Cortical stimulation study of the role of rhinal cortex in déjà vu and reminiscence of memories

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Abstract—*Objective:* To study the role of perirhinal (PC) and entorhinal cortices (EC) in dreamy state symptoms (déjà vu and reminiscence of scenes). These phenomena have been attributed to functional alteration of memory networks supported by the medial temporal lobes, principally involving the amygdala and hippocampus. The role of sub-hippocampal structures (EC and PC) in inducing these phenomena has not previously been addressed. *Methods:* The authors studied the symptoms evoked by direct electrical stimulations of PC and EC in comparison with those obtained after stimulation of the amygdala and hippocampus. Stimulations were performed in a group of 24 patients with epilepsy, during stereo-electroencephalographic (SEEG) recordings in the setting of presurgical evaluation. All patients had electrodes that sampled the rhinal cortices, amygdala, and hippocampus. *Results:* A total of 280 stimulations were analyzed. Entorhinal and perirhinal stimulations induced classic mesial temporal lobe responses (emotional, dysautonomic) but also more specific responses, particularly the déjà vu phenomenon and reminiscence of scenes. Such déjà vu or déjà vécu type responses were produced proportionately more often by stimulation of the EC than by stimulation of the amygdala and hippocampus. In particular, déjà vu was associated with stimulation of the EC and reminiscence of memories with PC stimulation. *Conclusion:* This study strongly suggests that experiential symptoms are largely dependent upon functional modification of the physiology of the rhinal cortices.

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Experiential mnemonic phenomena may occur during partial seizures involving the temporal lobe and may be reproduced by direct human brain stimulation.[1] Jackson[2] gave the first description of the phenomena that he called "dreamy state." He included two main alterations of memory: feeling of reminiscence, in which the subject has the feeling that the current experience is a repetition of an episode already lived, now usually termed déjà vu or déjà vécu (DV), and vivid hallucination of scenes, in which the subject remembers an earlier episode of his or her life. For Jackson, the dreamy state resulted from hyperactivity of normal neural regions, due to liberation from the control of higher centers impaired by epileptic activity.

Later, Penfield and Jasper also studied the phenomenon of dreamy state and, unlike Jackson, grouped sensory illusions (auditory, sensory, or visual), emotional disturbances, and the feeling of reminiscence[3] all within the same category ("interpretive illusions"). Under "experiential hallucinations," Penfield and Perrot[4] included the reminiscence of memories ("tape recorder recall") but also crude auditory and visual hallucinations. Penfield and Perrot found that both categories of phenomena were evoked by electrical stimulations of the lateral temporal neocortex, particularly the superior temporal gyrus.

However, subsequent studies using depth electrode stimulation found that memory-related symptoms could be evoked by the stimulation of medial temporal lobe (MTL) structures[5-9] and occurred rarely after stimulation of the temporal neocortex. More recently, it has been proposed that dreamy state depends upon a neural network engaging both medial and lateral structures.[10]

These studies collected data from stimulation of the hippocampus (anterior and posterior), the amygdala, and, less frequently, the posterior part of the parahippocampal gyrus, in the setting of presurgical evaluation with depth electrode recording. Even though memory phenomena were rarely induced, for most authors the most sensitive stimulation sites to do so were the anterior part of the medial temporal lobe and particularly the amygdala.[1]

The role of anterior sub-hippocampal structures (entorhinal and perirhinal cortices) in dreamy state is unknown. These structures belong to the mesial temporal lobe, and recent models of memory proposed that they may have specific functions in declarative memory.[11-13] It thus may be that they contribute specifically to dreamy state. We report in this study the results of direct electrical stimulation of the rhinal cortices and other MTL structures in patients with epilepsy.

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Methods. *Subjects.* All patients were investigated at the Unité d'Epileptologie–Service de Neurophysiologie Clinique between September 2000 and June 2002. All had drug-resistant partial epilepsy and were investigated using stereotactically implanted intracerebral electrodes as part of their planned presurgical evaluation. We selected 24 patients from a series of 101 consecutive patients explored by stereoelectroencephalography (SEEG), on the basis of their having had electrodes implanted that sampled the amygdala, hippocampus, and rhinal cortices.

