Dreaming: a neuroimaging view

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Summary

Dang-Vu TT, Desseilles M, Albouy G, Darsaud A, Gais S, Rauchs G, Schabus M, Sterpenich V, Vandewalle G, Schwartz S, Maquet P. Dreaming: a neuroimaging view. Schweiz Arch Neurol 2005;156: 415–25.

Our neurobiological knowledge about human dream organisation results primarily from the study of Rapid-Eye-Movement (REM) sleep. In humans, functional neuroimaging techniques, using $H_2{}^{15}O$ or ^{18}FDG positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), allowed the mapping of the regional cerebral activity during this sleep stage, which is dominated by the activation of the pons, the thalamus, temporo-occipital and limbic/paralimbic areas (including the amygdala, the hippocampal formation and the anterior cingulate cortex), along with a relative quiescence of dorsolateral prefrontal and inferior parietal cortices. These results are in agreement with animal neurophysiological data about REM-sleep generation. They may also explain several hallmarks of dreaming experience that are found in dream reports after awakening from REM sleep. For instance, amygdala activation is consistent with the predominance of threat-related emotions. Temporo-occipital activation is in keeping with visual dream imagery. Prefrontal deactivation is suggestive of the lack of orientational stability, the alteration in time perception, the delusional belief of being awake, the decrease in volitional control and the fragmented episodic memory recall. Inferior parietal deactivation may contribute to the lack of distinction between first- and third-person perspectives. Conversely, specific cognitive and emotional features in individual dreams could be used to predict some aspects of the regional functional organisation of the human brain during dreams, and also inspire the design of future dedicated neuroimaging studies by offering constraints to the analysis and interpretation of sleep data acquired just before dream reports. Therefore, we suggest that future functional brain imaging in humans should be combined with a careful neuropsychological analysis of dream reports, and especially their categorisation based on the presence of specific bizarre features, to test hypotheses about the brain correlates of dreams. As little is known about the physiology of non-REM sleep dreaming, future neuroimaging studies should also attempt to link dreaming experiences during this sleep stage with patterns of regional cerebral activity. Overall, although many questions arising from the study of oneiric behaviour remain unanswered, recent neurophysiological and neuroimaging research about REM sleep offers an increasingly detailed picture of the cerebral correlates of dreaming that might also provide new insights into dream functions.

Keywords: dreaming; sleep; functional neuroimaging; REM

Introduction

Dreaming is experienced every night by most humans as multi-sensory mental representations occurring spontaneously during sleep, often organised in a narrative manner. Formal features of dreams can be summarised as follows. Dreams are characterised by their perceptual features (mostly visual and auditory) and emotional content (frequently negative such as anxiety and fear). They typically appear bizarre due to the incongruity,

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1 T. D., M. D., V. S., G. V. and P. M. are supported by the Fonds National de la Recherche Scientifique (FNRS) (Belgium). S. S. is supported by the Swiss National Science Foundation (grants: 3100-AO-102133 and 3200B0-104100). A. D. and G. R. are supported by the Fyssen Foundation (France). Additional support for the work presented here comes from the University of Liège and the Queen Elisabeth Medical Foundation.

discontinuity and instability of time, places and persons [1, 2], but are nevertheless taken as real by the dreamer. On awakening, the memory of the dream, if any, vanishes quickly.

As part of the study of consciousness, the investigation of dreams shares several challenging characteristics with it. For instance, dream research relies critically on phenomena that are known only via introspection and are not accessible to direct observation. The dreamer is the unique observer of his or her dream. Moreover, the genuineness of dream reports is affected by issues inherent to dream evaluation such as forgetting, reconstruction mechanisms, verbal description difficulties and censorship [1].

