The Laterality of Dreaming

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The cortical locus of dream generation could be lateralized to the right or left hemisphere, or be bilaterally represented with either equal or unequal contributions from each hemisphere. In this paper we review the neurological literature for cases of loss or alteration of dream report after brain damage. The distribution of lesion sites is used to test the various hypotheses concerning the laterality of dreaming. The hypothesis receiving best support is that dreaming is lateralized to the left hemisphere in individuals with typical neurologic organization. © 1986 Academic Press, Inc.

Several authors have posited a specialized role for the right cerebral hemisphere in the process of dreaming (Bakan, 1977; Broughton, 1975, 1982; Galin, 1974; Hoppe, 1977; Stone, 1977; VanValen, 1973). This claim is based upon the parallels between the nature of dream mentation and the cognitive specializations of the waking right hemisphere (see Ehrlichman & Barrett, 1983). Dreams typically consist of vivid visual images (Thompson, 1914; Weed & Hallam, 1896), and the study of hemispheric specialization has revealed a right hemisphere superiority for a variety of visual/spatial cognitive processes (Benton, 1979). In addition, the emotional

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and typically dysphoric content of dreams could reflect the putative right hemisphere advantage for the processing of affect (Heilman, Scholes, & Watson, 1975; Sackeim, Greenberg, & Weiman, 1982; Schwartz et al., 1975).

In contrast, Epstein (1979; Epstein & Simmons, 1983) has argued that dreaming is not exclusively a right hemisphere activity, based on his observations of aphasic patients who report a loss of dreaming. Foulkes (1978) maintains that the deep structure of dreams reveals syntactical rules that are analogous to a linguistic grammar. He cites Luria’s (1973) clinical observations to support his contention that the left hemisphere plays a decisive role in the construction of dream images.

Two recent studies of commissurotomized epileptics directly address the issue of the laterality of dreaming. If the split-brain patient reports dreams, then one may infer that the disconnected left hemisphere can dream. Hoppe (1977) conducted retrospective clinical interviews with 12 split-brain patients from the Sperry, Vogel, and Bogen series. Ten of these patients had complete commissurotomies. Patients were asked to recall dreams from both before and after their operations. Although 8 patients could not recall any postsurgical dreams, 9 patients could not recall any presurgical dreams. The postsurgical dreams that were recalled were remarkable for their brevity, simplicity, and mundane realism.

Greenwood, Wilson, and Gazzaniga (1977) examined the dream reports of three split-brain patients after awakenings from REM and NREM sleep. The two partial commissurotomy patients recalled a total of three dreams from three REM awakenings, and no dreams from two NREM awakenings. The one completely commissurotomized patient (who, unlike the two partial patients in this study, could not transfer visual information between the hemispheres while awake) nevertheless also recalled dreams, two dreams from four REM awakenings and none from four NREM awakenings. Moreover, two of the four REM reports contained explicit references to visual elements.

In the absence of baseline data collected prior to surgery for comparison, the dream reports of commissurotomy patients are difficult to interpret. However, both of these studies converge to suggest that the isolated left hemisphere retains the capability of experiencing a visual dream. This result is inconsistent with the hypothesis that dreaming is the exclusive province of the right hemisphere.

The majority of split-brain patients have early onset, severe neurologic compromise, years of frequent, poorly controlled seizuring, and heavy medication regimens. These factors suggest atypical patterns of neurological organization. Therefore, inferences drawn from this population regarding patterns of normal lateralization require converging support from other clinical populations. Furthermore, even if the neurological substrate of dream generation is not exclusively within the right hemisphere, many
competing hypotheses remain, all equally consistent with the finding that an isolated left hemisphere can dream.

In the present paper we review the neurological literature on loss or alteration of dreaming following brain damage, in an attempt to select among the possible sites of dream generation.

Consideration of two dimensions of lateralization—degree and direction of asymmetry of function—yields the following set of hypotheses.

_Hypothesis U._ Dreaming is unilaterally represented. There are at least three variants of this hypothesis.

