Handbook of Clinical Neurology, Vol. 98 (3rd series)
Sleep Disorders, Part 1
P. Montagna and S. Chokroverty, Editors
© 2011 Elsevier B.V. All rights reserved

Chapter 34

Neurobiology and the neurological basis of dreaming

MARK SOLMS*
Department of Psychology, University of Cape Town, Rondebosch, South Africa

BACKGROUND

Understanding the brain mechanisms of dreaming has lagged behind that of other mental functions. There are two main reasons for this. First, unlike most phenomena that were the focus of 19th- and 20th-century behavioral neuroscience, dreaming is almost entirely subjective. The observable data are retrospective, single-witness verbal descriptions, only indirectly related to dreaming itself. This poses special methodological problems.

The second reason for the undeveloped state of this field is closely related to the first. Researchers seeking an objective marker of dreaming eagerly alighted on a physiological state that correlates closely with it – rapid eye movement (REM) sleep (Aserinsky and Kleitman, 1953, 1955; Dement and Kleitman, 1957a, b) – which was then conflated with dreaming itself. This resulted in the development of neuropsychological models of dreaming which were in fact models of REM sleep (Hobson and McCarley, 1977). This conflation was confounded by the fact that the models were empirically grounded in animal studies (where dream reports are precluded) rather than human lesion studies of the kind that informed neuropsychological models of most other mental functions. When the conventional human lesion studies were eventually performed, it became apparent that dreaming and REM sleep were in fact doubly dissociable states (Solms, 2000).

A traditional neuropsychology, grounded in the systematic application of clinicoanatomical correlation, which was widely applied to other mental functions since the mid 19th century, is little more than 20 years old in the case of dreaming. Incidental reports of changes in dreaming associated with focal cerebral damage did nevertheless accumulate over a long period, albeit without any systematic attempt to synthesize the scattered observations into a coherent picture. The available evidence was not comprehensively reviewed before the 1990s (Solms, 1991, 1997; Doricchi and Violani, 1992). Clinicoanatomical group comparisons were first published a decade earlier (Cathala et al., 1983; Murri et al., 1984, 1985). The clinicoanatomical studies have since been complemented by a slew of functional brain imaging studies, with strongly convergent findings (Pace-Schott and Hobson, 1998).

CLINICOANATOMICAL FINDINGS

Charcot–Wilbrand syndrome

The concept of Charcot–Wilbrand syndrome, based on two case reports by Charcot (1883, 1889) and Wilbrand (1887, 1892), was first articulated by Pötzl (1928). He defined the syndrome as "mind-blindness with disturbance of optic imagination" (p. 306). Nielsen (1946) defined it as "visual agnosia plus loss of the ability to revisualize" (p. 74). Critchley's (1953) widely cited definition was:

> *a patient loses the power to conjure up visual images or memories, and furthermore, ceases to dream during his sleeping hours (p. 311).*

Critchley described proposagnosia and topographical agnosia or amnesia as associated features. The localization of the lesion producing this syndrome was never precisely defined, but the occipital cortex was implicated by most early authors (especially area 19), usually bilaterally. The Charcot–Wilbrand syndrome remained in late 20th-century nosographical usage, although the condition was (until recently) considered rare. A modern definition of the syndrome reads:

> *the association of loss of the ability to conjure up visual images or memories and the loss of*

*Correspondence to: Professor Mark Solms, Department of Psychology, University of Cape Town, Private Bag, Rondebosch 7700, South Africa. Tel: +2721 6503437, E-mail: mark.solms@uct.ac.za

> *dreaming ... [indicating] a lesion in an acute phase affecting the posterior regions (Murri et al., 1984, p. 185).*

Deficient revisualization (called "irreminiscence" in the nomenclature of Nielsen (1946)) was the fundamental symptom in almost all definitions of the syndrome. Cessation of dreaming ("or at least, an alteration in the vivid visual component of the dreaming state"; Critchley, 1953, p. 311) was seen as a secondary consequence of the visual imagery deficit. The associated visual agnosias, too, were originally considered to be secondary consequences of defective revisualization, since visual agnosia was classically understood as a loss of "visual memory images" (Munk, 1878; Lissauer, 1890).