Originally, dreams were thought to be part of the supernatural world. Dreams were messages sent from gods to prevent disaster or misfortune. The Egyptians are thought to be the first civilisation interested in dreams, interpreting them when they had troubles in their life. The Greeks also believed that dreams carried divine messages but only priests and priestesses, such as the Pythia of Delphi, were able to interpret them. Later on, dreams were progressively considered in a more rational way. Heraclitus suggested that a person's dream world was a concept created in one's own mind. Aristotle also challenged the divinatory and premonitory aspects of oneiric experience. He emphasised that dreams would amplify the perception of external stimuli and suggested an endogenous origin for dreams. In brief, efforts to explain how and why we dream date back to Antiquity and progressed slowly through the centuries. During the second half of the 19th century, several scientists conducted ingenious experimental studies on dreaming while proposing theories about the cerebral mechanisms underlying it that are strikingly close to some recent theories [3]. However, these first experimental attempts were drastically dismissed not only by Freud and his disciples, but also by the Behaviourists whose models of human behaviour excluded mental representations. As a result, dream research made little progress during the beginning of the 20th century until a major neurophysiological discovery was published 50 years ago. In 1953, Aserinsky and Kleitman [4] described for the first time recurrent periods of rapid eye movements during sleep. Moreover, when subjects were awakened during this particular physiological state called "Rapid-Eye-Movement sleep" (REM sleep) or paradoxical sleep [5], they reported vivid dreams [6, 7]. This discovery provided the basis for a new field of research to emerge since sleep was not a homogeneous state of relative neuronal quiescence any more, but included periods of increased neurophysiological activity underlying the production of dream experiences. Previously supposed to be restricted to REM sleep, dreaming, however, seems to occur also during non-REM sleep [8]. Nevertheless, most studies about human dream organisation have focused on REM sleep, because dreams during this sleep stage are more frequent, longer, more vivid and contain more bizarre features [9].

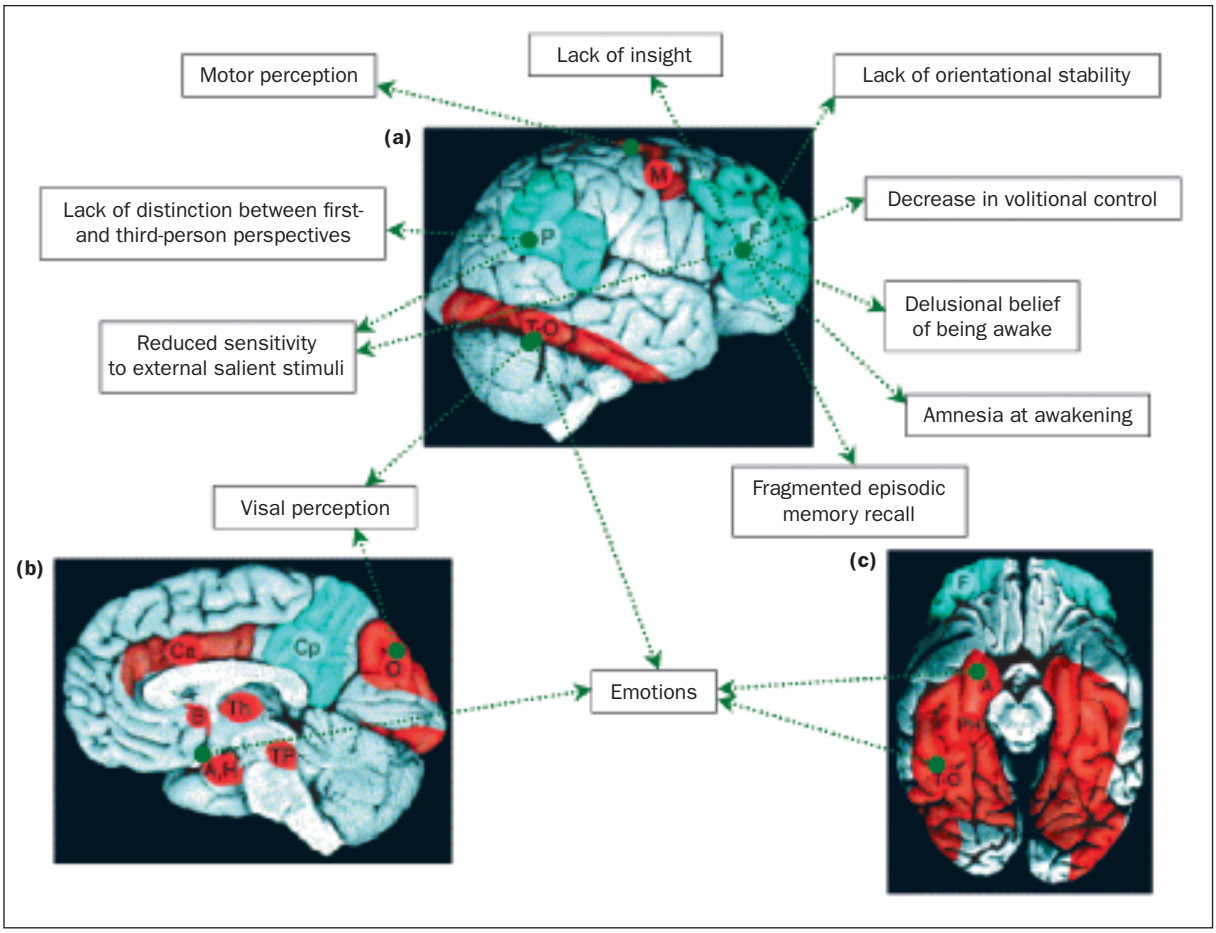
Following the seminal description of REM sleep by Aserinsky and Kleitman, neurobiological studies have further clarified the relationship between brain activity and dreaming during REM sleep [2, 10]. Animal studies and human psychopharmacological data suggest that REM sleep neurophysiology is dominated by neuromodulatory changes [2, 9]. In particular, REM sleep is generated by cholinergic processes arising from brain-stem structures, located in the pediculopontine tegmentum (PPT) and laterodorsal tegmentum (LDT) [11–18]. Cholinergic REM-sleep generation is facilitated by the decrease of aminergic (noradrenergic [NA] and serotoninergic [5-HT]) inhibition on the cholinergic generators [19–25]. Other neuromodulatory systems, such as GABA [26], NO [27], glutamate [28], glycine [29], neuropeptides [30], orexin [31], and other non-pontine structures, such as basal forebrain [32], hypothalamus [33], thalamus [34, 35], amygdala [36], periaquaeductal grey area [37] and medulla [38], might also participate in REM-sleep modulation. Dream research in humans has greatly benefited from the integration of neuropsychological analysis of dream experience, observations of patients with brain lesions and functional neuroimaging techniques. In this article, we will review available functional imaging data that described the mapping of regional cerebral activity during REM sleep. We will then discuss how these results may relate to dreaming experience and also how future neuroimaging studies should help refine such cerebral correlates of dreaming in humans.

