Neural Correlates of Dream Lucidity Obtained from Contrasting Lucid versus Non-Lucid REM Sleep: A Combined EEG/fMRI Case Study

Martin Dresler, PhD*1; Renate Wehrle, PhD*1; Victor I. Spoormaker, PhD1; Stefan P. Koch, PhD1; Florian Holsboer, MD, PhD1; Axel Steiger, MD1; Hellmuth Obrig, MD2,3,4; Philipp G. Sämann, MD1; Michael Czisch, PhD1

*Drs. Dresler and Wehrle contributed equally.

1Max Planck Institute of Psychiatry, Munich, Germany; 2Berlin NeuroImaging Center, Charité University Hospital, Berlin, Germany; 3Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany; 4Clinic for Cognitive Neurology, University Hospital Leipzig, Germany

Study Objectives: To investigate the neural correlates of lucid dreaming.

Design: Parallel EEG/fMRI recordings of night sleep.

Setting: Sleep laboratory and fMRI facilities.

Participants: Four experienced lucid dreamers.

Interventions: N/A.

Measurements and Results: Out of 4 participants, one subject had 2 episodes of verified lucid REM sleep of sufficient length to be analyzed by fMRI. During lucid dreaming the bilateral precuneus, cuneus, parietal lobules, and prefrontal and occipito-temporal cortices activated strongly as compared with non-lucid REM sleep.

Conclusions: In line with recent EEG data, lucid dreaming was associated with a reactivation of areas which are normally deactivated during REM sleep. This pattern of activity can explain the recovery of reflective cognitive capabilities that are the hallmark of lucid dreaming.

Keywords: REM, lucid dreaming, fMRI

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INTRODUCTION

In REM sleep we experience the most vivid and intense sleep mentation; however, these are deficient in reflective thought and metacognition. Instead the internally generated perceptions and emotions experienced during dreaming typically show many cognitive peculiarities, with a bizarre dream plot full of gaps, delusional thought, and a complete lack of insight into the fact that we are dreaming. These cognitive constraints have been proposed to rely on the neural activation patterns associated with REM sleep, particularly deactivation of the dorsolateral prefrontal cortex.1,2 In contrast to normal dreaming, lucid dreaming denotes a rare state of sleep comprising cognitive features of both waking and dreaming3: During lucid dreams subjects become aware of their dreaming state, have full access to memory, and are able to volitionally control dreamed actions. Although standard polysomnographic criteria of REM sleep are maintained and REM sleep muscle atonia prevents overt motor behavior, lucid dreamers are able to communicate their state by predefined volitional eye movements, clearly discernable in the electrooculogram.4 Lucid dreaming can be trained and has been successfully utilized for the treatment of nightmares.5 However, neural changes that accompany dream lucidity and their role in the amelioration of dream disorders are not well understood. When compared to non-lucid REM sleep, lucid dreaming is associated with increased 40-Hz activity and increased coherence in frontal regions, as has recently been shown by quantitative EEG.6 Neuroimaging data delineating the neural correlates of lucid dreaming are missing so far. While in our recent report the skill of lucid dreaming served as a tool for tracing specific motor activity in dreams,7 the current study aimed to reveal the neural correlates of lucidity obtained from contrasting lucid versus non-lucid REM sleep using a combined EEG/fMRI approach.

METHODS

Participants and Instructions

Inclusion criteria for this study were a self-reported ability to achieve lucidity in dreams more than once per week and the perceived ability to sleep in a MRI scanner environment, i.e., in a fixed supine position during loud noise. Four experienced lucid dreamers (all male; aged 27, 29, 31, and 32 years) were recruited through internet advertisements. They had been training lucid dreaming for 4, 20, 17, and 5 years, respectively. All subjects were screened for presence of medical conditions conflicting with their participation (diagnostic interview, EEG, MRI), gave written informed consent, and were paid for their participation. Participants repeatedly slept in a MRI scanner under concurrent polysomnographic monitoring during 2 to 6 successive nights (15 nights in total), depending on their availability and compliance. In order to trace the temporal onset and fading of the lucid dream, participants were instructed to repeatedly perform a simple task during their dream: Immediately after achieving lucidity, in their dream they should move their eyes left-right-left-right (LRLR), then clench their left hand for about 10 seconds, give the LRLR signal again, clench the right hand, etc., as long as possible. Figures of the polysomnographic signal and neural activation patterns reflecting the dreamed hand movements are given elsewhere.8 When LRLR
signals were followed by a period of about 40 seconds without any further signals, one of the investigators entered the scanner room, thereby awaking the participant, and inquired about task performance. The brief dream report was used to verify the lucidity signal seen in the EOG.