Each had a comprehensive evaluation including detailed history and neurologic examination, neuropsychologic testing, high-resolution MRI, interictal/ictal single photon computerized tomography (SPECT), and video-EEG recording of seizures. Following these noninvasive investigations, SEEG was subsequently performed to define the distinct role of temporal lobes or extratemporal structures in seizure generation. Patients underwent continuous SEEG monitoring for 4 days, or longer (up to 2 weeks) if no seizures had been recorded during this period. Reduction of antiepileptic drug treatment was carried out where this was believed to be clinically necessary to facilitate recording of seizures. Fully informed consent was obtained form each patient prior to electrode implantation.

Patients included in the study had temporal lobe epilepsy in 21 cases, frontal lobe epilepsy in 1, and occipito-temporal epilepsy in 2 cases.

Stereotactic implantation of depth EEG electrodes. The technique of SEEG used was that described by Talairach et al.[14] It requires implantation of multiple contact intracerebral electrodes (diameter: 0.8 mm; number of contacts: 5 to 15; length of each contact: 2 mm; spacing interval: 1.5 mm), using a standard double-grid system fastened to the Talairach stereotactic frame. Six to 12 electrodes were implanted in each case, providing more than 100 recording points and consequently an extended electrophysiologic sampling of the brain areas of interest.

Contact localization. Planning the stereotactic trajectory for each electrode was carried out preoperatively with non-stereotactic MRI, using software platforms (Surgiplan, Elekta, Sweden, and Anatomist, France[15,16]). These allow a computerized three-dimensional image of the sulci to be created by extraction from other cerebral structures, with automatic labeling and navigation in three-dimensional images. Postoperatively, a CT scan without contrast was performed to check for absence of bleeding and to confirm the precise topography of each contact. After the period of continuous video SEEG recording, electrodes were removed, and an MRI performed the following day in order to locate the trace of each electrode in the brain (figure 1B). The combination of the postoperative CT scan and the postimplantation MRI allowed precise anatomic location of each contact of each electrode (see figure 1).

The perirhinal cortex in the human is a transitional cortex between allocortex and isocortex located in the collateral sulcus.[17] It consists of Brodmann area 35 on the medial wall of the collateral sulcus and of Brodmann area 36 in its lateral part.[18] Its limits in the present study were defined according to previous studies.[19,20] The anterior boundary starts about 1 mm from the anterior part of the collateral sulcus and ends at the lateral geniculate nucleus level on a coronal plane. The entorhinal cortex corresponds to the rostral part of the parahippocampal gyrus extending from the limen insulae (anteriorly) to the hippocampal fissure (posteriorly) and from the subiculum (mesially) to the medial wall of the collateral fissure (laterally).

In each patient, an electrode running through the anterior and basal portion of the temporal lobe (temporo-basal electrode) recorded from the lateral middle temporal gyrus (MTG) cortex, the lateral and mesial walls of the occipito-temporal and collateral sulcus, and then ended in the entorhinal cortex (figure 2).

Electrical stimulation. Electrical stimulation was carried out routinely as part of the standard presurgical assessment, to provide additional electroclinical data about the epileptogenic zone (in determining the sites in which seizures can be triggered) as well as functional mapping of eloquent cortex (in inducing memory or language alterations). Electric stimulation was produced by a regulated neurostimulator designed for safe diagnostic stimulation of the human brain (Inomed).