_Hypothesis U:R._ Dreaming is unilaterally represented in the right hemisphere.

_Hypothesis U:L._ Dreaming is unilaterally represented in the left hemisphere.

_Hypothesis U:L/R._ Dreaming is unilaterally represented in the right hemisphere in some individuals and in the left in others.

_Hypothesis B._ Dreaming is bilaterally represented. Two variations are as follows.

_Hypothesis B:S._ Dreaming is bilaterally represented with symmetric contributions of the two cerebral hemispheres.

_Hypothesis B:A._ Dreaming is bilaterally represented with differential contributions of the two hemispheres.

Each of the above hypotheses suggests a prediction about the distribution of lesion sites in cases of loss or alteration of dream function.

If hypothesis U:R, then loss or alteration of dreaming should be associated with both right and bilateral lesions.

If hypothesis U:L, loss or alteration of dreaming should be associated with left or bilateral lesions.

If hypothesis U:L/R, loss or alteration of dreaming should be associated with right, left, and bilateral lesions.

If hypothesis B:S, loss of dreams should be associated only with bilateral damage.

If hypothesis B:A, loss of dreams should be associated with bilateral damage, and alteration in dreaming should be associated with either left, right, or bilateral lesions.

All available English language case reports documenting an alteration of dreaming following neurologic insult were collected and reviewed according to the following criteria:

(1) A loss or alteration of dream recall was self-reported by the patient either spontaneously or in response to direct inquiry.

(2) Loss or alteration of dreaming had to be a "focal" deficit and not
merely a consequence of a more generalized impairment in arousal, attention, orientation, or memory. For example, Weinstein's (1963) report on alteration in dream recall in the acute stage of recovery from head trauma was excluded. These patients were inattentive, confused, and tended to confabulate.

(3) Cases that were merely mentioned in the course of an article, rather than being the subject of an article, had to include sufficient clinical information to describe patterns of deficit, localize the lesion, and isolate the etiology. Three cases of loss of dreaming were excluded on these grounds: Brain's (1950) reference in a footnote to another physician's patient with a right parietal lesion (no information about etiology, accompanying deficits, sex or handedness), and Broughton's (1982) references to autobiographical accounts of loss of dreaming accompanying aphasia after stroke (one male, one female, no other information).

(4) The case report had to be drawn from an unselected, unbiased sample. Therefore, the observations of Jus et al. (1973) on the loss of dream recall in schizophrenics following prefrontal lobotomy were excluded. Epstein and Simmons (1983) screened aphasic patients for loss of dream recall and found eight such cases. These patients were excluded because they were drawn from a sample heavily biased towards left hemisphere damage.

In each case, the original author's judgments regarding the localization of damage and the nature of the associated deficits were reviewed by the present authors. In Boyle and Nielsen's (1954) case E.S., a discrepancy in localization arose, with the original authors describing right-sided damage and the present authors inferring bilateral damage from the presence of bilateral visual field defects. In a second case, Adler (1944) initially did not attempt localization. In a later paper (1950) she localized the focus of damage to the left parietal-occipital region based on clinical signs and repeat EEGs. The present authors concurred with the assessment of diffuse damage with parietal involvement, but judged that there was insufficient evidence to draw conclusions about laterality. Both cases were submitted to a neurologist who was blind to the hypotheses under consideration. In both cases he confirmed the present authors' judgments. There were no discrepancies concerning associated cognitive impairments.

The empirical investigations of dreaming reviewed here all use patients' self-reports as data for inferences about dream dysfunction. In general, reliance on some form of self-report is inevitable in the study of any covert mental activity. This holds for the study of attitudes, moods, and personality constructs as well as for dream generation. In spite of the inherent limitations of self-report methods, they provide the only means available for investigating the covert activity of dreaming. A brief summary
of the available cases of loss and alteration of dream recall is presented below.