Subsequent advances in the agnosia concept, and a misreading of the original case reports, has resulted in considerable nosological confusion regarding this syndrome (Solms et al., 1996). It is widely assumed that Wilbrand's case could not *visualize* familiar places (Nielsen, 1946; Critchley, 1953; Gloning and Sternbach, 1953; Farah et al., 1988). However, the original report stated only that she was unable to *recognize* those places. This symptom (which we would today call topographical agnosia) was conceptualized, in accordance with classical theory, as a disorder of "topographical *memory*" (Wilbrand, 1887, p. 52, emphasis added). This conceptualization was then misconstrued by secondary authors as a disorder of topographical revisualization. The original report reveals that Wilbrand's case lacked the cardinal feature of the so-called Charcot–Wilbrand syndrome. As the patient herself clearly stated:

> *With my eyes shut I see the old Hamburg in front of me again (Wilbrand, 1887).*

Charcot's case was quite different. He described a striking absence of visual mental imagery. The Charcot–Wilbrand syndrome is therefore misnamed.

It is also misconceived. Charcot's patient ceased to dream in visual images, but he continued to dream in words. Wilbrand's patient on the other hand dreamed "almost not at all any more" (Wilbrand, 1887, p. 54). The original report is ambiguous as to whether Wilbrand's patient merely dreamed infrequently or lost the capacity to dream completely (and then gradually recovered it); but in either case, there is no question of an isolated loss of visual dream imagery, which is what Charcot's patient unequivocally described. The Charcot–Wilbrand syndrome therefore appears to be two different (but related) syndromes, one characterized by loss of visual dream imagery and the other by global cessation or suppression of dreaming. This distinction is supported by a review of the world literature (Solms, 1997).

Charcot's variant: isolated loss of visual dream imagery

At least 10 case reports of isolated loss of visual dream imagery have been published, together with five further reports of patients who experienced submodal deficits of specific aspects of visual dream imagery (e.g., faces, color, movement). These cases are summarized in Table 34.1. Defective revisualization (irreminiscence) is a constant feature in the cases, although it is typically restricted to the affected aspect of vision in cases where the loss of visual dream imagery is submodal. This strongly suggests a common underlying image generation deficit causing the same disorder in both waking and dreaming cognition. Various forms of visual agnosia are commonly associated features, but agnosia is definitely absent in some cases and therefore cannot be considered integral to the syndrome. The lesion responsible for isolated loss of visual dream imagery usually affects the occipital lobe, frequently bilaterally, but precise localizing information is lacking in most reports.

Most published reports of changes in visual dream imagery derive from retrospective accounts in clinical settings. However, the reports have been confirmed upon REM awakening in at least 3 cases (Efron, 1968; Brown, 1972; Kerr et al., 1978).

Negative findings

Interestingly, modality-specific deficits of dream imagery outside the higher visual sphere cannot be demonstrated. Cortically blind and hemianopic patients report full visual fields in their dreams. Hemiplegic patients experience normal somatomotor and somatosensory function in their dream imagery. Nonfluent aphasics speak normally in their dreams (Solms, 1997).

Of related interest, perhaps, is the fact that the dreams of patients with substantial impairments of executive function due to dorsolateral prefrontal lesions are indistinguishable from the dreams of controls (Badenhorst and Solms, unpublished, 2006). These findings point to a differentiated network of forebrain structures involved in dream cognition – a conclusion to which we shall return below.

Wilbrand's variant: global loss or suppression of dreaming

At least 106 cases of global loss or suppression of dreaming have been reported, excluding leukotomy cases, which will be discussed separately. (This also excludes a large number of patients in group studies, for which individual case data were lacking, and "not dreaming" was defined in variable ways: Cathala et al., 1983; Murri et al., 1984, 1985.)