Functional neuroanatomy of normal human REM sleep

During the last decade, positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) were used to describe the functional neuroanatomy of normal human REM and non-REM sleep.

REM sleep is characterised by sustained neuronal activity [39, 40], high cerebral energy requirements [41] and cerebral blood flow [42, 43]. Several human PET studies described REM-sleep regional

Figure 1



Schematic representation of the relative increases and decreases in neural activity associated with REM sleep. Regions coloured in red are those in which rCBF increases during REM sleep; those in blue correspond to rCBF decreases. Green arrows show the proposed relationships between brain areas and several dreaming features which may be accounted for by regional patterns of activity during REM sleep. (Adapted from *Trends in Cognitive Sciences*; Vol. 6 [1]; Schwartz S, Maquet P. Sleep imaging and the neuro-psychological assessment of dreams. p. 23–30; Copyright 2002, with permission from Elsevier.)

(a) lateral view; (b) medial view; (c) ventral view.

A = amygdala; B = basal forebrain; Ca = anterior cingulate gyrus; Cp = posterior cingulate gyrus and precuneus; F = prefrontal cortex (middle, inferior and orbito-frontal cortices); H = hypothalamus; M = motor cortex; O = occipital-lateral cortex; P = parietal cortex (inferior parietal lobule); PH = parahippocampical gyrus; Th = thalamus; T-O = temporo-occipital extrastriate cortex; TP = pontine tegmentum.

patterns of activity compared to wakefulness and/or non-REM sleep, using $H_2{}^{15}O$ or ^{18}FDG [44–48] (fig. 1). Activation of pontine tegmentum and thalamic nuclei were reported [44, 45], in agreement with REM-sleep generation mechanisms in animals [11, 35, 49]. Another consistent feature was the activation of limbic and paralimbic structures, including amygdaloid complexes, hippocampal formation and anterior cingulate cortex [44–46]. This finding was suggested by earlier evidence for a high regional glucose metabolism in the limbic system of rats [50] and cats [51, 52]. Animal data show that the amygdala plays a role in REM-sleep modulation. For example, ponto-geniculo-occipital (PGO) waves, a major component of REM-sleep phasic endogenous activity, were increased in cats by electrical stimulation of the central nucleus of amygdaloid complexes [53], while carbachol (cholinergic agonist) injections in the same nucleus enhanced both REM sleep and PGO activity [54]. Moreover, a shift from non-REM-sleep to REM sleep was observed in rats after serotonin injection into the amygdala [36]. The amygdala also seems to modulate other key features of REM sleep. For instance, the large variability in heart rate during REM sleep could be explained by a prominent influence of the amygdaloid complexes (Desseilles et al., submitted).

