Tonus psychique, rêves et lésions cérébrales unilatérales : Une analyse de cas multiples

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Résumé

Les lésions cérébrales causant un arrêt des rêves sont le plus souvent rapportées à l'hémisphère gauche. Les cas rapportés, par contre, incluent souvent des patients épileptiques et ne font pas toujours mention de la dominance manuelle, de l'étiologie de la lésion, de la localisation précise de la lésion, des comorbidités, de sexe et de l'âge des patients, etc. Certains auteurs (Dorrichi & Violanni, 1992; Joseph, 1988; Solms, 1991) ont aussi soulevé l'hypothèse selon laquelle l'aphasie pourrait être une cause de la perte de rêve et de la localisation gauche de la lésion l'accompagnant le plus fréquemment, mais ils n'ont pas testé cette hypothèse de façon statistique. La présente étude analyse des cas de cessation de rêves suite à une lésion cérébrale répondant à des critères de sélection stricts pour tester la latéralisation hémisphérique ainsi que l'apport de l'aphasie dans ce phénomène. Dans les 31 cas retenus pour analyse, les lésions hémisphériques gauches étaient statistiquement plus fréquentes que les lésions hémisphériques droites, mais les lésions hémisphériques gauches étaient très souvent accompagnées d'aphasie. Les patients sans aphasie présentaient aussi souvent des lésions droites ou gauches. Il est proposé que l'aphasie prive les patients d'un système d'encodage des rêves assez important pour induire une amnésie des rêves.

Dreaming and unilateral brain lesions:

A multiple lesion case analysis

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Abstract

Lesions causing cessation of dreaming are thought to be more frequently left 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, etc., on a case by case basis. Some authors (Dorrichi & Violanni, 1992; Joseph, 1988; Solms, 1991) 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 which answered to strict criteria for testing hemispheric lateralization and the effect of aphasia on it. In the 31 cases, left hemisphere lesions were significantly more frequent than right, as predicted, but the left hemisphere lesions were very often associated with aphasia. Non aphasic cases of total dream loss had lesions equally often in the right as 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.

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Le modèle de tonus psychique de Braun (1999, 2002, 2004) est testé dans cet article sur la perte de rêves suivant une lésion cérébrale. Ce modèle postule que l'hémisphère gauche est responsable des augmentations du niveau d'énergie, ou tonus, psychique, alors que le droit cause des diminutions. Des lésions à l'hémisphère gauche causent, de façon générale, une diminution du niveau de tonus psychique, ceci dans les sphères comportementales, cognitives, émotionnelles et perceptuelles, alors que des lésions à l'hémisphère droit produisent l'inverse. Dans le cas des rêves, ce modèle postule que des lésions à l'hémisphère gauche ont plus de chance de causer des réductions ou des arrêts des rêves que celles à l'hémisphère droit. D'autres théories de spécialisation hémisphériques dans la génération des rêves ont été émises par le passé, souvent basées sur l'association entre sommeil MOR (mouvements oculaires rapides) et les rêves.

Les théories de la dominance de l'hémisphère droit comme celles de Galin (1974), Broughton (1995) et Joseph (1996) se basent essentiellement sur quelques cas sélectionnés de perte des rêves suite à une lésion de l'hémisphère droit et sur l'association souvent retrouvée dans la littérature entre le sommeil MOR et les rêves. Il est connu depuis Jouvet (1962) que le sommeil MOR est associé à une plus grande activation de l'hémisphère droit. Cependant, comme le démontre Solms (2004), l'association entre le sommeil MOR et les rêves n'est pas nécessairement causale. Le sommeil MOR peut exister sans que le patient ne rêve, suite à des lésions corticales qui épargnent le mésencéphale, de même que des rêves peuvent survenir en dehors de

la période de sommeil MOR. Le fait que le sommeil MOR soit fortement associé à l'activation de l'hémisphère droit ne prouve en rien que les rêves soient générés par cet hémisphère.