Table 34.1

Cases of cessation or restriction of visual dream imagery

Source	Case	Lesion	Irreminiscence	Dreams
Charcot (Bernard) (1883)	Monsieur X	No information ? Bilateral medial occip. temp. (thrombosis)	+	Cessation of visual imagery
Grünstein (1924)	Patient N., 23y F	L. lat occip.* (thrombosis)	+	Cessation of visual imagery
Adler (1944, 1950); Sparr et al. (1991)	H.C., 22y F	Bilat. Occip. par.* (CO poisoning)	+ −	Cessation of visual imagery, with subsequent global cessation of dreaming
Brain (1950, 1954)	Case 1, 36y M	Front. occip.* (trauma)	+	Cessation of visual imagery
Gloning and Sternbach (1953)	W. Josef, 53y M	L.† thalam* (hemorrhage)	−	Cessation of visual imagery
Macrae and Trolle (1956)	32y M	Bilat. par.* (trauma)	+	Cessation of visual imagery
Tzavaras (1967)	Monsieur P. Maurice, 54y	L. occip. temp., R. temp.* (hemorrhage)	+ for faces	Cessation of facial imagery
Efron (1968); Benson and Greenberg (1969); Brown (1972)	25y M (Brown's case 13)	Bilat. occip. par.* (CO poisoning)	+	Nonvisual nightmares for 1 week; residual cessation of visual imagery or global cessation of dreaming (descriptions ambiguous)[¶]
Kerr et al. (1978)	21y F	?R. par.* (not localizable) (Turner's syndrome)	+	Absence of visual imagery
Botez et al. (1985)	38y M	?R.† hemisphere, ?Corp. callosum (?dysgenesis)§	+	Reduction or absence of facial imagery (description ambiguous) and absence of hypnagogic imagery
Sacks (1985)	Dr. P., M	"Visual parts of his brain" (tumor or atrophy)	?	Cessation of visual imagery
Sacks and Wasserman (1987)	Jonathan I., 65y M	Bilat. med. occip. temp. (trauma)	+ for color	Cessation of color imagery and reduction of tonal gradation
Sacks (1991)	No information	"Diffuse damage to the occipital cortex"* (Alzheimer's disease)	(No information)[‖]	Cessation of visual imagery
Solms (1997)	Patient 201, 26y F	R. par. (AVM)‡§	+ −	Cessation of visual imagery
	Patient 208, 31y M	R. med. occip. temp. (astrocytoma)‡§	+ for faces and spatial relationships	Cessation of kinetic imagery

y, years; F, female; M, male; CO, carbon monoxide; AVM, arteriovenous malformation.
*Clinical localization or diagnosis.
†Left-handed or ambidextrous patient.
‡Localization or diagnosis based on intraoperative observation.
§Localization or diagnosis based on in vivo imaging techniques.
[‖]Unless otherwise indicated, it is assumed that the authors comprehensively investigated and reported the neuropsychological status of their cases.
[¶]Benson and Greenberg (1969, p.85) described this patient as a case of global cessation of dreaming and reported that "the patient denied dreaming" upon rapid eye movement (REM) awakening, whereas Brown (1972, p. 210) stated that "a REM study was normal in character but revealed absence of *visual elements* in his dream" (italics added).

The 106 cases are summarized in Table 34.2. Defective revisualization (irreminiscence) is a common but by no means invariable feature of these cases. It was overrepresented in the earlier case reports for the probable reason that patients were only asked about their dreams once irreminiscence had been established. The more recent cases reported by Solms (1997) were part of an unselected clinical series and are therefore more likely to be representative. Global cessation of dreaming (unlike visually deficient dreaming) therefore cannot be reduced to irreminiscence.

No other single cognitive deficit has been identified that discriminates statistically between global nondreamers and dreamers, apart from visuospatial short-term memory disorder, which is also not an invariable feature (Solms, 1997). The lack of association between cessation of dreaming and long-term memory disorder of any kind excludes the possibility that cessation of dreaming is really a disorder of episodic memory – failure to remember dreams as opposed to loss of dreaming *per se* (Feinberg, 2000; LaBerge, 2000; Ogilvie et al., 2000). This applies also to the various disorders of language that have been thought to explain absence of dream recall (Zinkin, 1959; Anan'ev, 1960; Moss, 1972; Jakobson, 1973; Doricchi and Violani, 1992).

Retrospective absence of dreaming on morning awakening has repeatedly been confirmed on REM awakening (Michel and Sieroff, 1981; Schanfald et al., 1985; Bischof and Bassetti, 2004; Poza and Marti, 2006). This further supports the assumption that this disorder concerns cessation of dreaming *per se*. Even severe amnesiacs with bilateral hippocampal lesions report dreams on REM awakening (Torda, 1969; Ramachandran, 2004, personal communication).