The hippocampal formation was also found activated during REM sleep [46], suggesting an activation during this sleep stage of the whole limbic system rather than the amygdala alone. Both amygdala and hippocampal formation are critical for memory systems [55] and may thus participate in the processing of memory traces during REM sleep. These relationships between sleep and

memory processes have extensively been studied at multiple levels and are reviewed elsewhere [56–60].

Activation of temporo-occipital areas was also observed [44], confirming earlier neuroimaging results [41, 43]. These areas included inferior temporal cortex and fusiform gyrus, which are extrastriate cortices belonging to the ventral visual stream. In addition, the functional relationship between posterior cortical areas appeared to be different during REM sleep when compared to wakefulness [61]: extrastriate cortex activation was significantly correlated with primary visual cortex (striate cortex) deactivation during REM sleep, while their activities are usually positively correlated during wakefulness. Along with the concomitant paralimbic/limbic activation and association areas deactivation (see below), this pattern of functional connectivity is consistent with the model in which REM sleep allows internal information processing (between extrastriate and their paralimbic projections) in a closed system, dissociated from input (via striate cortex) or output (via frontal cortex) to the external world.

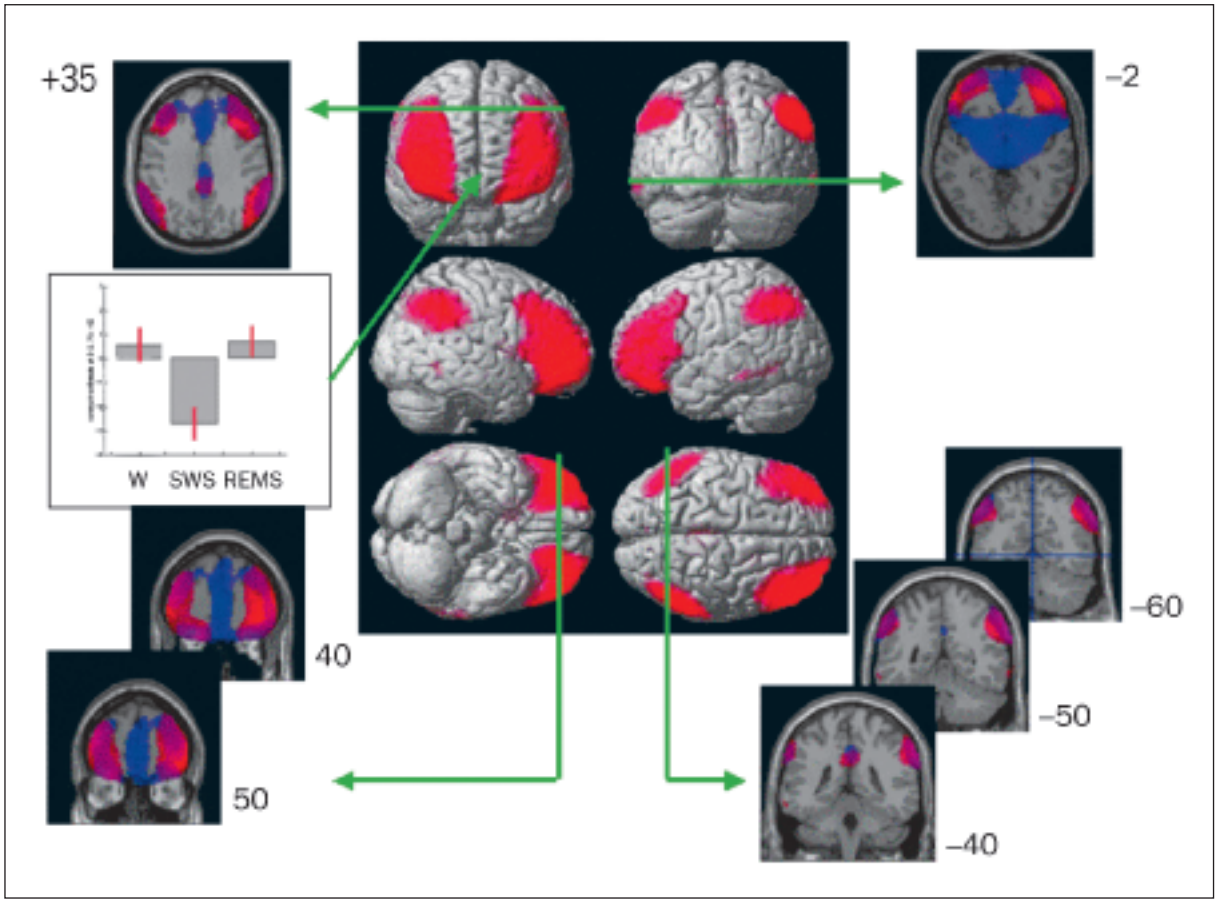
Activations during REM sleep were also observed, less reproducibly, in the basal forebrain, cerebellum and caudate nucleus [44]. However, the role of these structures in REM-sleep physiology remains highly speculative. Activation of the basal forebrain (anterior hypothalamus and caudal orbital cortex) [44] was interpreted as supporting the concept of a ventral cholinergic route from the brain stem through the basal forebrain, which may be involved in the processing of the ascending reticular activation during REM sleep. Activation of the cerebellar vermis [44] might reflect input from the brain-stem vestibular nuclei during REM sleep. Activation of the caudate nucleus [44, 46] might play a role in the ascending thalamocortical activation, as part of a network linking brain stem to basal ganglia via intralaminar thalamic nuclei and then proceeding to the cortex via the ventral anterior and ventromedial thalamic nuclei [44].

