Dreaming and Unilateral Brain Lesions: A Multiple Lesion Case Analysis

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Lesions causing cessation of dreaming are thought to be more frequently left hemispheric than right hemispheric. However, reports of this phenomenon have not excluded epileptic cases and have not reported handedness, etiology of the lesion, lesion location, comorbidity, gender, age, and so forth, on a case-by-case basis. Some authors were also concerned about aphasia being a cause of dream loss and its lateralization, but they never measured its impact statistically. The present investigation reviews cases of post lesion dream cessation that answered to strict criteria for testing hemispheric lateralization and the effect of aphasia on it. In the 31 subjects, left hemisphere lesions were significantly more frequent than right, as predicted, but the left hemisphere lesions were very often associated with aphasia. Nonaphasic cases of total dream loss had lesions equally often in the right and in the left hemisphere. It is proposed that aphasia deprives patients of a second dream-encoding system, which is important enough to induce amnesia of dream occurrence.

KEY WORDS: dreaming, right, left, lesions, hemispheric specialization, lateralization, aphasia

Unilateral brain lesions causing dream cessation are reviewed here to determine lateralization, controlling for several other variables. Charcot (1883) was the first to describe a patient who lost all capacity for visual imagery, awake as well as in his dreams, because of a brain lesion. The patient also had color agnosia and what Charcot called verbal blindness (*cécité verbale*), a concept seemingly regrouping visual agnosia and mild alexia. Wilbrand (1892) described a patient with complete loss of dreaming, prosopagnosia, simultagnosia, and no color agnosia. These two cases were blended in the literature to form Charcot-Wilbrand syndrome. This syndrome was recently challenged by Solms (1997), who despite a certain overlap between the two cases, dissociates them in two distinct pathologies.

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The Charcot variant consists of cessation or reduction in visual dream imagery, whereas the Wilbrand variant consists of complete cessation of dreaming. Since the publication of these two cases, Doricchi and Violani (1992) reviewed 102 additional cases of dream reports following brain injury published in French, English, German, or Italian, while Solms (1997) reviewed 109 cases. However, these cases are often incomplete, and both authors analyzed more modest samples (Doricchi & Violani: 70 cases, Solms: 73 cases). These two extensive reviews are discussed later.

THE LATERALITY OF DREAMING

We distinguish three basic interpretations of laterality effects in the lesion literature on dreaming. A right hemisphere proposal, elaborated by Galin (1974) and Broughton (1975), states that dreams are generated in the right hemisphere. Galin based this assertion on a few selected cases with a right hemisphere lesion, as well as on hemispheric specialization of cognitive modalities. He suggested an analytical, logical mode for the left hemisphere and a holistic, gestalt mode for the right. Noting the sometimes nonlogical and vivid visual nature of dreams, he proposed a right hemisphere localization of dream generation. Joseph (1988) further developed the argumentation for the right hemisphere proposal of dream laterality based on a nonsystematic review of cases with loss of dreaming and an amalgam of various hemispheric asymmetry theories. The main basis of this right hemisphere specialization for dreams in Joseph's argumentation was the right hemisphere's high level of arousal during rapid eye movement (REM) sleep. The association between REM sleep and dreams has been almost a constant in the dream literature since the work of Jouvet (1962), who discovered brainstem mechanisms that control REM sleep. However, Solms (2000) showed clearly that loss of dreaming can occur without loss of REM sleep after forebrain lesions sparing the brainstem. The fact that REM sleep is associated with brainstem arousal and right hemisphere activation does not mean that dreams are generated in the right hemisphere. Dreams occur during non-REM sleep, just as REM sleep can happen without dreams. Joseph also dismisses reports of dream loss after left hemisphere lesions as aphasia artifacts, stating that "[. . .] when the left hemisphere has been damaged, particularly the posterior portions (i.e., aphasic patients), the ability to verbally report and recall dreams also is greatly attenuated (p. 641)." This point will be discussed later.

A left hemisphere proposal is more recent and states that the left hemisphere is sufficient to provide dream generation. Greenberg and Farah (1986) reviewed nine cases of complete cessation of dreaming and concluded that in all cases, the dominant hemisphere for speech was damaged. No theoretical explanation was offered. Solms (1997), however, pointed out that this study was too restrictive, because the authors excluded some cases of right hemisphere lesion on the basis that the dream cessation was not complete and ignored others on the basis of language (German).