High frequency stimulation at 50 Hz (pulse duration 1 msec) was applied in a bipolar fashion to each contact in the gray matter during a 5-second period. The current intensity was gradually increased ranging from 0.5 to 2.5 milliamperes (mA), until a clinical effect or the appearance of an EEG afterdischarge was obtained. During stimulation, patients were sitting in bed and were asked to read or to count. Patients were asked to report any

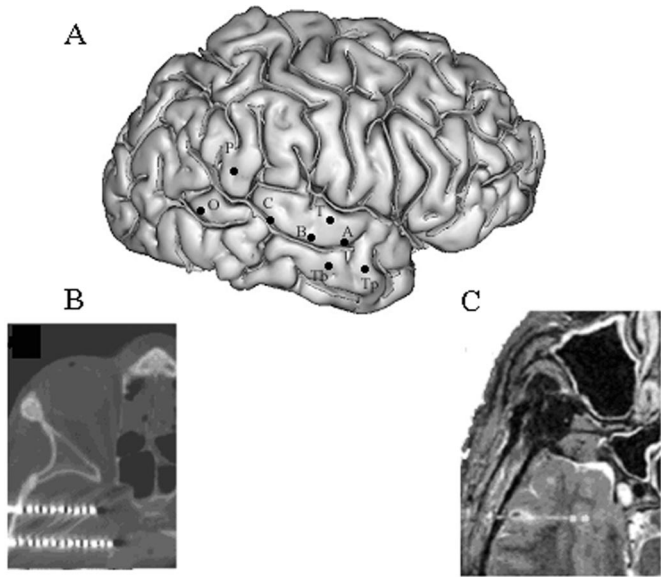

Figure 1. Example of SEEG electrodes mapping represented on a lateral view of the brain. (A) Figure shows the MRI three-dimensional surface reconstruction of the right hemisphere of Patient P2, with the site of implantation of each electrode. Electrodes are identified by one or two capital letters (e.g., A, B) and recording contacts are numbered from 1 to 10 or 15 depending on the number of contacts per electrode. The lowest numbers (e.g., 1, 2, 3) correspond to the deepest, most medial structures (for instance, contacts A1 and A2 record signals from the amygdala while A9 and A10 record from the lateral neocortex of the middle temporal gyrus). Bipolar signals are obtained from subtraction of signals recorded on two adjacent contacts (for instance, bipolar signal A1-A2 may be obtained from monopolar signals A1 and A2). Stimulations were applied to two adjacent contacts (for instance, TB1-TB2 for entorhinal cortex stimulation). A: Electrode exploring the amygdala (medial contacts) and the anterior part of the middle temporal gyrus (MTG) (lateral contacts). B: Electrode exploring the anterior hippocampus (medial contacts) and the mid part of MTG (lateral contacts). T: Electrode exploring the posterior part of the superior temporal gyrus. TP: Electrode exploring the temporal pole. TB: Electrode running through the anterior and basal portion of the temporal lobe (temporo-basal electrode). This electrode records the lateral MTG cortex, the walls of the occipito-temporal sulcus and the collateral sulcus (perirhinal cortex), and then ends the anterior part of the parahippocampal gyrus (entorhinal cortex). O: Electrode exploring the occipito-temporal region. P: Electrode exploring the inferior parietal region. (B) Axial CT scan of right temporal lobe that allows calculation of the precise distance of each contact from the midline vertical plane. (C) Post SEEG axial MRI of the anterior part of the right temporal lobe showing the thin trace left by the electrode. Each contact (white dots) can be positioned on this trace according to its distance from the midline calculated from the CT scan.