PET studies also showed a deactivation of dorsolateral prefrontal (DLPF) cortex, precuneus, posterior cingulate cortex and most of the parietal cortex during REM sleep [44, 45]. In a recent meta-analysis of a large set of PET data acquired during normal human sleep, we focused on the topography of these most deactivated areas during REM sleep compared to wakefulness [48]. It appeared that only parts of the parietal and frontal cortices were hypoactive during REM sleep, bilaterally: the temporo-parietal region, inferior parietal lobule, inferior and middle frontal gyrus of the DLPF cortex. In contrast, the activity in the superior parietal lobe and in the superior and medial prefrontal cortex are similar to waking levels (fig. 2). The reasons of these deactivations are still unclear. Interestingly, neuroanatomical data in monkeys showed that the cortical areas deactivated during REM sleep (parietal cortex, DLPF cortex) received only sparse inputs from the amygdala, while areas activated during REM sleep (anterior cingulate, right parietal operculum) received rich amygdalar inputs [62], suggesting that amygdalar complexes may modulate cortical activity during REM sleep. This assumption was further supported by the demonstration that functional interactions between the amygdala and the occipito-temporal cortices were different in the context of REM sleep than in non-REM sleep or wakefulness [63]. The amygdalo-cortical network during REM sleep might contribute in particular to the selective processing of emotionally relevant memories [45], but this hypothesis still deserves experimental confirmation.

Finally, besides these "canonical" maps of normal human REM sleep, other studies were focused on more dynamic interactions during REM sleep, such as reactivations of areas involved in learning prior to sleep. For instance, motor areas, especially premotor cortical areas, belong to the structures that were reactivated during REM sleep in subjects previously trained on a procedural motor learning task compared to non-trained subjects [47]. By showing this REM sleep-related reactivation of areas activated during task learning, the study suggests a reprocessing during REM sleep of memory traces acquired during previous wakefulness and thus gives further support for a role of sleep in learning and memory [56–60].

In summary, these studies show that not only the functional neuroanatomy [44–46, 48] but also the functional interactions between neuronal populations [61, 63] are different during REM sleep compared to wakefulness. The functional neuroanatomy maps show regional activations and deactivations. Regional activations are found in subcortical structures (including pons and thalamus), limbic/paralimbic areas (notably the amygdala) and posterior cortical areas. These results contrast with regional deactivations found in associative frontal (inferior and middle lateral prefrontal) and parietal (inferior parietal lobule and temporo-parietal region) cortices. As discussed above, many of these patterns of activity could be linked to REM-sleep neurophysiological mechanisms, mainly arising from animal data. Two additional systems should also be incorporated in this discussion about the physiological basis of the brain activity during REM sleep. Firstly, a unifying neurobiological hypothesis proposes a link between

Figure 2



Central panel: Representation of the brain areas significantly less active during REM sleep than during wakefulness (in red), including the lateral part of the prefrontal cortex (inferior and middle frontal gyri, orbito-frontal cortex) and the inferior parietal lobule.