A third proposal positing a hemispheric interaction states that both hemispheres have a role to play in dream generation and recall. Kerr and Foulkes (1981) believe, for example, that the right hemisphere modulates visual integration in dreams, whereas the left modulates the narrative content. Antrobus (1987) devel-

oped a similar model of hemispheric specialization of dream generation. Doricchi and Violani (1992) and Solms (1997) agree, based on extensive reviews of the literature, that the relationship between hemispheres and dreams is more complicated than it first appeared. Although the literature presents more cases of left hemisphere lesion associated with loss of dreaming, there have indubitably been cases with only right hemisphere lesions. Doricchi & Violani propose that the right hemisphere provides the base material for the dreams, whereas the left hemisphere provides the means of decoding it. As such, a lesion in either hemisphere could be sufficient to cause dream loss. Ramachandran (1996) postulates a similar model based on his work on anosognosia. In short, he proposes that the left hemisphere tries at all costs to maintain a coherent model of reality, confabulating and repressing conflictual information to do so, whereas the right hemisphere is an "anomaly detector" that forces a paradigm switch when the inconsistencies between reality and the model elaborated by the left hemisphere are too great. He proposes that during dreams, the right hemisphere is allowed to try to incorporate elements that do not fit the model without the left hemisphere blocking it. If the new information can be integrated in the reality model, the left hemisphere accepts it. If it does not fit, it is forgotten after sleep, like most dreams. There is another dimension of dreams that could solicit complementary contributions from each hemisphere. Dreams with highly negative content could be generated in the right hemisphere, whereas those with positive content could be generated by the left. This would be in accordance with the "emotional valence" model of hemispheric specialization (Demaree, Everhart, Youngstrom, & Harrison, 2005). This eventuality is not about to be resolved, because there are too few reports of dream content following unilateral lesions or during unilateral epileptic ictae. We found single case reports of 19 patients with a unilateral epileptic focus, no evidence of a lesion, and ictal dreams (the patient woke up from a seizure experiencing a dream). Of these, the content of the dreams was described in 16 cases. The dreams were described as night terrors, nightmares, or as comprising strongly negative emotions in 13 of the 16 cases (Epstein, 1967, 1979; Epstein & Freeman, 1981; Huppertz, Franck, Korinthenberg, & Schulze-Bonhage, 2002; Ide, Mizukami, Suzuki, & Shiraishi, 2000; Lombroso, 2000; Montplaisir, Laverdiere, Saint-Hilaire, Walsh, & Bouvier, 1981; Reami, Silva, Albuquerque, & Campos, 1991; Rodin, Mulder, Faucett, & Bickford, 1955; Snyder, 1958;). The epileptic focus was in the right hemisphere in 9 of the 13 cases with unpleasant dreams (nonsignficant) and all three cases with emotionally "neutral" dreams (Alliez, Roger, & Miaille, 1978; Epstein & Ervin, 1956; Reami et al., 1991). Of the four cases with undescribed dream content, two had a left focus and two a right focus (Cirignotta et al., 1975; Harvey & Barnes, 1996; Silvestri & Bromfield, 2004). Mendez and Doss (1992) reported a case of right temporal lobe epilepsy with a normal magnetic resonance image who complained of repetitive morbid dreams. In short, epilepsy data suggest that night terrors and perhaps also morbid dreams might indeed originate primarily from the right hemisphere. However, more cases are needed to confirm the trend. Unfortunately, epilepsy data can tell us nothing about whether pleasant dreams originate from the left hemisphere, because unusually pleasant dreams are not reported in the literature.

GROUP STUDIES OF POST LESION DREAM CESSATION

As Solms (1997) pointed out, dream cessation after brain surgery is not a rare occurrence, at least when the frontal lobes are involved. He reviewed eight articles on groups of patients undergoing leucotomy. In every group, at least some of the patients reported cessation or reduction of dreaming. Jus et al. (1973) compared 13 patients with schizophrenia having undergone prefrontal lobotomy with 13 patients with schizophrenia who had not on immediate dream recall following REM awakening. They found a notable lack of dream report in lobotomized patients when compared with the nonlobotomized group (χ^2 test, $p < .001$). There is no indication, however, that a unilateral locus for dream generation or recall is involved is this study. Cathala and Laffont (1981) compared patients with either a frontal lesion (seven patients) or a parietal lesion (nine patients) with equivalent control groups on immediate dream recall following awakening from REM sleep. They found a decrease in both dream frequency and dream description richness only for parietal patients compared with their control group (χ^2 test, $p < .01$). No difference was found between frontal patients and their control group.