REVIEW OF CASES

Adler (1944, 1950) reports the case of H.C., a 22-year-old woman who suffered a presumed anoxic event in the Coconut Grove fire. Upon admission, the patient was totally blind, and remained in an agitated confusional state for weeks, characterized by a press of fluent but meaningless speech and a marked tendency toward perseveration. Her vision cleared rapidly, but she was left with alexia without agraphia, visual simultanagnosia, and constructional apraxia. One month after injury she reported having had three dreams which were most remarkable because they lacked any visual elements. Her reading improved somewhat over 5 months, as did her ability to draw from memory. Deficits in copying, object recognition, calculation, and finger naming remained.

Basso, Bisiach, and Luzzatti (1980) described the case of M.G., a 63-year-old dextral male trolley driver who had sudden onset of headache, giddiness, and inability to read. Neurological exam revealed a slight right hemiparesis, dysphasia, and right homonymous hemianopsia. One month later the motor deficit improved, but there was severe loss of cortical sensation in the right upper extremity. The right visual field defect was unchanged. Cognitive testing revealed anomia for objects without agnosia, and dyslexia with dysgraphia. CT scan of the head revealed a reduced density in the lower mesial region of the left occipital lobe and mesial left temporal lobe, with signs of left cerebellar involvement as well. Seventeen months later the clinical picture remained unchanged. The patient was unable to describe familiar visual scenes from memory and could not, for example, describe the route his tram had traveled. He reported that he lost the ability to conjure up images in the visual realm, as well as in the auditory, gustatory, and olfactory modalities. He complained of losing the rich hypanogic imagery that he enjoyed premorbidly, as well as a total loss of dreaming.

Boyle and Nielsen (1954) and Nielsen (1955) describe a 31-year-old male mathematician (E.S.) with acute onset of severe headache. Pneumoencephalogram revealed a tumor of the third ventricle. At surgery a colloid cyst was removed and a drainage tube was passed through the right occipital lobe. Immediately postsurgery, there was a left hemiparesis (arm worse than leg), Anton's syndrome, hyperactive deep tendon reflexes, and urinary incontinence. Three months later he was disoriented to time and place. Vision in the right inferior quadrant returned, but he was agnosic for objects with greatest impairment in recognizing inanimate objects. Furthermore, his ability to describe visual information from memory was impaired. Reading, writing, and calculations were spared.
A mild hemiparesis remained; the patient reported that he had suddenly ceased to dream following the operation.

The second case (H.S.) presented with gradual onset of headache and signs of increased intracranial pressure. Examination revealed a right homonymous hemianopsia and "pathological reflexes." Following pneumoencephalogram a left occipital lobectomy was carried out. Histology revealed an astrocytoma. Seven months after surgery the patient demonstrated alexia without agraphia, finger agnosia, right-left confusion, and "disorientation in space." Map reading was impaired. The patient reported a complete cessation of dreaming since the surgery.

Boyle and Nielsen (1954) briefly mention two additional cases of unknown age, sex, and handedness, both with tumors of the left occipital lobe. The first presented with a right hemianopsia. The second case revealed a right superior quadrantopsia. Both cases report total cessation of dreaming in addition to loss of waking visual imagery.

Their fifth case was a young female who received a skull fracture from a blow to the occiput. Total blindness ensued. Surgical exploration revealed bilateral occipital damage, and a double dural tear on each side of the midline. Her vision improved somewhat over time, but her capacity to dream was lost.

Brain (1954) describes a 36-year-old right-handed male who sustained a depressed skull fracture in the mid-frontal region in a motor vehicle accident. He was comatose for 8 days. Five years later he complained of losing his "picture memory." He was unable to generate visual images of familiar objects or people, yet his visual recognition was unaffected, as were reading and writing. He continued to dream but remarked that his dreams were no longer visual: "When I dream, I seem to know what is happening, but I don't seem then to see a picture." Ten years later his complaints were unchanged. He had slight aphasia, and an EEG revealed theta slowing in the frontal regions, more pronounced on the left side. Cognitive testing revealed no overall intellectual impairment and mild spelling impairments. There is no further information on his dream recall.

