurons “locally” without leaving and reentering the cortex are not myelinated and show rather slow propagation values (of ~0.2–1.1 m/s, depending on the axon diameter) (cf. Nunez 1995; p. 512). Thus our findings suggest that the P1-N1 complex represents an occipital-to-parietal traveling alpha wave, propagating along corticocortical fibers. This interpretation, however, does not rule out the possibility that the thalamus may exert an important influence on corticocortical propagation via thalamocortical reentrant loops.

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