Arena, Murri, Piccini, and Muratorio (1984) compared 52 patients with a unilateral hemispheric brain lesion and 18 patients without a brain lesion, paired for age and educational level, on dream recall one hour after awakening. They also measured short- and long-term, spatial and verbal memory in all participants. They found a higher proportion of nonrecallers in patients with right hemisphere lesion (χ^2 test, $p < .01$) and left hemisphere lesion (χ^2 test, $p < .05$) than in the control group. The difference between right hemisphere and left hemisphere lesion groups did not reach significance. Memory test results did not differentiate recallers from nonrecallers, excluding a pure memory deficit sufficient to explain dream recall problems in nonrecallers. A later study from the same group (Murri, Arena, Siciliano, Mazzotta, & Murtorio, 1984) focused on the differences in dream recall as a function of lesion localization in the antero-posterior axis. They studied 53 right-handed patients with unilateral lesion on dream recall one hour after awakening. The control group consisted of 28 patients hospitalized in the same ward. After 10 consecutive days of testing, they found that nonrecallers (defined as those who did not report any dream at all for the whole 10 days) were more frequent in the group of posterior right lesions than anterior right lesions (Fisher's exact test, $p < .02$), as well as more frequent in the group of posterior left lesions than anterior left lesions (Fisher's exact text, $p < .005$). There was no significant difference as a function of side of the lesion. Conscious of the potential flaws of morning dream recalls, Murri, Massetani, Siciliano, Giovanditti, and Arena (1985) compared morning diary reports with REM awakening reports in another group of 19 patients with unilateral hemispheric lesion (8 left, 11 right). They divided their subjects on the basis of lesion localization, either inside the temporo-parieto-occipital (TPO) region (defined as a lesion involving one, two, or three of these lobes) or outside the TPO. They validated the morning dream recall technique, because there was no significant difference in dream recall between the REM awakening and the morning interviews. They also found that lesion localization inside the TPO discriminated between the recallers (36% of lesions inside the TPO) and nonrecallers (79% of lesions inside the TPO) (Fisher's exact text, $p < .05$). Once again, there was no difference in dream recall as a function of hemisphere.

Some factors could account for this nonlateralization in group studies. (1) The patients are usually tested for a very short time, only a few days or weeks. It can be hypothesized that some patients would have remembered at least one dream if given a longer period. (2) Patients in group studies are usually in the acute phase of the brain insult. They are recruited on the hospital ward and so have not had time to recover from certain widespread stressors on the brain (e.g., edema, etc.). It would be interesting to know how many of these patients recovered their dream capability after a few weeks or months' time. (3) Patients in group studies represent a range of deficit that is not necessarily pathological. In such cohorts, it is generally not known whether there is any pathology at all because there is usually not a normal control group, or it is obvious from psychometric tests there is really not much pathology. Single case studies, on the other hand, report cases with severe deficits or striking manifestations. When selected with strict inclusion/exclusion criteria, the multiple case analysis approach to hemispheric specialization appears more promising to us, overall, than the group study method, even prospective.

MULTIPLE CASE REVIEW STUDIES OF DREAM CESSATION

Two extensive reviews of the literature of published cases of dream cessation after brain injury can be found in the literature. The first reviewed 104 cases (Doricchi & Violani, 1992). The second reviewed 111 (Solms, 1997). Solms' review is in his PhD dissertation (1991) along with an original study of dream disorders in 361 brain-injured patients. In this review, Solms listed 45 cases of brain-injured cases presenting with a complete loss of dreaming. From these, 31 had unilateral lesions, 28 of which were right-handed. It is well known that left-handers often present with a different brain organization, at least regarding dominance for speech. Thus, when handedness is known, left-handed cases will always be excluded from the analysis or, if they are in sufficient numbers, treated in a separate analysis. Twenty-two dream nonrecallers presented with left hemisphere lesions, whereas only six had unilateral right hemisphere lesions. That bias in favor of the left hemisphere is highly significant (binomial, $p = .004$). However, these data must be interpreted with caution because no mention was made in this review of seizures or epilepsy in the table listing the cases. Solms provided individual descriptions of a few patients with global cessation of dreaming in his dissertation. Of the seven cases described with global cessation of dreaming, two had seizures or epilepsy. We assume that other patients in the "global cessation of dreaming" group also presented epilepsy in addition to lesions, so inferences regarding hemispheric specialization remain tenuous with these cases.