Une théorie de l'hémisphère gauche dans la génération des rêves (Greenberg & Farah, 1986) postule que l'hémisphère gauche est suffisant pour la génération des rêves. Dans cette étude, les auteurs ont analysé 9 cas de perte de rêves suite à une lésion cérébrale et ont trouvé que l'hémisphère dominant pour le langage était atteint dans chaque cas. Certains cas ont cependant été exclus de cet article sur la base de la langue (les articles en allemand n'ont pas été pris en compte) et d'autres ont été rejetés parce la perte des rêves n'était pas complète.

Enfin, un autre ensemble de théories prétend que les deux hémisphères participent à la génération des rêves. Kerr & Foulkes (1981), Antrobus (1987) et Doricchi & Violani (1992) et Ramachandran (1996) ont développé de telles théories, plus ou moins complexes. Par exemple, Kerr & Foulkes ont attribué des fonctions complémentaires à chaque hémisphère dans la génération du rêve. Selon eux, l'hémisphère droit se chargerait du contenu visuel alors que l'hémisphère gauche se chargerait du contenu narratif.

Le type d'études effectuées a une influence sur les résultats dans le cas de la spécialisation hémisphérique de la génération des rêves. Les études de groupe de

patients qui se sont penchées sur la question (Cathala & Laffont, 1981; Arena et al., 1984; Murri et al., 1984; Murri et al., 1985) n'ont pas trouvé d'effet de latéralisation dans la perte des rêves. Les études cas multiple, par contre ont trouvé une prédominance de lésions à l'hémisphère gauche dans le cas de la perte des rêves (Doricchi & Violani, 1992; Solms, 1997). Cette disparité entre les études de groupe et les études de cas multiple peut s'expliquer par plusieurs facteurs. Premièrement, les études de groupe sont habituellement limitées dans le temps à quelques jours ou au mieux à quelques semaines. Sur une plus longue période de temps, il est probable que quelques patients se seraient souvenu d'au moins un rêve. Il peut donc y avoir confusion entre perte de rêve et réduction de la fréquence des rêves. Ensuite, les patients étudiés dans les études de groupe sont souvent en phase aiguë et n'ont pas eu le temps de récupérer de certaines atteintes cérébrales, comme l'œdème, qui ont des effets plutôt diffus. Enfin, les études de groupe incluent des patients avec des degrés de déficits variables, pouvant ou non être considérés comme pathologiques (à deux écart-types de la moyenne). Contrairement aux études de cas, qui présentent habituellement des cas extrêmes ou sortant de l'ordinaire, les résultats de ces études de groupes sont rarement aussi marqués que ceux des études de cas. Un tel manque d'homogénéité peut venir brouiller les cartes et gommer des différences qui seraient peut-être apparues dans des cas extrêmes, comme ceux présentés dans les études de cas.

Dans la présente étude, les critères d'âge, de sexe, de dominance manuelle, de présence ou d'absence d'épilepsie, d'étiologie, de comorbidité psychiatrique et de présence ou non d'aphasie ont été notés lorsque disponibles. Tous ces critères peuvent avoir une influence sur la latéralisation hémisphérique et doivent être pris en compte dans une étude de cas multiples.

Dreams and unilateral brain lesions: a multiple lesion case analysis

Introduction

Charcot (1883) was the first to describe a patient who lost all capacity for visual imagery, awake as well as in his dreams, due to 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 have been blended in the literature to form the Charcot-Wilbrand syndrome. This syndrome has recently been challenged by Solms (1997) who, despite a certain overlap between the two cases, dissociates them in two distinct pathologies. The Charcot variant consists of cessation or reduction in visual dream imagery, while the Wilbrand variant consists of complete cessation of dreaming. Since the publication of these two cases, Doricchi & Violani (1992) reviewed 102 additional cases of dream reports following brain injury published in French, English, German or Italian while Solms (1997) reviewed 109. These cases are often incomplete however, and both authors analyzed more modest samples (Doricchi & Violani: 70 cases, Solms: 73 cases). These two extensive reviews are discussed later.