(1st row) anterior and posterior views; (2nd row) lateral views; (3rd row) bottom and top views.

Left and right upper panels: Transverse sections 35 (left) and –2 (right) mm from the anterior-posterior commissural plane. The least active areas during REM sleep do not extend to the mesial frontal cortex. In contrast, the medial frontal cortex is one of the least active areas during slow-wave sleep (SWS) as compared to wakefulness (in blue). Purple areas indicate regions where rCBF is decreased in both SWS and REM sleep as compared to wakefulness.

Left lower panel: Frontal sections through the frontal cortex, at various distances from the anterior commissure (mm). The least active areas during REM sleep do not extend to the superior frontal gyrus or the mesial frontal cortex. The latter are significantly deactivated during SWS.

Inset (left middle panel): The adjusted cerebral blood flow in the mesial frontal area is similar during wakefulness and REM sleep and is decreased during SWS.

Right lower panel: Frontal sections through the parietal cortex, at various distances from the anterior commissure (mm). The least active areas during REM sleep involve only the inferior parietal lobule but do not reach the intraparietal sulcus or the superior parietal cortex.

brain functional mapping and neuromodulatory changes [2]. According to this view, changes in neuromodulation might participate in modifications of forebrain activity and responsiveness during REM sleep, as this sleep stage is characterised by a prominent cholinergic activity and a decrease in noradrenergic and serotonergic modulation [39]. However, no study has directly explored how neuromodulatory variations may influence the regional brain function during REM sleep. Secondly, the influence of PGO waves on regional cerebral activity during REM sleep may also be considered, as several observations suggest their existence in human sleep [64]. Evidence for such human PGO waves came from direct intracerebral recordings in epileptic patients [65], surface electroencephalography (EEG) [65], magnetoencephalography (MEG) [66] and, more recently, PET data [67]. Although PGO waves were most easily recorded in the pons [68], the lateral geniculate bodies [69] and the occipital cortex [70], they were also observed in many other parts of the cat brain [71], including limbic areas (amygdala, hippocampus, cingulate gyrus). Moreover, the generator of

pontine waves in rats, located in the dorsal part of the subcoeruleus nucleus, projects to a set of brain areas shown to be active during REM sleep, including occipital cortex, hippocampus, amygdala and brain-stem structures [72]. The integration of all these data supports the hypothesis claiming that activities of similar appearance as PGO waves may contribute to shape the distribution of regional brain activity during REM sleep in humans.

Towards the integration of canonical maps of REM sleep and predominant dream features

A straightforward way of interpreting neuroimaging data obtained during REM sleep is to consider possible relationships with dream features. Indeed, analyses of dream content have identified several specific features of dream phenomenology which appear to fit remarkably well with the distribution of brain activity during REM sleep [1, 2]. Numerous interpretations have been proposed, but one should be aware that these assumptions remain speculative and still deserve further experimental confirmation. These hypothesised cerebral correlates of dreaming could be summarised as follows and are illustrated in figure 1.

Perceptual features are essential characteristics of dreams, and the respective frequency of the different sensory modalities represented in dreams is quite stable across studies: visual components are literally always present, auditory components are present in 40 to 60% of dreams, movement and tactile sensations in 15 to 30%, and finally smell and taste in less than 1% [73]. The activation of posterior (occipito-temporal) cortices may thus be related to the perceptual aspects of dreams, consistently dominated by visual and auditory elements [44]. Interestingly, a cessation of visual dream imagery was reported in some patients with occipito-temporal lesions [74] (see also article by Schwartz et al. in this issue).

The prominence of emotions, and especially negative emotions, such as fear and anxiety, is almost inherent to a dream narrative [73]. It is known that the amygdala plays a central role in the modulation of responses to threatening stimuli or stressful situations during wakefulness [75]. Moreover, a recent fMRI study conducted during wakefulness emphasised the positive relationship between emotional intensity of visual stimuli and both amygdalar and infero-temporal cortex activity. Additionally, a strong linear correlation was found between activity in these two structures across picture contents [76]. Together, these data suggest that the high limbic activity during REM sleep, and especially amygdalar activation [45], could be related to the high emotional load of dream contents. The organisation of emotional experience during dreams may not imply one structure alone but a network of functionally connected areas including the amygdala and extrastriate cortices [63].