Doricchi and Violani (1992) analyzed 104 patients with brain lesions reporting information about their dreams. From these, 43 reported complete cessation or important reduction of dreaming. From these, 31 were unilateral and 2 were ambidextrous (one had a left-sided lesion and the other a right-sided one). From the 29 remaining cases, 21 had a left hemisphere lesion, whereas only 8 had a unilateral right hemisphere lesion. This laterality effect reaches statistical significance (binomial, $p = .05$, two-tailed). However, they found this laterality effect only in the posterior region of the brain (parietal and occipital lobes), frontal lesions manifesting no laterality effect. Despite their own findings, the authors doubted

that the relationship between damage to the dominant hemisphere and loss of dreaming is real. They suspected a bias of "clinical relevance" toward the left hemisphere in the literature. However, it is worthy to note that in their reviews of the literature neither could find a single case of unilateral lesion in the occipito-temporal region confined to the right hemisphere, only cases with bilateral or left hemisphere lesions. In addition, a few cases of near complete right hemispherectomy were studied in the sleep laboratory with multiple awakenings. Dreaming was intact (McCormick et al., 1997). The total absence of right posterior (occipital) cases is striking and can hardly be dismissed as a bias in the literature.

LIMITATIONS OF PREVIOUS MULTIPLE CASE REVIEW STUDIES ON POST LESION DREAM CESSATION

Because Solms's and Doricchi and colleagues' reviews did not describe individual subjects, there are a multitude of issues that remain pending and that cast doubt on the laterality findings observed. Epilepsy was a common comorbidity and was not analyzed statistically. Some patients could have been suffering from irritative lesions. Age and gender were not analyzed. Dream reduction was not absolute in all patients. Presence of aphasia was not treated quantitatively.

In the context of an inference test of a hemispheric specialization model of dreaming, etiology of the lesion, handedness, presence of epilepsy, gender, and age need to be at least controlled statistically. Etiology of the lesion is important because certain etiologies (particularly virulent tumors, for example) can activate more than inactivate the hemisphere (Lisanby, Kohler, Swanson, & Gur, 1998). Handedness is important because it is probably related to most types of hemispheric specialization (Hugdahl, 2000). Presence or absence of epilepsy is important because an epileptic focus is most often irritative, thus canceling out the tissue loss effect of lesions. Gender is important because men and women differ in most expressions of hemispheric specialization (Grabowska, Herman, Nowicka, Szatkowska, & Szelag, 1994). Age is important because hemispheric specialization is weaker in juveniles than in adults (Montour-Proulx et al., 2004). In the context of any model of hemispheric specialization, patients presenting subjective complaints of minor anomalies ought not be considered reliable.

PROBLEMS IN STUDYING DREAM CESSATION

Dream cessation or reduction in frequency occurs often after a brain injury, and it is probable that neither the patient nor the clinician typically investigates it. A reduction in dream frequency would not be perceived as a major issue by the patient compared to other deficits a brain injury can cause, so he or she would not be likely to report it, even if he or she were aware of it. Most clinicians probably reason the same way. Compared with language or motor problems, reduction of dream frequency would be viewed as minor, even if the clinician were aware of it. Patients would probably note an *increase* in dream frequency even less, or, if they did, they would not complain about it. Epileptic patients, on the other hand, could

be more likely to note an increase in their dream frequency, or occurrence of an unusual dream, if the dream appears related to epileptic seizures.