The first systematic attempt to identify the lesion site responsible for global cessation of dreaming pointed to the inferior parietal lobule of either hemisphere (Solms, 1997). Unilateral lesions were shown to be commonplace, with no lateralizing bias. However, at least one case has since been reported in which the parietal lobe was unequivocally spared (Bischof and Bassetti, 2004), as indeed it appears to have been in Wilbrand's original case (Wilbrand, 1892). A reanalysis of Solms's data by Yu (2001a) revealed that the lesions in his parietal cases almost always extended into adjacent occipitotemporal tissues (especially areas 22, 19, and 37).

It is therefore still not possible to make a more precise localizing statement than the one offered by Murri et al. (1984, p. 185): "a lesion in an acute phase affecting the posterior regions." The reference here to an acute phase is not superfluous. Solms (1997) observed that almost all cases recover the capacity to dream within 12 months. This fact, which suggests diaschitic effects, may help explain the imprecise localization of the associated lesion.

Cessation or suppression of dreaming following prefrontal leukotomy

In a survey of 200 cases of prefrontal leukotomy, Frank (1946) observed that a common result of the procedure was "a poverty or entire lack of dreams" (p. 508). In a later report on the same series of cases, then comprising more than 300 patients, he confirmed his observation, adding that dreaming was typically "simplified" in leukotomy cases with preserved dreaming (Frank, 1950, p. 38). Replication of these findings was forthcoming from other authors (Table 34.3). Jus et al. (1973) confirmed the absence of dream reports on REM awakening.

In apparent contradiction to these reports, however, Humphrey and Zangwill (1951), Cathala et al. (1983), Murri et al. (1984, 1985), and Doricchi and Violani (1992) all observed a relatively low incidence of cessation of dreaming with anterior versus posterior cerebral lesions. The same applies to the observation reported above to the effect that the dreams of frontal lobe patients are indistingishable from those of controls (Badenhorst and Solms, unpublished). This contradiction was resolved when Solms (1997) reviewed the lesions in the previously reported cases and described 9 new cases of his own with cessation of dreaming following naturally occurring frontal lesions (Table 34.4). His conclusion was that dreaming was spared with dorsolateral prefrontal cortical lesions and affected only with deep white-matter lesions in the ventromesial quadrant of the frontal lobes (Figures 34.1 and 34.2). The lesion site in his 9 cases coincided exactly with the area that was targeted by prefrontal leukotomy: "a circumscribed lesion just anterior to the frontal horns of the ventricle, in the lower medial quadrant of the frontal lobes" (Walsh, 1994, p. 177).

A reanalysis of the original data in 35 cases from Solms's series with global cessation of dreaming associated with subcortical lesions revealed that the lesion was located in either the deep frontal white matter (areas F09 and F14 in the classification of Damasio and Damasio, 1989; Figure 34.3), or the head of the caudate nucleus, or both (Yu, 2001b). Of theoretical importance is the fact that the region defined as the head of the caudate nucleus (located in the ventromesial prefrontal region, attached to the inferior caudal part of the frontal horn of the lateral ventricle, surrounded by the white matter enclosing the frontal horn) included the nucleus accumbens, which is situated immediately beneath it.