Motor activity is also very important in dream contents [77]. During normal REM sleep, no coordinated motor behaviour is observed because of muscular atonia. However, such atonia can be removed after ponto-medullary lesions in cats, causing the animals to seemingly "act out" their dreams [78]. Likewise, human patients suffering from REM-sleep behaviour disorder display a coordinated motor behaviour during REM sleep, which is often related to the dream narrative reported upon awakening [79]. In healthy subjects, although not resulting in active motor behaviour, these coordinated activities might be programmed centrally, in the motor and premotor cortical areas, as suggested by PET data [47].

The hypoactivity of prefrontal cortex is usually considered as explaining the lack of insight, the alteration in time perception, the delusional belief of being awake during dreams and the amnesia at awakening [2]. The recent reassessment of deactivated cortical areas during REM sleep, confining them to the inferior and middle lateral prefrontal cortex and inferior parietal lobule [48], has added new elements of discussion in terms of dreaming features (fig. 2).

Considering the prefrontal cortex, its functional architecture has recently been described as a cascade of three nested levels of processing underlying cognitive control and which are topographically segregated into distinct subregions [80]. Firstly, the premotor cortex would account for sensory control, i.e. the selection of stimulus-response associations. Secondly, the caudal lateral prefrontal cortex would subserve contextual control, i.e. the selection of premotor representations according to contextual signals accompanying stimulus occurrences. Thirdly, the rostral lateral prefrontal cortex would subserve episodic control, i.e. the selection of caudal lateral prefrontal cortex representations according to events that occurred previously or according to ongoing internal goals. The prefrontal regions deactivated during REM sleep overlapped with the regions supporting the so-called contextual and episodic control, suggesting that these two control levels would be less efficient during REM sleep while the sensory control would be maintained [48]. This pattern of activity could then be linked to several features of dream reports after awakening from REM sleep. Indeed, it would

explain the lack of "orientational stability", i.e. the fact that the dreamer is generally unable to integrate information of a whole episode, and the "persons, times and places are fused, incongruous and discontinuous" [2]. It may also account for the decrease in volitional control and the failure to organise one's mental representation toward a well-identified internal goal and to "control the flow of dream events" [9].

These prefrontal areas, hypoactive during REM sleep, also overlapped with frontal regions that were reported to have a role in episodic memory. This memory system refers to the ability to encode and recollect personally experienced events set in a particular spatio-temporal context [81]. Functional neuroimaging data reported activation of lateral prefrontal cortices during episodic memory retrieval [82–86]. Prefrontal areas would indeed participate in the monitoring of episodic memory retrieval, for instance, by checking the accuracy and completeness of the processed information [48]. The deactivation of these areas during REM sleep might explain that, while 65% of dream reports contain residues of recent waking activity, only 1.4% of them are considered as representing the replay of full memory episodes [87]. Therefore, episodic elements might be reactivated in a fragmented fashion during dreams because the hypoactivity of the lateral prefrontal cortex would prevent the various details of past events to be integrated into an identifiable life episode [88].

The inferior parietal lobule and the inferior and middle frontal gyri are also included in the so-called ventral attentional network [89]. Although bilateral, the hypoactive areas were more extended on the right hemisphere, as is the ventral attentional system. The activity in this system is increased in response to targets occurring at unexpected locations or to low frequency targets, mostly when relevant to the current task [90]. This network is therefore specialised in the detection of salient, unexpected, behaviourally relevant stimuli, and helps to reorient the focus of attention. It acts as an alerting mechanism when salient stimuli arise outside the present focus of processing, interrupting the current attentional set and shifting attention toward the incoming stimulus [48]. It was suggested that the activity in this ventral network would be modulated by the locus coeruleus (LC), as this structure is involved in selective attention, especially to salient and unexpected stimuli [91], and has important projections to the inferior parietal cortex [92]. During REM sleep, the firing rate of noradrenergic LC cells drops significantly and parietal areas are thus deprived from a critical positive modulation, therefore predicting a possible quiescence of the ventral attentional network during this sleep stage. In line with this hypothesis, the focus of attention should be less sensitive to external salient stimuli during REM sleep than during wakefulness. Although difficult to assess experimentally during REM sleep, the focus of attention may be reflected through dream reports. Thus it may be predicted that the dream narrative, reported after awakening from REM sleep, would hardly be modified by external stimulation, even if behaviourally relevant [48].