Data Acquisition and Sleep Characteristics

Functional images were acquired using a 1.5 MRI scanner (GE, Milwaukee, WI, USA) in the early morning hours (04:30 onwards), when highest probability of REM sleep can be expected. Simultaneous polysomnography included 19 EEG channels (10/20 system), EOG, EMG, and ECG (MRplus 32-channels, BrainAmp, Germany) referenced against FCz (sampling rate 5 kHz, no filters used at data acquisition). Since the MR scanner’s software only allowed acquisition of a maximum of 20000 volumes, the scanner was run without actual data acquisition but with all other experimental features including gradient noise while the subject was falling asleep as well as during initial NREM sleep stages. Online artifact correction (RecView 1.0) was applied to identify REM sleep during the ongoing polysomnographic recordings and to start fMRI and EEG data collection for a maximum of 20000 slices. fMRI volumes covered 25 slices (AC-PC-orientation, 64 × 64 matrix, 3 mm thickness, 1 mm gap; echo planar imaging (EPI), repetition time 2 s, echo time 40 ms). All 4 subjects fell asleep during scanning, with 2 eventually reaching unambiguous REM sleep. While all participants reported subjectively experienced lucidity at least once, only one subject had 2 independent and objectively verified lucid dreams with ≥ 4 consecutive sets of LRLR signals not directly followed by awakening.

Polysomnography Analysis

Offline MR-gradient correction and ICA-based cardioballistic artifact correction (Vision Analyzer 1.05), digital filtering (0.5-30 Hz for EEG, 0.1-30 Hz for EOG, 16-250 Hz for EMG recordings), and referencing of the EEG to linked mastoid electrodes were performed. As in our previous study, fMRI epochs showing lucid dreaming were defined by LRLR signals in the EOG. Lucidity was assumed to start with the first LRLR signal, indicating also the onset of the dreamed hand movements. During dreamed performance of the predefined hand clenching task, LRLR signals were repeatedly given whenever the subject changed the hand, thus also indicating continuation of lucidity. The end of lucidity was defined 6 s after the last LRLR signal if dream reports confirmed passage to non-lucid dreaming after last execution of the predefined task. Lucid epochs spanning ≥ 4 repeated LRLR signals were contrasted against flanking non-lucid REM epochs. Thus, 2 lucid periods of different nights with a duration of about 50 sec each (~25 fMRI volumes), flanked on both sides by 20 sec each (10 volumes) of REM sleep without signs of lucidity were extracted from the fMRI raw data. fMRI volumes outside these temporal windows were discarded.

fMRI Data Analysis

Postprocessing and statistical analyses were performed with SPM5 (http://www.fil.ion.ucl.ac.uk). Images were slice time corrected and realigned to the first volume using rigid body transformation. Functional and anatomical data were normalized to the EPI and T1 template, respectively, in Montreal Neurological Institute (MNI) space and resliced (voxel resolution 2 × 2 × 2 mm³). Functional data were smoothed using an 8 mm Gaussian kernel. For data analysis, the same model as in our previous study was used. While our previous analysis focused on dreamed hand movements, the present analysis focuses on the lucid/non-lucid difference, now considering the dreamed hand movement as a nuisance regressor. Thus, the fixed effect model comprised the following regressors and was estimated for all grey matter voxels after high-pass filtering (256 s): (1) lucid epochs convoluted with the canonical hemodynamic response function (HRF); (2-7) six affine motion correction parameters; (8-9) HRF convoluted epochs of the right and left first clenching task; and (10) a constant. To test for activation during lucid epochs we calculated a positive T-contrast for the first regressor, resulting in relative activity changes during lucid dreaming as compared to the background of non-lucid REM sleep. The false discovery rate (FDR) method was used for multiple test correction, and significance was accepted at the voxel level for FDR < 0.005.