Epstein (1979) describes a 56-year-old female with mixed handedness who complained of sudden right-sided weakness and loss of sensation. Neurological exam revealed aphasia, alexia, right homonymous hemianopsia, right hemiparesis, and a right hemisensory deficit. Two months later, the aphasia and hemiparesis had cleared. The right homonymous hemianopsia, dyslexia, and increased deep tendon reflexes on the right remained. The right extremities were dysmetric. The presumed etiology was a cerebrovascular accident. Upon inquiry, she reported the total cessation of dreaming. Prior to her stroke she had been a prolific dreamer, keeping a written dream diary and often discussing her dreams with a family member in the morning. Three months later color recognition and
Facial recognition were impaired. There was no agnosia for objects, and the right homonymous hemianopsia remained. She then reported having had one dream in the interim. In the following 3 months, dreaming was totally absent. Nineteen months postonset she reported a sudden return of dreaming.

Humphrey and Zangwill (1951), in their classic paper on cessation of dream recall, described three patients who spontaneously reported alterations in the dream experience after sustaining penetrating head wounds. In case 1, a 26-year-old dextral male was wounded in the right posterior parietal region. Shrapnel was removed at surgery. Immediately postsurgery a left homonymous hemianopsia, a left hemianesthesia, and mild pyramidal tract findings were observed. One month later, the left homonymous hemianopsia remained and "a fairly marked topographical loss," and "defects in visual recognition" were observed. Six years later the visual defect remained. The patient reported that he could summon up visual images with difficulty and then they "were dim." He reported a dramatic reduction in, but not cessation of, dreaming.

In case 2, a 21-year-old dextral male received a parasagittal posterior parietal mortar wound, involving the parietal–occipital regions bilaterally, with greater damage on the left. After a confusional state cleared, neurologic exam revealed a left homonymous hemianopsia, aphasia, and reduced joint position sense in the right extremities, with mild dyspraxia and visual memory deficits as well. The patient reported an impairment in waking visual imagery, and a total loss of dreaming. On follow-up 6 years later, the clinical picture was unchanged except for improvement in language function.

In case 3, a 32-year-old male sinestral received a right posterior parietal missile wound. One week after injury the neurological findings were a left inferior quadrantopsia, a dense left hemiplegia and loss of cortical sensation, and moderately severe aphasia. Three weeks later, sensory/motor deficits improved, but there was a "topographic loss," dressing dyspraxia, right–left confusion, and a visual construction deficit. Five months later there was residual left-sided sensory/motor impairment, slowness of speech, and some topographical loss. The visual field defect had resolved. Five years later, the patient reported the sudden return of dreaming (including visual dreams) which had been absent since his injury.

Lyman, Kwan, and Chao (1938) report a case of a 42-year-old bilingual Chinese male who presented with a gradual onset of headache, diplopia, dyslexia with dysgraphia, and dyscalculia. The patient was unable to draw common objects from memory but could copy them perfectly. In addition, he had difficulty describing local topography from memory. Physical exam revealed a right homonymous hemianopsia, positive Romberg sign, and diminished deep tendon reflexes in the right knee and
ankle. Surgical resection of the superior and inferior parietal lobules revealed a large fibroblastoma extending from the occipital pole to the border of the postcentral gyrus. Most of the tumor was excised, along with portions of the angular and supramarginal gyri. Recovery was complicated by an infection. Seven months postsurgery, reading and writing were remarkably improved in spite of a dense right hemianopsia. Calculations remained severely impaired. Drawing from memory and topographic memory improved dramatically. The patient was mildly euphoric and optimistic. He reported "he had been having no dreams for a long time."