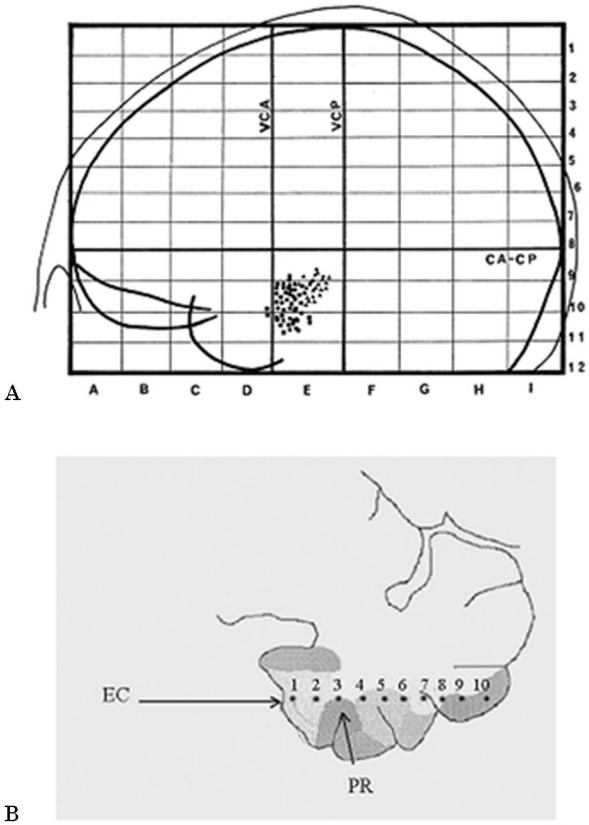

Figure 2. (A) Representation of the placement of the electrodes from the 24 patients, exploring the amygdala (circles), the anterior hippocampus (triangles), and the basotemporal region (squares). Right and left electrode positions are projected on a lateral view of the left hemisphere normalized in the proportional stereotactic grid system of Talairach and Tournoux. CA-CP = anterior commissure–posterior commissure plane; VCA = vertical plane through CA; VCP = vertical plane through CP. (B) Schematic reconstruction of the temporo-basal electrode route exploring the perirhinal cortex (PR) and the entorhinal cortex (EC) in Patient P1. The numbers refer to the electrode contacts.

symptom or clinical change, however subtle. They were not aware of when, or whether, stimulation was applied. Patients expressed memory phenomena only during the stimulation periods.

Statistical analysis. Statistical analysis was performed with the χ^2 test or the Fisher exact for qualitative data test between two groups and with the χ^2 test among more than two groups (comparison of induced symptoms between the different sites of stimulation) and analysis of variance (ANOVA) to compare the stimulation intensities over different sites. Significant values were retained for p values less than 0.05.

Results. A total of 280 stimulations were analyzed: 146 in the rhinal cortices (RC), of which 83 were in entorhinal (EC) and 63 in the perirhinal (PR) sites, 46 in the anterior hippocampus (H), and 88 in the amygdala (A). The mean applied intensity of stimulation was not different in the three sites of stimulations (RC: 1.37 mA; A: 1.2 mA; H: 1.30 mA; $p = 0.31$). Stimulations that induced overt seizures (fully developed seizure with loss of consciousness) were not studied here.

Comparison of stimulations of rhinal cortex, amygdala, and hippocampus. The results of these stimulations are summarized in table 1. These stimulations induced viscero-sensitive, emotional, or experiential phenomena. In particular, the most frequent effects were the induction of epigastric sensation and anxiety. Although rare (two stimulations in two patients), olfactory hallucinations were only observed after stimulation of the amygdala. In one case, olfactory hallucination was the core of a more complex memory-related phenomenon: stimulation evoked an olfactory hallucination of the scent of burned wood, which reminded the patient of a precise evening in Brittany on a beach around a campfire when she was 14 years old. This hallucination was categorized as a memory reminiscence.

DV was associated with stimulation of the rhinal cortex, occurring with 16 stimulations in 7 patients (30% of patients), compared to the amygdala in only 1 patient (2 stimulations) and 1 patient with hippocampus stimulation (1 stimulation) ($p = 0.01$). Reminiscence of scenes was more often induced by perirhinal stimulation ($p = 0.38$, non significant) (occurred after five entorhinal stimulations in four patients vs only once with amygdala stimulation in one patient and never with hippocampus stimulation).