An important aspect of prefrontal activity during REM sleep is that the medial prefrontal cortex remained as active during REM sleep as during wakefulness (fig. 2). This stands in contrast with its significant deactivation during non-REM sleep [48, 93]. This area is involved in the ability to attribute intentions, thoughts and feelings to oneself and to others [94], generally described by the so-called "theory of mind" [95, 96]. In other words, it is an inductive reasoning allowing the interpretation and understanding of others' actions and speech, and the prediction of their behaviour. The similar level of activity in the medial prefrontal cortex during REM sleep and wakefulness could thus be hypothesised as contributing to mind representation during REM sleep [48]. Indeed, mind representation may be a key feature of oneiric experience: dreaming usually appears as a perceptual narrative involving multiple characters who are credited with thoughts, emotions and intentions by the dreamer him- or herself. Moreover, the inferior parietal lobule and temporo-parietal junction in the right hemisphere are parts of this network involved in mind representation during wakefulness and would also be involved in the distinction of first- versus third-person perspective in the representation of action, mind and emotion [97–101]. Contrasting with this medial prefrontal preserved activity, the hypoactivity in the right temporo-parietal junction might be related to a lack of distinction between first- and third-person perspectives during REM sleep [48]. In line with this hypothesis, dream reports show that the self can take part in the dream scenario both in a first-person (the self sees and acts) and in a third-person perspective (the dreamer sees the self acting in the dream).

Conclusions and perspectives

The integration of experimental results extracted from different levels of analysis seems to be an optimal and promising approach for the understanding of dream physiology. In particular, we sug-

gest that future functional brain imaging in humans should be combined with cognitive assessment and neuropsychological analysis of dream reports to test hypotheses about the brain correlates of dream features [1].

In the present article we reviewed several lines of evidence indicating that "canonical" maps of regional cerebral activity during REM sleep might underlie the generation of typical dreaming features. For instance, amygdala activation is consistent with the predominance of threat-related emotions. Temporo-occipital activation is in keeping with visual dream imagery. And associative prefrontal and parietal deactivation is suggestive of the lack of orientational stability and insight, the fragmented episodic memory recall and the reduced sensitivity to external relevant information (fig. 1).

However, dreaming involves more than experiencing broad categories of sensory representations or emotions during sleep. Dreams are multifarious and often bizarre. Can we relate the singularity of certain typical dream experiences to brain activity? Recent dream research demonstrated that some bizarre but common dream features of normal human sleep share strong similarities with neuropsychological syndromes caused by lesions in distinct brain regions [1] (see article by Schwartz et al. in this issue). Based on this idea, it has been proposed that the categorisation of dream reports grounded on the presence of specific bizarre features in dreams could offer useful constraints to the analysis and interpretation of REM-sleep data acquired just before dream reports [1]. Furthermore, the complexity and richness of visual experiences during REM sleep strongly suggests that activity within the visual ventral stream might be spatially more heterogeneous at any given time than what neuroimaging studies could find until now. This might explain why the activation of temporo-occipital areas is not systematically reported in functional neuroimaging studies [44, 45].

Overall, the question arises whether neuroimaging techniques could contribute to a comprehensive theory of dreaming. Will neurobiological theories of dreaming ever be able to satisfactorily address the infinity of questions arising from the study of oneiric behaviour, and especially the crucial and immensely debated point of dream functions? At present, it appears that any theory of dreaming is condemned to remain largely speculative and partial, notably because combined dream and functional imaging data are still very sparse. However, we anticipate that much insight would be gained if the content of dreams was systematically quantified in terms of explanatory variables to model neuroimaging data. This implies the use of scales to parameterise the dream narrative in order to assess its different perceptual, emotional or bizarre elements to provide genuine functional maps of the dreaming brain. Finally, little is known about the physiology of non-REM-sleep dreaming and future neuroimaging studies should also attempt to link dreaming experiences during this sleep stage with patterns of regional cerebral activity.

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