Wilbrand (translated by Critchley, 1953) published a case of a 63-year-old multilingual woman who experienced a sudden loss of consciousness followed by an acute confusional state. The presumed etiology was a CVA. Examination revealed a bilateral field defect with greater impairment on the left. Reading was spared, but she was unable to visualize familiar objects, people, or places. Four years later she reported "hardly dreaming at all." Her ability to summon up visual images returned, although well-known objects and sights (including her own reflection) were not recognized or else were experienced as unfamiliar. Autopsy revealed bilateral ischemic areas in the "posterior lobes."

RESULTS

Table 1 summarizes the clinical findings. Despite the paucity of cases, diversity of etiologies, and the varying quality and quantity of clinical data, a consistent pattern emerges.

In nine cases, patients reported a total loss of dream recall. In each case, the lesion was localized to the posterior cerebral regions. Damage was confined to the left hemisphere in five cases, the right in one case, and was bilaterally distributed in three cases. Although there are too few cases for an adequate statistical test, this pattern suggests a clear trend toward greater left hemisphere involvement. Moreover, the one right-sided case (Humphrey & Zangwill, case 3) was left-handed with dysphasia, implying reversed lateralization, and this case was the one case in which loss of dreaming was temporary. Therefore, all cases presumably involve the "dominant" hemisphere and none involve only the "nondominant" hemisphere. These results provide support for the hypothesis that dreaming is unilaterally represented in the dominant hemisphere—the left hemisphere in individuals with typical cerebral organization.

Of these nine cases of loss of dreaming, waking imagery could be inferred to be absent or greatly impaired in seven; the remaining two cases (Boyle & Nielsen, 1954, case H.S. and their unnamed fifth case) contained insufficient information to assess waking imagery. Farah (1984) analyzed cases of loss of waking image generation (including some of
**TABLE 1**