A second problem frequently raised is that left hemisphere lesions cause language problems, and these could suffice to explain the left lateralization of dream cessation. Thus, it would be dream recitation or verbally mediated recall of the dream that would give the patient the impression that he or she has ceased to dream per se (Moss, 1972). Several authors (Epstein & Simmons, 1983; Doricchi & Violani, 1992; Joseph, 1988) have cautioned that dream loss could be a direct consequence of aphasic disorders (loss of the ability to narrate dreams and thus recall them). Solms (2000) reported that only 52% of the nondreamers reported in the literature were aphasics. Cathala et al. (1983) and Schanfald, Pearlman, and Greenberg (1985) also reported that some patients with aphasia do dream, and they sometimes even regain language in their dreams, Although aphasia could introduce a bias in the study of lateralization of dream loss, no authors tried to measure this relation statistically.

METHOD

We selected cases in the literature using two inclusion criteria: (1) subjects with exclusively unilateral lesions, radiologically or surgically confirmed and (2) cases of complete loss of dreaming. Presence of epilepsy was an exclusion criterion. When available, etiology of the lesion, age at the time of the lesion, gender, handedness psychiatric comorbidity, and presence of aphasia were noted. After exclusion of all the nonusable cases in light of the postulated model, 31 cases were retained for analysis.

RESULTS

Of the 31 unilateral lesion cases presented in the Table 1, 22 had a left hemisphere lesion and 9 had a right hemisphere lesion. The preponderance of left hemisphere lesions reached statistical significance (binomial: $p = .031$, two-tailed).

We believe left- and right-hemisphere lesions have equiprobable chances of occurring and of being selected in the absence of selection for dreaming disturbance. In their IQ study of 635 previously published unilaterally lesioned cases, Montour-Proulx and her colleagues (2004) collected 328 left hemisphere damage cases and 307 right hemisphere damage cases (binomial probability against equiprobabilty, $p > .12$, two-tailed).

SECONDARY ANALYSES (FOR CONTROL PURPOSES)

At first glance, the results reported earlier on unilateral lesioned nondreamers seem to go clearly in the direction of a left lateralization. However, several contaminating variables could challenge the main result. It is therefore important to determine whether the lateralization effect still stands when controlling for the effect of possible intervening variables.

Table 1. Cases With Unilateral Lesions Entailing Complete Dream Loss

Age/gender/ handedness	Lesion localization	Etiology, EEG, seizures	Neuropsychological symptoms	Dreams	Psychiatric comorbidity	Reference
63/M/R	Left parieto-occipital	Stroke, EEG not mentioned, no seizures mentioned	Anomia without agnosia, dyslexia, dysgraphia, loss of mental imagery	Complete loss of dreams	?	Basso, Bisiach, & Luzzatti, 1980
31/M/?	Left occipital	Tumor and lobectomy, EEG not mentioned, no seizures mentioned	Alexia without agraphia, finger agnosia, right-left disorientation	Complete loss of dreams beginning after operation	?	Nielsen, 1955
47/F/R	Left fronto-temporal	Hemorrhage, EEG not mentioned, no seizures mentioned	Broca's aphasia	Complete loss of dreams	?	Epstein & Simmons, 1983
35/F/R	Left frontal	Embolism following cardiac operation, EEG not mentioned, no seizures mentioned	Dysnomia, dysgraphia, dyslexia	Complete loss of dreams	?	Epstein & Simmons, 1983
56/F/R	Territory of the left middle cerebral artery	Thrombosis, EEG not mentioned	Anomia, alexia, dysgraphia, auditory comprehension problems	Complete loss of dreams	?	Epstein & Simmons, 1983
52/F/R	Territory of the left middle cerebral artery	Thrombosis, EEG not mentioned, no seizures mentioned	Broca's aphasia	Complete loss of dreams	?	Epstein & Simmons, 1983
43/M/R	Territory of the left middle cerebral artery	Thrombosis, EEG not mentioned, no seizures mentioned	Broca's aphasia	Complete loss of dreams	?	Epstein & Simmons, 1983
33/M/R	Left fronto-temporal	Left middle cerebral artery thrombosis, EEG not mentioned, no seizures mentioned	Broca's aphasia	Complete loss of dreams	?	Epstein & Simmons, 1983
59/F/R	Left fronto-parietal white matter	Left middle cerebral artery thrombosis, EEG not mentioned, no seizures mentioned	Mild dyslexia and dysgraphia	Complete loss of dreams	?	Epstein & Simmons, 1983
56/F/A	Left posterior	Stroke, EEG not mentioned, no seizures mentioned	Dysphasia, color agnosia, prosopagnosia	Complete loss of dreams, recovery after 19 months	?	Epstein, 1979
57/M/R	Right parieto-temporal	Infarct, EEG slowing over the right parieto-temporal region, no seizures mentioned	Left hemineglect, dressing apraxia, visuoconstructive deficits, topographical disorientation	Complete loss of dreams	?	Ettlinger, Warrington, & Zangwill, 1957