Hemispheric specialization for dreaming. There are three basic interpretations of hemispheric specialization for dreaming. Galin (1974) and Broughton (1995) state 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 non-logical and vivid visual nature of dreams, he proposed a right hemisphere localization of dream generation. Joseph (1988) further developed this proposal based on a non-systematic 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 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) clearly showed 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." 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 & 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, as 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 of publication (German).

A third proposal, positing a hemispheric interaction, states that both hemispheres have a role to play in dream generation and recall. Kerr & Foulkes (1981) believe, for example, that the right hemisphere modulates visual integration in dreams while the left modulates the narrative content. Antrobus (1987) developed a similar model of hemispheric specialisation of dream generation. Doricchi & Violani (1992) and Solms (1997) agree, based on extensive reviews of the literature, that the relationship between hemispheres and dreams is more complicated than 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 while 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 cost to maintain a coherent model of reality, confabulating and repressing conflictual information to do so, while the right hemisphere is an "anomaly detector" which 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 which could solicit complementary contributions from each hemisphere. Dreams with highly negative content could be generated in the right hemisphere while 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 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; Snyder, 1958; Huppertz, Franck, Korinthenberg & Schulze-Bonhage, 2002; Ide, Mizukami, Suzuki & Shiraishi, 2000; Lombroso, 2000; Montplaisir, Laverdière & Saint-Hilaire, 1981; Reami, Silva, Albuquerque & Campos, 1991; Rodin, Mulder, Faucett & Bickford, 1955). The epileptic focus was in the right hemisphere in 9 of the 13 cases with unpleasant dreams (NS) 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, Forti, Franetti, Donato, Giovanardi & Coccagna, 1975; Harvey & Barnes, 1996; Silvestri & Bromfield, 2004). Mendez and Doss (1992) report a case of right temporal lobe epilepsy with a normal MRI 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 by the patient.

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 8 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 schizophrenic patients having undergone prefrontal

lobotomy with 13 schizophrenics patients who did not on immediate dream recall following REM awakening. They found a notable lack of dream report in lobotomized patients when compared with the non-lobotomized group (X^2 test, $p < 0.001$). There is no indication, however, that a locus for dream generation or recall is involved is this study. It is a disconnection rather than a lesion problem. Cathala & Laffont (1981) compared patients with either a frontal lesion (7 cases) or a parietal lesion (9 cases) 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 as compared with their control group (X^2 test, $p < 0.01$). No difference was found between frontal patients and their control group.

Arena and colleagues (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 (X^2 test, $p < 0.01$) and left hemisphere lesion (X^2 test, $p < 0.05$) 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 was formed 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, $p < 0.02$) as well as more frequent in the group of posterior left lesions than anterior left lesions (Fisher, $p < 0.005$). There was no significant difference as a function of side of the lesion. Conscious of the potential flaws of morning dream recalls, Murri and colleagues (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, as 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, $p < 0.05$). Once again, there was no difference in dream recall as a function of hemisphere.

This lack of a laterality effect in group studies appears somewhat puzzling, especially in light of all the theoretical work on dream lateralization reviewed above. Some factors could account for this non-lateralization 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., oedema, 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 which 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 applied 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 one (Doricchi & Violani, 1992) reviewed 104 cases. The second one (Solms, 1997) reviewed 111. 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 non recallers presented with left hemisphere lesions while only six had unilateral right hemisphere lesions. That bias in favor of the left hemisphere is highly significant (binomial, $p = 0.004$). However, these data must be interpreted with caution since 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.

Dorrichi & 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 two 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 while only 8 a unilateral right

hemisphere lesion. This laterality effect reaches statistical significance (binomial, p = 0.05, 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 occipitotemporal region confined to the right hemisphere, only cases with bilateral or left-hemisphere lesions. In addition, a few cases of near complete right hemispherectomy have been studied in the sleep laboratory with multiple awakenings. Dreaming was intact (Mc Cormick et al, 1997). The total absence of right posterior (occipital) cases is striking and can hardly be dismissed as a bias in the literature. It seems more likely to indicate fragility in the left hemisphere concerning the dream process than a clinical bias toward the left hemisphere.