**CLINICAL FINDINGS IN CASES OF LOSS AND ALTERATION OF DREAMING**

<table>
<thead>
<tr>
<th>Author</th>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Hand</th>
<th>Etiology</th>
<th>Neurologic signs/SX</th>
<th>COG signs/SX</th>
<th>Waking Imagery</th>
<th>Dreaming</th>
<th>Site</th>
<th>Outcome (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Loss of dreaming</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basso, Bisiach, and Luzzatti</td>
<td>2</td>
<td>21</td>
<td>M</td>
<td>R</td>
<td>Cerebrovascular accident</td>
<td>RHP/RHH/SRUE</td>
<td></td>
<td>Absent</td>
<td>Absent</td>
<td>L temp-occ</td>
<td>&gt;1.5</td>
</tr>
<tr>
<td>Humphrey and Zangwill</td>
<td>3</td>
<td>51</td>
<td>M</td>
<td>L</td>
<td>Penetrating head wound</td>
<td>LHH</td>
<td></td>
<td>Impaired</td>
<td>Absent</td>
<td>B1 par-occ</td>
<td>&gt;6</td>
</tr>
<tr>
<td>Lyman, Kwan, and Chao Neilsen/Boyle and Neilsen</td>
<td>1</td>
<td>42</td>
<td>M</td>
<td>?</td>
<td>Neoplasm/surgical trauma</td>
<td>RHH/↓DTRs(R)</td>
<td>VA</td>
<td>Absent</td>
<td>Absent</td>
<td>BI temp-par-occ</td>
<td>&gt;1</td>
</tr>
<tr>
<td>Neilsen/Boyle and Neilsen</td>
<td>3</td>
<td>?</td>
<td>M</td>
<td>?</td>
<td>Neoplasm/lobectomy</td>
<td>RHH</td>
<td>?</td>
<td>Absent</td>
<td>Absent</td>
<td>L par-occ</td>
<td>&gt;0.6</td>
</tr>
<tr>
<td>Neilsen/Boyle and Neilsen</td>
<td>4</td>
<td>?</td>
<td>M</td>
<td>?</td>
<td>Neoplasm</td>
<td>RSQ</td>
<td>?</td>
<td>Absent</td>
<td>Absent</td>
<td>L occ</td>
<td>&gt;2.5</td>
</tr>
<tr>
<td>Neilsen/Boyle and Neilsen</td>
<td>5</td>
<td>F</td>
<td>?</td>
<td>?</td>
<td>Trauma/skull fracture</td>
<td>Blind</td>
<td>?</td>
<td>Absent</td>
<td>Absent</td>
<td>BI occ</td>
<td>&gt;5</td>
</tr>
<tr>
<td><strong>Alteration of dreaming</strong></td>
<td></td>
<td></td>
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<tr>
<td>Adler</td>
<td>2</td>
<td>22</td>
<td>F</td>
<td>?</td>
<td>Anoxic event</td>
<td>?</td>
<td>AL/SA/CA/DC/RL</td>
<td>Impaired</td>
<td>Nonvisual Dreaming spared</td>
<td>Diffuse</td>
<td>&gt;5</td>
</tr>
<tr>
<td>Brain</td>
<td>1</td>
<td>36</td>
<td>M</td>
<td>R</td>
<td>Trauma/skull fracture</td>
<td>WNL</td>
<td>DP</td>
<td>Absent</td>
<td>Nonvisual Dreaming spared</td>
<td>B1 frontal</td>
<td>&gt;5</td>
</tr>
<tr>
<td>Epstein</td>
<td>1</td>
<td>56</td>
<td>F</td>
<td>BI</td>
<td>Cerebrovascular accident</td>
<td>RHH/RHS/↑DTRs(R)</td>
<td>DL/CN/PA</td>
<td>Impaired</td>
<td>Reduced</td>
<td>L posterior</td>
<td>1.5 remit</td>
</tr>
<tr>
<td>Humphrey and Zangwill</td>
<td>1</td>
<td>26</td>
<td>M</td>
<td>R</td>
<td>Penetrating head wound</td>
<td>LHH</td>
<td>VM/TG</td>
<td>Impaired</td>
<td>Reduced</td>
<td>R par-occ</td>
<td>&gt;6</td>
</tr>
<tr>
<td>Wilbrand (Critchley)</td>
<td>1</td>
<td>63</td>
<td>F</td>
<td>?</td>
<td>Cerebrovascular accident</td>
<td>BVF</td>
<td>?</td>
<td>Absent</td>
<td>Reduced</td>
<td>BI posterior</td>
<td>?</td>
</tr>
</tbody>
</table>

* For each case, the anatomical site of lesion and presumed etiology are listed, along with the patient's age, sex, and handedness, where known. In addition, neurological signs and symptoms, accompanying cognitive deficits, and outcome are shown.

* Abbreviations. BVF, bilateral visual field defect; LHH, left homonymous hemianopsia; LIQ, left inferior quadrantopsia; LHP, left hemiparesis; LHS, left hemisensory deficits; RHH, right homonymous hemianopsia; RSQ, right superior quadrantopsia; RHP, right hemiparesis; RHS, right hemisensory deficits; SRUE, sensory loss of right upper extremity; WNL, within normal limits; AL, agraphia; AP, agnosia; CA, constructional apraxia; CN, color agnosia; DC, dyscalculia; DD, dressing dyspraxia; DL, dyslexia; DP, dysphasia; DX, dyspraxia; FA, finger agnosia; PA, prosopagnosia; RL, right/left confusion; SA, simultanagnosia; TG, topographic disorientation; VM, visual memory deficits; SX, symptoms; PAR, parietal; OCC, occipital; TEMP, temporal.
the cases reported here), and found a left posterior focus of pathology. Further support for localizing imagery generation to the left hemisphere comes from Farah, Gazzaniga, Holtzman and Kosslyn's (1985) study of a commissurotomy patient and Farah's (in press) study of normals using tachistoscopic techniques. These findings suggest that the formation of waking and dream images may share the same neuroanatomic substrate.