(*table continues*)

Table 1. (*continued*)

Age/gender/ handedness	Lesion localization	Etiology, EEG, seizures	Neuropsychological symptoms	Dreams	Psychiatric comorbidity	Reference
64/M/R	Left temporo-occipital	Stroke, EEG abnormal in the left parieto-occipital region, no seizures mentioned	Decreased visual imagery, decreased visual memory, color agnosia, alexia	Complete loss of dreams	Depressive symptoms	Farah, Lavine, & Calvanio, 1988
52/M/?	Right frontal	Stroke, EEG not mentioned, no seizures mentioned	Left hemiparesis, akinesia	Complete loss of dreams	?	Gloning & Sternbach, 1953
26/F/A	Right temporal hippocampus injured	Stroke, EEG slowing over the right posterior temporal region, no seizures mentioned	Topographical disorientation, decreased visual memory	Complete loss of dreams	?	Habib & Sirigu, 1987
67/M/?	Right lateral medulla	Infarct, EEG normal, no seizures mentioned	Ataxia, vestibular deficits, right facial paresthesia, transient insomnia, visual hallucinations	Complete loss of dreams followed by partial recovery	?	Hobson, 2002
32/M/L	Right parietal	Penetrating injury (mortar), EEG not mentioned, no seizures mentioned	Topographical disorientation, right-left disorientation,	Complete loss of dreams, recovery after five years	?	Humphrey & Zangwill, 1951
42/M/7	Left parieto-occipital	Resected tumor, infection, EEG not mentioned, no seizures mentioned	Dyslexia with dysgraphia, topographical disorientation, dyscalculia	Complete loss of dreams	Depressive symptoms	Lyman, Kwan, & Chao, 1938
47/M/R	Left temporo-occipital	Stroke, EEG not mentioned, no seizures mentioned	Alexia without agraphia, color agnosia, optic aphasia, optic apraxia, loss of visual imagery	Complete loss of dreams	?	Pena-Casanova, Roig-Rovira, Bern & Tolosa-Sa, 1985
77/M/R	Right parieto-occipital	Glioma, EEG not mentioned, no seizures mentioned	Slight left hemispatial neglect, slight paralexia, topographical agnosia	Complete loss of dreams	?	Solms, 1991
66/M/R	Right frontal	Stroke, EEG not mentioned, no seizures mentioned	Long term verbal memory deficits	Complete loss of dreams (previously poor recaller)	?	Corda, 1985 (from Doricchi & Violani, 1992)
74/M/R	Left frontal	Stroke, EEG not mentioned, no seizures mentioned	Mild motor aphasia, long term verbal memory deficits	Complete loss of dreams (previously poor recaller)	?	Corda, 1985 (from Doricchi & Violani, 1992)
56/F/R	Right parietal	Stroke, EEG not mentioned, no seizures mentioned	Long term verbal memory deficits	Complete loss of dreams (previously poor recaller)	?	Corda, 1985 (from Doricchi & Violani, 1992)

Table 1. (*continued*)