Symptoms evoked by rhinal cortice stimulations. Subjective experiences were produced in 21/24 patients (87%) after stimulation of EC or PR or both. An afterdischarge located in the rhinal region or in the medial structures of the temporal lobe (hippocampus, amygdala, and rhinal region) (57) was induced in 78/146 (53%) RC stimulations. Table 2 summarizes the main responses obtained after stimulation of RC. Anxiety or fear was the most frequently reported symptom, occurring with 39 (26%) stimulations in 13 patients (22 on the right side and 17 on the left). Memory-related phenomena were frequently obtained: DV following 16 stimulations in 7 patients, and reminiscence of scenes following 5 stimulations obtained in 4 patients. DV was observed after stimulation of the right rhinal cortex in 5 patients and stimulation of the left in 2. Reminiscences of scenes were observed equally often after right (3) and left (2) sided stimulation. An epigastric sensation (13 stimulations: 7 right side, 6 left side) or a sensation of cooling or warmth of the body (6 stimulations: 3 right side, 3 left side) was also frequently reported, as well as speech disturbances (arrest of reading out loud) for left hemisphere stimulations. Other responses included headache and vague or bizarre sensations (such as the feeling of something happening in the head, or an indefinable sensation).

Differences between PR and EC stimulations were demonstrated (tables 2 and 3). Epigastric sensation symptoms ($p < 0.01$), emotional symptoms ($p = 0.01$), and DV ($p < 0.01$) were observed more often after EC than PR stimulation. In contrast, only PR stimulations induced memory reminiscence ($p = 0.01$). These reminiscences were reported immediately after the end of the stimulation and were brief and transient. After stimulation of the left PR at 2 mA, Patient P3 described the prow of a steamer on the sea associated with a transient feeling of familiarity. Patient P2, after right PR stimulation at 2 mA, saw the chromed part of a motor, which reminded him of his neighbor going by on his motorbike in the street, a familiar scene to him. Patient P7 had right PR stimulation at 1 mA, which evoked the reminiscence of scenes from his infancy in which the patient was with her father in their kitchen.

Table 1 *Main symptoms evoked by rhinal cortices, amygdala, and hippocampus stimulation*

Induced symptoms	Rhinal cortices	Amygdala	Hippocampus	*p* Value
No.	146	88	46	
Experiential phenomena				
Déjà vu–déjà vécu	16 (11)	2 (2.2)	1 (2.1)	0.01
Reminiscence of memory	5 (3.4)	1 (1.1)	0	0.38*
Emotional				
Fear or anxiety	39 (26)	20 (22)	13 (28)	0.72
Viscero-sensitive				
Epigastric sensation or throat striction	13 (9)	12 (13)	10 (21)	0.06
Warming or cooling feeling	7 (4.8)	9 (10.1)	2 (4.3)	0.21
Speech disturbances	5 (3.4)	1 (1.1)	0	0.38*
Other	11 (7.5)	11 (12.5)	9 (19.5)	0.06

The results were obtained in 24 patients. The number (%) of stimulations that induced symptoms is indicated. Statistical analysis (comparison of the symptoms in the three regions) was done using chi-square analysis (or Fisher exact test indicated by *).

Stimulation of the left PR in Patient P8 provoked the reminiscence of a dream that he had had the night before, involving a scene from everyday life (a discussion with a colleague in their workplace).

Discussion. In this study, we report the effects of intracortical rhinal cortice stimulations performed in patients during SEEG investigation of drug-resistant epilepsy, comparing them with those obtained after stimulation of the amygdala and hippocampus in the same group of patients. Rhinal cortice stimulation induced classic MTL responses. The responses we report here are thus in accordance with past studies using high frequency train direct stimulation of the limbic system. In particular, limbic stimulation evoked emotional, viscero-sensitive, and autonomic responses as well as memory phenomena.[1,5,7-10] As such, rhinal stimulations (in particular EC) did not basically differ from stimulations of other MTL structures. In addition, except for the speech disturbances, the side of stimulation (right or left) did not seem to influence the occurrence of such phenomena.