Limitations of previous multiple case review studies on post lesion dream cessation. Because Solms's and Dorricchi and colleagues' reviews did not describe individual cases, there are a multitude of issues which remain pending and which cast doubt on the laterality findings observed. Epilepsy was a common comorbidity and was not analyzed statistically. Some cases could have been suffering from irritative lesions.

Age and gender were not analyzed. Dream reduction was not absolute in all cases. Presence of aphasia was not treated quantitatively.

In the context of an inference test of a hemispheric specialisation 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 specialisation (Hugdahl, 2000). Presence/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 specialisation (Grabowska, Herman, Nowicka, Szatkowska & Szelag, 1994). Age is important because hemispheric specialisation is weaker in juveniles than adults (Montour-Proulx, Braun, Daigneault, Rouleau, Kuehn & Begin, 2004). In the context of any model of hemispheric specialisation, cases 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. As 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/she has ceased to dream per se (Moss, 1972). Other authors (Epstein & Simmons, 1983; Broughton, 1982; Dorrichi & Violani, 1992; Joseph, 1988) also suggested 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 & Greenberg (1985) have also reported that some patients with aphasia do dream, and they sometimes even regain language in their dreams Although aphasia seems to bring a bias to the study of lateralization of dream loss, no authors tried to measure this relation statistically.

Method

We selected cases in the literature in French, English, German, Spanish and Italian found in Medline and Psyclit databases without restriction of date and cross-referencing using two inclusion criteria: 1) cases with exclusively unilateral lesions, radiologically or surgically confirmed, 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 non-usable cases in light of the postulated model, 31 cases were retained for analysis. The cases are presented in Table 1. The localization of the lesion is taken "as is" from the articles cited. The precision in these localizations varies greatly, ranging from a vascular territory to a precise anatomic localization. The assessing of dream loss varies greatly from source to source, ranging from self-report for the older papers to laboratory awakening. Each case, however, was reported as a true dream loss and not simply an absence of dream, in the papers.

Results

Table 1. Cases with unilateral lesions entailing complete dream loss

Age/ sex/ 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 et al, 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	Thrombosis,	Broca's aphasia	Complete	?	Epstein &

	the left middle cerebral artery	EEG not mentioned, no seizures mentioned		loss of dreams		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/Bi	Left posterior	Stroke, EEG not merntioned, 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 et al, 1957
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, Levine & 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/Bi	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	Penetrating	Topographical	Complete	?	Humphrey

	parietal	injury (mortar), EEG not mentioned, no seizures mentioned	disorientation, right-left disorientation,	loss of dreams, recovery after five years		& Zangwill, 1951
42/M/?	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 et al, 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)
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	Wernicke's aphasia, long term verbal	Complete loss of	?	Corda, 1985

		seizures mentioned	memory deficits	dreams (previously poor recaller)		(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 et al, 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 et al, 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 et al., 2006

Of the 31 unilateral lesion cases presented in the table just above, 22 had a left hemisphere lesion and 9 had a right hemisphere lesion. The preponderance of left hemisphere lesions reached statistical significance (binomial: $p = 0.031$, two-tailed).

We believe left and right hemisphere lesions have equiprobable chances of occuring and being selected for single case reports (in the absence of selection for dreaming disturbance). For example, in their post-lesion IQ study of 635 previously published 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 purpose)

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

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 laterilazation of lesions causing dream cessation). Hemispheric specialization is also known to be weaker in juveniles than adults (Montour-Proulx, Braun, Daigneault, Rouleau, Kuehn & Begin, 2004). The correlation between age and side of lesion was not significant. No further 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 further analysis was required.

Handedness. Non-right handers usually show less structural asymmetries than right handers (Bear et al., 1986). They are also known to show less hemispheric specialisation for language (Isaacs, Barr, Nelson, Devinsky, 2006) . This variable could thus bring a caveat to our main result of laterality. In this research, there was no link between hand writing preference and lesion side.