Age/gender/ handedness	Lesion localization	Etiology, EEG, seizures	Neuropsychological symptoms	Dreams	Psychiatric comorbidity	Reference
53/M/R	Right parietal, internal capsule	Stroke, EEG not mentioned, no seizures mentioned	Mild long term verbal memory deficits	Complete loss of dreams	?	Corda, 1985 (from Doricchi & Violani, 1992)
48/F/R	Left temporo-parietal	Stroke, EEG not mentioned, no seizures mentioned	Fluent aphasia, long term verbal memory deficits	Complete loss of dreams (previously poor recaller)	?	Corda, 1985 (from Doricchi & Violani, 1992)
63/M/R	Left parietal	Stroke, EEG not mentioned, no seizures mentioned	Wernicke's aphasia, long term verbal memory deficits	Complete loss of dreams (previously poor recaller)	?	Corda, 1985 (from Doricchi & Violani, 1992)
67/M/R	Left temporal	Stroke, EEG not mentioned, no seizures mentioned	Wernicke's aphasia, long term verbal memory deficits	Complete loss of dreams	?	Corda, 1985 (from Doricchi & Violani, 1992)
51/F/R	Left temporo-parietal	Etiology not mentioned, EEG not mentioned, no seizures mentioned	Long term verbal memory deficits	Complete loss of dreams	?	Corda, 1985 (from Doricchi & Violani, 1992)
52/M/R	Right temporo-parietal	Etiology not mentioned, EEG not mentioned, no seizures mentioned	Sensory aphasia, long term verbal memory deficits	Complete loss of dreams	?	Corda, 1985 (from Doricchi & Violani, 1992)
59/M/R	Left occipito-temporal	Stroke, EEG not mentioned, no seizures mentioned	Alexia without agraphia, visual anomia, loss of mental imagery	Complete loss of dreams	?	Michel & Sieri, 1981 (from Doricchi & Violani, 1992)
18/F/R	Left corpus callosum	Stroke + surgery, EEG not mentioned, no seizures mentioned	Alexia without agraphia, visual anomia	Complete loss of dreams	?	Michel & Sieroff, 1981 (from Doricchi & Violani, 1992)
24/M/R	Left temporo-occipital	Arterovenious malformation causing a hemeatoma	Deficit in memory for new information, no aphasia	Complete loss of dreams	?	Poza & Martí Massó 2006

Note. M = male; F = female; R = right-handed; L = left-handed; EEG = electroencephalogram; ? = not reported; A = ambidextrous.

Age

Normal aging is associated with a diffuse bilateral stress on the brain, which could add noise to the main result of the current study (the left lateralization of lesions causing dream cessation). Hemispheric specialization is also known to be weaker in juveniles than adults (Montour-Proulx et al., 2004). The correlation between age and side of lesion was not significant. No additional analysis was required.

Gender

Men with unilateral lesions typically outnumber women (more CVAs, more tumors, more head injuries, etc.; Montour-Proulx et al., 2004). Women are also known to be less lateralized then men in general (Miller, Jayadev, Dodrill, & Ojemann, 2005). For these reasons, it is important to determine the effect of gender on our main result. Gender was not related to side of the lesion. No additional analysis was required.

Handedness

Nonright handers usually show less structural asymmetries than right handers (Bear, Schiff, Saver, Greenberg, & Freeman, 1986). They are also known to show less hemispheric specialization for language (Isaacs, Barr, Nelson, & Devinsky, 2006). This variable could thus bring a caveat to our main result of laterality. There was, however, no link between handwriting preference and lesion side.

Lesion Locus

Several authors studied cerebral activation associated with dreaming. They generally found a hypometabolism of dorsolateral prefrontal cortex and of the associated parietal regions and activation of the pontine tegmentum, thalamus, limbic and paralimbic structures, and of the temporo-occipital lobes (Dang-va et al., 2005). In general, unilateral posterior lesions are also more common than anterior ones (Montour-Proulx et al., 2004). Because lesions could involve several lobes in the present review, and in order to keep replicates and cases concordant, we processed lobar lesion location lobe by lobe. In other words, a distinct analysis was carried out for each lobe. In the present study, the relation with lesion locus was not significant for any lobe.

Lesion Volume

Lesion volume could be asymmetrically distributed between the left hemisphere and right hemisphere damage groups because of sampling artifact, casting doubt on the meaning of a lesion laterality effect. For the present analysis, we determined the volume of the lesion by the number of lobes damaged. The interactions between the number of lobes damaged and the lesion side was significant: larger lesions were more frequently in the left hemisphere ($r = .472$; $p = .01$). In the small lesion group, there was almost an equal number of left hemisphere lesions ($N = 5$) and right hemisphere lesions ($N = 6$), whereas in the large lesion group, the effect of side of the lesion was highly significant ($N = 18$; $p = .001$). Small lesions could contribute noise to the lateralization effect. Worse, the left lesion preponderance in the present group could be a simple artifact of lesion size.

Etiology of the Lesion

Etiology of the lesion is important as mentioned earlier because certain etiologies, like tumors, can activate more than inactivate the hemisphere (Lisanby et al., 1998). The few tumor cases of the present study could bias the main result (lesion side). However, lesion etiology was in fact statistically unrelated to lesion side.