Visualization

Brain surface reconstruction and fMRI cluster visualization was performed using BrainVisa software (http://www.brainvisa.com) and MRICro. T-values for projection on the surface mesh were averaged from a sphere (radius 5 mm) centred at the surface vertex point. For the precuneus (right hemisphere local peak voxel [6,-74,40]), predicted and fitted responses were plotted against time. All imaging data are presented in the neurological convention (LPI to RAS coordinate frame).

RESULTS

All 4 subjects fell asleep during scanning, with 2 of them showing unequivocal signs of REM sleep during the early morning hours. One of the subjects had 2 long and stable lucid dreams, occurring at 06:25 and 05:45 in different nights. These periods showed all features of REM sleep and were clearly distinguishable from adjacent non-lucid REM by the predefined EOG markers and were subsequently verified through dream reports. According to the dream reports, both lucid dreams started out of rather confused sleep mentation without clearly memorized content and ended in fading of lucidity—the first as result of a change in dream scenery, the second seemingly without an obvious cause. fMRI analysis was based on these 2 episodes. During lucid dreaming, we observed neural activations in a network of neocortical regions only (Figure 1 and Table 1). Cortical areas significantly (FDR < 0.005) activated during lucid REM sleep compared to non-lucid REM sleep comprised the bilateral precuneus [10,-88,42, peak z = 5.38], bilateral inferior and superior parietal lobules [left: 50,-52,52, peak z = 6.42; right: -36,-64,52, peak z = 5.77], bilateral basal occipitotemporal cortex [left: -54,-60,-16, peak z = 5.97, right: 64,-38,-14, peak z = 6.54], left frontopolar cortex [-26,62,10, peak z = 4.90] and right frontopolar / dorsolateral prefrontal cortex [48,48,8, peak z = 5.76], right cuneus [18,-80,8, peak z = 4.27], left frontal eye field [-28,26,50, peak z = 4.95], and bilateral lingual gyrus [8,-78,-16, peak z = 5.29].
**DISCUSSION**

Neuroimaging studies have shown that human REM sleep is related to characteristic patterns of regional brain activity: During REM sleep, neural activity in the brain stem, thalamus, amygdala, and extrastriate temporo-occipital cortices increases, while, e.g., the dorsolateral prefrontal cortex and the precuneus show deactivation. This specific pattern of neural activity has been proposed to reflect the visual hallucinations, emotional intensifications, and cognitive abnormalities typically experienced in dreams. In contrast, lucid dreaming is characterized by a regaining of higher cognitive capabilities, eventually leading to the awareness of the dreaming state. Recent quantitative EEG data have shown that this wake-like intellectual clarity is paralleled by neural activations in frontal and frontolateral re-

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**Figure 1**—Activity related to lucid dreaming. Color coded clusters represent areas significantly activated during lucid epochs in REM sleep ($p_{FDR} < 0.005$): left hemisphere (A), right hemisphere (B), midline view (C). Predicted (green) and fitted (black) fMRI data of the peak activation in the right precuneus, showing combined analysis of two independent lucid epochs in a single subject (boxed) (D).