Lesion locus. Several authors have studied cerebral activation associated with dreaming. They generally find 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-Vu 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, 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 and right hemisphere damage groups du to sampling artefact, casting doubt on the meaning of a 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 ($r = 0.472$, $p = 0.010$). In the small lesion group, there was almost an equal number of left side lesions ($N = 5$) and right side lesions ($N = 6$), while in the large lesion group, the effect of side of the lesion was highly significant ($N = 18$, $p = 0.001$). Small lesions could contribute noise to lateralization effect. Worse, the left lesion preponderance in the present group could be a simple artefact 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, Kohler, Swanson & Gur, 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). Unipolar depressives are

also known to dream less than non-depressive patients (Kramer et al, 1966), while 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 further analysis was required.

Date of publication and presence of an EEG. The poorer resolution of the CT scan used in the 70s' compared to MRI used nowadays could have brought more errors when localising 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 localisation of the lesion. Presence/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 ($r = 0.419$, $p < 0.019$). The relation between aphasia and lesion side exceeded the lesion side effect itself. In fact, among the 14 non aphasic 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 ($p = 0.002$). See table 2.

Table 2. Distribution of lesion side and lesion volume in aphasics and non aphasics.

Aphasia	Non aphasics (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 of this review of 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 a lesion of the right hemisphere. The current review replicates almost perfectly the results already obtained by Solms (1991) and Doricchi & 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 (Epstein & Simmons, 1983; Broughton, 1982; Dorrichi & Violani, 1992; Moss, 1972; Joseph, 1988). None of the authors studying post lesion dream cessation had 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 show that an important portion of the variance of the effect of left lateralization for dream loss is explained by the presence of aphasia as well as lesion size. The so-called cessation of dreaming may thus consist more of an inability to verbalize dream content and to use

linguistic abilities to improve recall of dreams. The effect of aphasia could be so strong that it might suffice to obliterate recall of any dream activity occuring at all.

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 artefact of lesion size and aphasia. Unilaterally lesioned non aphasic 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.

Discussion

L'analyse des 31 cas de perte complète des rêves retenus dans cette étude montre qu'une lésion dans l'hémisphère dominant pour le langage est plus susceptible de causer une perte des rêves qu'une lésion dans l'hémisphère non dominant, principalement si la lésion est postérieure. Les résultats répliquent presque parfaitement ceux obtenus par Doricchi & Violani (1992) et Solms (1997).

Un des problèmes souvent soulevés par différents auteurs dans l'étude de perte de rêves après lésion cérébrale est que les lésions de l'hémisphère gauche entraînent souvent une aphasie qui peut interférer avec la capacité de se souvenir ou de narrer les rêves (Epstein & Simmons, 1983; Broughton, 1982; Dorrichi & Violani, 1992; Moss, 1972; Joseph, 1988). Les résultats de la présente étude de cas multiples montrent que les inquiétudes de ces auteurs sont justifiées : une large portion de la variance de l'effet de latéralisation des lésions associées à une perte de rêve est expliquée par la présence d'aphasie, de même que par la grosseur de la lésion. Ce qui est rapporté dans la littérature comme des pertes de rêves pourrait donc souvent être dû à une incapacité à verbaliser le contenu des rêves ou à utiliser des outils linguistiques pour aider le rappel de ces rêves. Dans certains cas, la présence d'aphasie seule pourrait peut-être expliquer l'incapacité de rêver.

La présente étude réplique à la fois les études de cas multiples, avec un plus grand nombre de personnes présentant une lésion de l'hémisphère gauche présentant une

perte de rêves et les études de groupes, qui ne trouvent habituellement aucune latéralisation dans les lésions associées à la perte de rêves. Elle permet même d'expliquer, au moins partiellement, les différences observées entre ces deux types d'études. Les plaintes spontanées de perte de rêves sont plus fréquentes suite à de grosses lésions de l'hémisphère gauche qu'après des lésions de l'hémisphère droit. Toutefois, cette différence s'explique mieux par la grosseur de la lésion et par la présence d'aphasie que par la latéralisation de la lésion. Chez les patients ne présentant pas d'aphasie, la lésion unilatérale associée à la perte de rêves se retrouve indifféremment à gauche ou à droite.

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