Three patients reported markedly reduced dream recall (Humphrey and Zangwill's 1951 first case, Wilbrand's 1953 case, and Epstein's 1979 case). These patients spontaneously comment on a quantitative alteration, but do not mention qualitative changes in their experience of dreaming. Again, there is a posterior locus of pathology in all cases. However, unlike the cases of total loss of dream recall, no pattern of lateralization emerges—with one case each of left, right, and bilateral damage. These findings are consistent with the unilateral left/right and the bilateral symmetrical hypotheses. The distribution of lesion sites is again inconsistent with the unilateral right hypothesis. However, the small number of cases involved precludes drawing strong inferences.

A third pattern of deficits involves the qualitative alteration in dream function, with the absence of the visual dream and sparing of nonvisual dreaming (Adler's 1944 and 1950 case and Brain's 1954 case). Localization of damage was most problematic in these two patients. Adler's case suffered an anoxic event with no focal neurologic signs, and no pathophysiological confirmation. In a subsequent paper (Adler, 1950) the author localized the damage to the parietal and occipital lobes of both hemispheres, with left damage greater than right on the basis of repeated EEGs. The independent neurologist whom we consulted about this case inferred diffuse damage with probable parietal and occipital involvement bilaterally, but judged the published data insufficient to conclude greater left involvement. Brain's case suffered a frontal injury with negative neurological exam and no pathological data. The original author raised the possibility of a contra coup lesion in the parastriate cortex. However, again the independent neurologist was unwilling to localize the damage on the basis of the published data. This finding of an alteration but not loss of dreaming with bilateral lesions does not rule out any of the three unilateral and two bilateral hypotheses.

**DISCUSSION**

Taken together, the results of an analysis of cases of loss and alteration of dream recall implicates a crucial role for the left hemisphere in the generation of dreams. The distribution of lesion sites suggests that the left hemisphere plays perhaps an exclusive role in generating dreams in right-handed subjects with typical neurological organization.

The finding that loss of dreaming implies the presence of dominant hemispheric damage is evidence that this hemisphere contains the neural substrate critical for dreaming. This does not imply that the nondominant
hemisphere does not normally contribute to dream function. An analogy with the neuroanatomy of language may help to clarify this distinction. Aphasia is clearly a localizing sign for left hemisphere damage, and this constitutes one of our major sources of evidence for the claim that language is subserved by the left hemisphere. However, recent data indicate that the right hemisphere also contributes to linguistic performance, for example, in the regulation of speech prosody (Heilman et al., 1975; Ross & Mesulam, 1979) and in the comprehension and use of connotation (Gardner et al., 1983). Nevertheless, the discovery of the right hemisphere’s contribution to language does not contradict the proposition that the critical structures for language are in the left hemisphere. Similarly, future research may well reveal important aspects of dreaming controlled by the right hemisphere.

More cases of alteration of dream recall following focal brain damage are required for an adequate test of the bilateral/asymmetrical hypothesis. Relevant to this issue is the recent finding of Murri, Arena, Siciliano, Mazzolta, and Muratorio (1984) that the majority of patients in the acute stage of either unilateral right or left posterior cerebral disease showed an absence of dream recall over a period of ten consecutive nights. However, the low criterion for loss of dream recall (only ten nights without recalling a dream), the high incidence of this symptom, and the acute stage of illness at which patients were tested, all suggest that the phenomenon described by Murri et al. is distinct from enduring loss of dream recall. An extension of this type of study is needed, using pre- and postmorbid dream reports, analyzed for qualitative as well as quantitative changes in dream mentation after unilateral left and unilateral right lesions. In addition, a longer sample of dream recall is required to distinguish among transient loss, quantitative reduction (which could be mistaken for loss of dreaming in a short sample), and permanent loss of dreaming.

The high probability of left posterior damage given a report of loss of dream recall leads to the question: what is the probability of losing dream recall given a left posterior lesion? The small number of case reports in the literature might lead one to conclude that this reverse conditional probability is indeed low. However, we believe that there are four distinct reasons why the number of reported cases grossly underestimates the true prevalence of neurologically induced loss of dreaming.