**Table 1**—Increased neural activity during lucid dreaming

<table>
<thead>
<tr>
<th>Brain region</th>
<th>Brodmann areas</th>
<th>Cluster size (voxel)</th>
<th>Peak voxel</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 LR Precuneus (cuneus 26%)</td>
<td>7,18(R), 19(R)</td>
<td>1278</td>
<td>5.38</td>
</tr>
<tr>
<td>2 L Superior/inferior parietal lobule, precuneus, supramarginal and angular gyrus</td>
<td>7, 19, 39, 40</td>
<td>1148</td>
<td>6.42</td>
</tr>
<tr>
<td>3 R Superior/inferior parietal lobule, precuneus, angular gyrus, middle temporal gyrus</td>
<td>7, 19, 39, 40</td>
<td>1018</td>
<td>5.77</td>
</tr>
<tr>
<td>4 R Inferior/middle temporal gyrus, middle occipital gyrus, fusiform gyrus</td>
<td>20, 21, 37</td>
<td>527</td>
<td>6.54</td>
</tr>
<tr>
<td>5 R Inferior/middle/superior frontal gyrus</td>
<td>10, 46</td>
<td>525</td>
<td>5.76</td>
</tr>
<tr>
<td>6 L Inferior/middle temporal gyrus, middle occipital gyrus, sub-gyral</td>
<td>19, 20, 21, 37</td>
<td>355</td>
<td>5.97</td>
</tr>
<tr>
<td>7 LR Lingual gyrus, declive</td>
<td>18</td>
<td>211</td>
<td>5.29</td>
</tr>
<tr>
<td>8 L Middle/superior frontal gyrus</td>
<td>10</td>
<td>163</td>
<td>4.90</td>
</tr>
<tr>
<td>9 R Cuneus</td>
<td>17, 23</td>
<td>159</td>
<td>4.27</td>
</tr>
<tr>
<td>10 L Middle/superior frontal gyrus</td>
<td>8</td>
<td>110</td>
<td>4.95</td>
</tr>
</tbody>
</table>
regions. Likewise, PET data show cognitive control in dreams to be associated with neural activations in a specific network of cortical regions. In line with the study of Voss et al., during lucid dreaming we observed increased activity in the right dorsolateral prefrontal cortex. The dorsolateral prefrontal cortex is thought to underlie a wide range of higher cognitive capacities, as evidenced also by the dysexecutive syndrome seen in patients with lesions in this region. In particular, the right dorsolateral prefrontal cortex was associated with self-focused metacognitive evaluation. While in normal dreams, working memory is strongly impaired, activation in the dorsolateral prefrontal cortex in combination with parietal lobules, which we also found to be activated during lucid dreaming, may reflect working memory demands related to task performance in our study. We further observed increased activation in bilateral frontopolar areas, which have been related to the processing of internal states, e.g., the evaluation of one’s own thoughts and feelings.

The strongest increase in activation during lucid compared to non-lucid REM sleep was observed in the precuneus, a brain region that has been implicated in self-referential processing, such as first-person perspective and experience of agency. While in normal dreams, attention is often hyper-associatively driven by the (pseudo-)external dream scenery, lucid dreaming is—by definition—characterized by a reflection on one’s own state of mind.

Interestingly, we found activation in the bilateral cuneus and occipitotemporal cortices. These areas are part of the ventral stream of visual processing, which is involved in several aspects of conscious awareness in visual perception. While these activations seem puzzling at first sight—since non-lucid dreams are also characterized by vivid dream imagery—they are in line with reports of lucid dreamers stating that lucidity is associated with an exceptional brightness and visual clarity of the dream scenery.

While lucid dreaming is an intriguing phenomenon in its own regard, it can also serve as a tool for the study of dream disorders. For example, lucid dreaming training has been shown to be effective in the treatment of recurrent nightmares. Neurocognitive models of disturbed dreaming emphasize a hyperresponsivity of the amygdala in nightmare generation, coupled with a failure of medial prefrontal regions to dampen this activation. Lateral prefrontal regions have been shown capable to influence amygdala function through connections to the medial prefrontal cortex. Increased lateral prefrontal activation during lucid dreaming therefore fits well with the therapeutic effects of lucidity training on recurrent nightmares. If lucid dreaming can also be utilized in the treatment of other dream disorders like terrifying hypnagogic hallucinations or pathological dream vividification is an open question that warrants further investigation.

Limitations

Due to the rarity of lucid dreaming in untrained subjects, we were able to recruit only four subjects who were highly trained lucid dreamers. Only one of them became lucid twice under concurrent EEG/fMRI conditions, rendering our data a case study. Another limitation is that tracing lucidity requires repeated signaling of the lucid state. Part of the observed activation therefore may well originate from the eye-signaling and hand-clenching task performed during lucidity, which comprises both task-switching and sustained attention. While this working memory load might have confounded our analyses, it is important to note that such working memory demands are inevitable for the lucid dream to be objectively recorded: without sustained attention towards the dreaming state, repeatedly signaled by eye movements, onsets and offsets of lucidity can not be detected. However, future studies on the neural correlates of lucidity might utilize a more specific task, such as active contemplation about the current state of consciousness.

CONCLUSION

In line with recent EEG data, lucid dreaming was associated with a reactivation of several areas normally deactivated during REM sleep. This pattern of activity can explain the recovery of reflective cognitive capabilities that are the hallmark of lucid dreaming.

DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

REFERENCES