Psychiatric Comorbidity

Dreaming is affected in some psychiatric conditions. For example, dream frequency is lower during the depressive phase of bipolar patients than during the manic phase (Beauchemin & Hays, 1995). Patients with unipolar depression are also known to dream less than are nondepressive patients (Kramer, Whitman, Bladridge, & Lansky, 1966), whereas unipolar manic patients dream more (Bastos & Suerinck, 1963). In the present study, we controlled for the presence of mania and depression in separate analyses. Neither of these variables was significantly related to lesion side. No additional analysis was required.

Date of Publication and Presence of an Electroencephalogram (EEG)

The poorer resolution of the computed tomograph scan used in the 1970s compared to magnetic resonance imaging used nowadays could have brought more errors when localizing the lesion locus and laterality. Other methodology procedures could also have improved with years of research. However, since date of publication held no statistical relation with lesion side, the matter was given no further consideration.

The presence of an EEG in the case reports reduces chances of contamination by epileptic activity and contributes to the localization of the lesion. Presence or absence of an EEG did not relate significantly to lesion side and was, therefore, given no further consideration.

Presence of Aphasia

The left hemisphere is well known to be specialized for language (see Hutsler & Galuske, 2003, for a review). The presence of aphasic symptoms (anomia, dysphasia, aphasia, impaired oral comprehension) was of interest because of the possibility of a relation between dream recall and the use of language to verbalize the content of the dreams, as mentioned earlier. As we expected, the presence of aphasic symptoms was highly significantly correlated with lesion side ($r = .419$; $p < .019$). The relation between aphasia and lesion side exceeded the lesion side effect itself. In fact, among the 14 nonaphasic patients, there was an equal number of cases with right ($n = 7$) and left ($n = 7$) lesions. On the other hand, among the aphasic patients, the lesion side effect was, of course, highly significant ($p = .002$; see Table 2).

Table 2. Distribution of Lesion Side and Lesion Volume in Aphasics and Nonaphasics

Aphasia	Nonaphasics ($N = 14$)				Aphasics ($N = 16$)			
Side of the lesion	Right ($n = 6$)		Left ($n = 7$)		Right ($n = 2$)		Left ($n = 14$)	
Lesion volume	Small (n=5)	Large (n=1)	Small (n=1)	Large (n=6)	Small (n=1)	Large (n=1)	Small (n=4)	Large (n=10)

DISCUSSION

The results from 31 unilateral cases of complete dream cessation show that a lesion of the left or dominant hemisphere is significantly more likely to induce a cessation of dreaming than of the right. The current review replicates almost perfectly the results already obtained by Solms (1991) and Doricchi and Violani (1992) in their respective reviews of the literature on dream cessation after a cerebral lesion, but the case selection was optimized to control for several possible intervening variables.

The feasibility of attempting to study post lesion dream cessation has been questioned by several authors. A major problem frequently raised is that left hemisphere lesions often cause aphasia, and this could interfere with the ability to narrate dreams or to recall the dream verbally which would give the impression of a dream loss (Dorrichi & Violani, 1992; Epstein & Simmons, 1983; Broughton, 1982; Joseph, 1988; Moss, 1972). None of the authors studying post lesion dream cessation controlled their results for the presence of aphasia, despite their concern about contamination of dream recall by aphasia. The results of the current multiple case report shows that an important portion of the variance of the effect of left lateralization for dream loss is completely explained by the presence of aphasia and lesion size. The so-called cessation of dreaming may therefore consist more of an inability to use linguistic abilities to recall dreams. It even appears that aphasia can obliterate recall of any dream activity occurring at all in some patients.

To summarize, the detailed review of published case reports of the present study replicates the discrepant findings of both previous multiple case reviews and prospective group studies. However, it clearly explains the source of that discrepancy. Spontaneous complaints of total dream cessation are indeed more common in patients with large left hemisphere lesions. This laterality effect, however, is entirely explainable as an artifact of lesion size and aphasia. Unilaterally lesioned nonaphasic patients with complaints of total dream cessation have lesions in either hemisphere, indifferently. This is observed in all the prospective post lesion, laboratory-based dream recall studies carried out so far.

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