First, the tendency to forget, ignore, and trivialize dreams is a ubiquitous phenomenon. Freud (1900) labeled this tendency “displacement” and argued that it served a defensive function in the psychic economy. This proclivity would affect neurologists as well as patients. In fact, standard references on the mental status exam and history taking in neurology make no mention of dreaming whatsoever (e.g., De Jong, 1967; Denny-Brown, 1957; Strub & Black, 1977). Second, because other neurological
deficits, such as hemiparesis and aphasia, are undoubtedly more disturbing and dysfunctional to the patient, there is a rational and practical basis for the neglect of dream function in neurology. Third, left hemisphere lesions that impair speech production and/or language comprehension will affect the patient's ability to report whether or not his/her dreaming has been affected. Fourth, right-sided parietal lesions which cause denial or minimization of illness (anosognosia) will affect the veracity of the patient's self report on dreaming.

Epstein and Simmons (1983) asked an unspecified number of aphasic patients attending a speech therapy program about their pre- and postmorbid dream function. Eight patients reported a loss of dream recall subsequent to their neurological events. (These patients were excluded from the above analysis since they clearly violated the criterion of unselected sampling—all had left hemisphere lesions.) This result suggests that the prevalence of loss of dream recall is greatly underestimated by the few available case reports. However, we recognize that the four points specified above pose formidable obstacles to both a determination of the true prevalence of the disorder, and establishment of its clinico-anatomic correlations. Nevertheless, Epstein and Simmons' data clearly support the conclusion derived from the unselected cases that the left hemisphere plays a crucial and perhaps exclusive role in dream formation in most individuals.

Dreaming is a complex, nonunitary process. Loss of dream recall can presumably result from disruptions at various points along the process of dream construction: interruptions in the waking experiences that provide the raw material for dreams, in the pacemakers that regulate the sleep-wake cycle, in the determinants of sleep architecture, in the phasic events of REM, in the arousal mechanism during the transition from sleep to wake, in the consolidation process upon awakening, and finally, in the elaboration of a verbal dream report. It is likely that different lesion sites will disrupt these processes in distinctive ways. For example, Michel and Sieroff (1981) have shown that left posterior lesions can disrupt dream recall while sparing the REM-NREM cycle. One can predict that a taxonomy of dream deficits, analogous to the present classification of the aphasias, will eventually emerge. A complete account of the neurological basis of dreaming will require that data on the role of cortical contributions be integrated with the large body of knowledge documenting diencephalic control of circadian pacemakers (Moore-Ede, Czeisler, & Richardson, 1983) and brainstem regulation of the REM-NREM cycle (Hobson, McCarley, & Wyzinski, 1975).

1 Schafeld et al. (1985) recently attempted to circumvent this difficulty by eliciting drawn dream reports. They found that this output mode was effective for both aphasic and nonaphasic stroke patients.
In conclusion, a review of reports of loss and alteration of dreaming following brain damage suggests that a region of the posterior dominant hemisphere is critical for dreaming. This finding is consistent with the results of split-brain studies showing the left hemisphere to be capable of generating dreams and disconfirms the prevailing notion that dreaming is an exclusively right hemisphere function. Further, our analysis indicates that if a lesion prevents the dominant hemisphere from generating dreams, the nondominant hemisphere is incapable of doing so.

Our initial conclusions raise several questions for future research to address: the precise localization of the critical areas for dream generation; the functional associations and dissociations between dreaming and other cognitive and personality functions after brain damage; the correlation of self-report of loss of dreaming with objective laboratory measures (e.g., EEG, and REM–NREM-correlated awakenings); the identification of neuropsychological markers for individual variations in the hemispheric control of dreaming; and the contribution of the nondominant hemisphere to dream function.

Note added in proof. Two additional case studies involving loss of dreaming have recently been reported (Farah, Levine, and Calvanio, in press; Pena-Casanova, Roig-Rovira, Bermudez and Tolosa-Sarro, 1985.) In both cases the patients suffered left posterior cerebral infarcts.

REFERENCES