Table 2 *Comparison of entorhinal and perirhinal cortex stimulations*

Induced symptoms	Entorhinal	Perirhinal	*p* Value
No.	83	63	
Experiential phenomena			
Déjà vu	14 (16.8)	2 (3.1)	0.008
Reminiscence of scenes	0	5 (7.9)	0.01*
Emotional symptoms			
Fear or anxiety	29 (35)	10 (15.8)	0.01
Viscero-sensitive symptoms			
Epigastric sensation	13 (15.6)	0	0.0006*
Feeling of warmth or cooling	6 (7.2)	1 (1.6)	0.11
Speech disturbances			
Blurred speech or reading arrest	1 (1.2)	4 (6.3)	0.16
Other	5 (6)	6 (9.5)	0.42

The results were obtained in 24 patients. The number (%) of stimulations that induced symptoms is indicated. Statistical analysis was done using chi-square analysis or Fisher exact test (*).

However, we also found that experiential memory phenomena (déjà vu and memory reminiscence) were more frequently induced by rhinal than by hippocampal-amygdala stimulations. Déjà vu phenomenon was more frequently obtained after stimulation of the entorhinal cortex whereas memory reminiscences were more often induced by stimulations of the perirhinal region. This differs from past studies, which suggested that all medial structures might be equally involved in the genesis of dreamy state. However, as stated above, these earlier studies did not specifically consider the role of rhinal cortex, since depth electrodes did not explore this region.

The DV phenomenon is a common feature of temporal lobe seizures involving the medial temporal region and has frequently been reported after stimulation of the medial temporal lobe. It is also often reported in normal subjects, but as a milder and isolated sensation.[21,22] Our study shows that an illusion of familiarity is often obtained after stimulation of the rhinal cortices (EC or PR) and more rarely after hippocampal or amygdala stimulation. The most sensitive site to provoke this phenomenon was the EC. To our knowledge, this has never previously been reported. Previous stimulation studies have indeed reported DV phenomenon after stimulation of the hippocampus or more often amygdala, but only rarely (8% in the largest series[8]). In addition, no specific site of stimulation likely to induce DV has been previously identified. In light of our results, this could be explained by the fact that no previous study specifically studied the stimulation of rhinal cortices and that, in previous findings, stimulation of

Table 3 *Summarized data of patients and stimulations inducing memory-related phenomena*

Patient	Type of epilepsy	Sex/age, y	Epilepsy duration, y	Etiology	MRI	Semiology of usual seizures
P1	R-TLE	M/32	27	Vascular malformation	Cavernoma of the right lateral temporal pole and HA	Déjà vu, anxiety, tachycardia, epigastric sensation, CPS
P2	R-TLE	M/30	12	FC at 1 year	HA*	Déjà vu, reminiscence of memories, laryngeal striction, CPS
P3	L-TLE	M/29	14	—	HA*	Déjà vu, fear, epigastric sensation, CPS
P4	R-OE	F/26	10	Cortical dysplasia (right occipital†)	Normal	Visual hallucinations, CPS
P5	R-TLE	M/44	18	Cortical malformation	Bilateral periventricular heterotopia	Epigastric sensation, déjà vu, CPS
P6	R-TLE	M/19	5	Tumor	Ganglioglioma of the right parahippocampal region	Déjà vu, CPS
P7	R-TLE	F/36	32	Tumor	Ganglioglioma of the right amygdala	Epigastric sensation, anxiety, CPS
P8	L-FLE	F/35	11	—	Normal	CPS

Electrode contacts are numbered from 1 to 15, with 1 being the most internal and 15 the most external.

* With MRI volumetric data confirmation.
† Histologic confirmation.

the amygdala or hippocampus might have provoked DV by spread of the discharge to the adjacent rhinal cortex. In agreement with this, previous authors have found that stimulations that produced DV were most often associated with an afterdischarge involving the limbic structures.[7,8]

It has been proposed that DV may result from a transient alteration in the "feeling of familiarity" function of the recognition memory system.[22] The fact that the rhinal region was the main site to induce illusions of familiarity when stimulated is in agreement with recent animal and human studies, which have shown that familiarity discrimination is strongly dependent upon subhippocampal structures within the medial temporal lobe.[23] Recent functional MRI studies have shown that a decrease in activation of PR/parahippocampal regions occurs as items became familiar.[24-26] Neuronal recordings in animals have shown that PR and EC contain neurons that decrease firing with subsequent presentation of a visual stimulus.[27] This reduction in neuronal response was supposed to be the basis of relative familiarity or recency of the stimulus.[12] In contrast, these kinds of response were not recorded within the monkey or rat hippocampus, which can however respond to the relative familiarity of a visual stimulus occurring in a particular spatial position.

The mechanisms by which stimulations produce an electroclinical effect are unknown. Direct electrical stimulation of a neural structure can inhibit function or stimulate its normal output.[28] It is thus possible that direct electrical stimulation of the EC and PR inhibits these structures and transiently mimics the effect of familiarity discrimination, by decreasing neuronal responses. Whatever the mechanisms underlying the effect of stimulation, these data support a major role of the rhinal cortices in familiarity discrimination and in production of DV phenomenon.

Stimulation of the PR induced memory reminiscences in four patients. The reminiscence of memories is a classic feature of dreamy state, and has previously been obtained by stimulation of medial temporal lobes including hippocampus, parahippocampal gyrus, and amygdala.[1,7,8,10,29] However, the qualitative nature of the memories elicited by PR stimulations may be different from those elicited by amygdala or hippocampus stimulations. Two memories in our patients dealt with visual objects (the prow of a steamer, the chromed part of a motor). This may be due to the fact that the PR belongs to the ventral visual stream and is located medial to area TE (Brodmann area 20), known to be important for visual memory in the monkey. Furthermore, these memories in our patients tended to be usual episodes they had lived many times. As such, these memories were not contextualized in time and could therefore be related to semantic memory. This contrasts with the memory elicited by amygdala stimulation described in this study, which was very precise in time and had happened only once. This also contrasts with other such memories reported in the literature such as that of Patient 5 of Gloor et al., who after stimulation of the amygdala remembered a precise episode when he was young.[7] Rhinal cortices have been hypothesized to be crucial for semantic, context-free memory[30,31] in recent models of declarative memory. Although too few memory reminis-

Table 3 *Continued*

Side of positive stimulation	Electrode contacts (TB electrode)	Stimulation intensity, mA	Localization	After discharge	Déjà vu	Reminiscence of memory
R	1–2	1	EC	Yes	Yes	No
	3–4	1.2	PR	Yes	Yes	No
R	4–5	2	PR	Yes	No	Yes
L	4–5	2	PR	No	No	Yes
R	1–2	1.5	EC	No	Yes	No
R	1–2	1.5	EC	No	Yes	No
L	1–2	1	EC	No	Yes	No
R	5–6	1.5	PR	No	No	Yes
L	5–6	1.5	PR	No	No	Yes

TLE = temporal lobe epilepsy; HA = hippocampal atrophy; CPS = complex partial seizures; FC = febrile convulsions; OE = occipital epilepsy; FLE = frontal lobe epilepsy.

cences were reported by our patients to draw any firm conclusions, our data suggest that the nature of the memory reminiscence elicited after PR stimulations may be qualitatively different from those elicited after stimulation of other MTL structures such as the hippocampus or the amygdala.

The relationship between these induced phenomena and the physiology of normal brain function is of course debatable, since epilepsy may produce functional alterations of the temporal lobe networks. The evoked phenomena habitually occurred in the course of their usual seizures in 5/8 patients with amnesic phenomena (see table 2). However, 3/4 of the patients with reminiscence of scenes never usually experienced this phenomenon during their spontaneous seizures. This relationship with normal physiology has been discussed by several authors, who contend that epileptic networks in the brain induce hyperexcitability and facilitation of some normal networks, but probably follow a normal functional connectivity rather than a fundamentally different organization of the brain.[1,10,29]

Overall, our results consolidate previous work showing that the dreamy state involves the medial structures of the temporal lobes. However, our study suggests that not all structures of the temporal lobes contribute equally to its genesis, and that functional alteration of rhinal cortice physiology may be important contributors to these symptoms.

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