Table 34.2

Cases of global cessation or suppression of dreaming

Source	Case	Lesion	Irreminiscence	Dreams
Wilbrand (1887, 1892)	Fräulein G.	R. med. occip. temp., L. deep occip. (thrombosis)*	_	Either global cessation with recovery over approx. 4 years or gross reduction in frequency (description ambiguous)
Müller (1892)	Frau Anna Hoffmann, 50y	Bilat. occip. (hemorrhage)[b]	+	Global cessation
Grünstein (1924)	(p. 420)	(L. lat. temp.)	_	Global cessation
	(p. 420)	(L. lat. temp.)	_	Global cessation
Lyman et al. (1938)	42y M	L. deep occip. par. (meningioma)§	+	Global cessation
Humphrey and Zangwill (1951)	Case 1, 26y M	R. par.‖ (trauma)	+	Global cessation with partial recovery or gross reduction in frequency (description ambiguous)
Humphrey and Zangwill (1951)	Case 2, 21y M	Bilat. lat. par.‖ (trauma)	+	Global cessation and cessation of hypnagogic imagery
Humphrey and Zangwill (1951)	Case 3, 32y M	R.¶ (lat?) par.§ (trauma)	+	Global cessation with recovery after approx. 5 years
Oldfield (Humphrey and Zangwill, 1951)	(p. 324n)	R. occip. par. (abscess)§	(No information)‡	Global cessation
Gloning and Sternbach (1953)	S. Johann, 48y M	R. deep front. temp. (glioma)§	_	Increased proprioceptive-vestibular imagery (falling and flying); residual reduction in frequency
	L. Josef, 64y M	L. med. occip. temp., R. post. occip. (thrombosis)*	+	Global cessation
	K. Franz, 56y M	L. med. occip. temp. (thrombosis)†	+	Global cessation with recovery after approx. 1 year; residual reduction of visual imagery
	W. Karl, 52y M	R.‖ front.† (and temp. par.) (thrombosis)	_	Global cessation
	M Klara, 32y F	R. deep front. temp. (glioma)§	_	Global cessation
	Sch. Gertrude, 56y F	Bilat. deep front. (glioma)§	_	Increased vivacity, later becoming global cessation
	M. Josef, 24y M	R. deep par. temp. (hemorrhage)‖	_	Gross reduction in frequency
Gloning and Sternbach (1953)	E. Ernst, 57y M	L. occip. thalam. (thrombosis)†	_	Global cessation with gradual recovery over approx. 3 years
	N. Johann, 51y M	L. thalam (thrombosis)†	_	Global cessation with recovery after 5 months

Continued

Table 34.2

Continued

Source	Case	Lesion	Irreminiscence	Dreams
Boyle and Nielsen (1954); Nielsen (1955)	E. S., 31y M	3rd. ventric. (cyst) (with hydroceph.)‖ L. deep occip. temp.,† R. occip.§ (surgical trauma)	+	Global cessation
Nielsen (1955)	Harley S., M	L. occip. (glioma)§	+	Global cessation
	(p. 52)	L. occip. (tumor)	+	Global cessation
	(p. 52)	L. occip. (tumor)	+	Global cessation
	F	Bilat. occip.§ (trauma)	_	Global cessation
Ettlinger et al. (1957)	N. H., 57y M	R. occip. temp.* (metastatic tumor)	+	Global cessation
Ritchie (1959)	Douglas Ritchie, 50y M	L. (thrombosis)†	_	Global cessation with recovery after approx. 2 years
Farrell (1969); Neal and Deneut (1988)	Patricia Neal, 39y F	L. temp. (par.) (hemorrhage)§	+	Global cessation
Feldman (1971)	36y F	Ventral pons (thrombosis)†	_	Global cessation
Moss (1972)	C. Scott Moss, 43y M	L. (thrombosis)†	_	Global cessation with recovery after 4 months
Wapner et al. (1978)	J. R., 73y M[f]	Bilat. occip. (thrombosis)‖	+	Global cessation
Epstein (1979)	Patient 1, 56y F	L.¶ med. par. occip. (thrombosis)†	+	Global cessation with gradual recovery after approx. 5 months
Basso et al. (1980)	M G., 63y M	L. med, occip. temp., cerebellum (thrombosis)‖	+	Global cessation and cessation of hypnagogic imagery
Epstein and Simmons (1983)	Case 1, 47y F	L. front. Temp. (hemorrhage)§	_	Global cessation
	Case 2, 35y F	L. front. (thrombosis)‖	_	Global cessation
	Case 3, 33y M	L. front. temp. (thrombosis)‖	_	Global cessation
	Case 4, 56y F	L. (thrombosis)†	_	Global cessation
	Case 5, 52y F	L. (thrombosis)†[b]	_	Global cessation
	Case 6, 43y F	L. (thrombosis)†	_	Global cessation
	Case 7, 59y F	L. deep. par. occip.‖	_	Global cessation
Peña-Casanova et al. (1985)	A. R. 47y M	L. med. occip. temp. (thrombosis)‖	+	Global cessation with gradual recovery after approx. 5 months
Habib and Sirigu (1987)	Case 2, 26y F	R.¶ med. temp. (thrombosis)‖	_	Either global cessation or specific amnesia (description ambiguous)
Farah et al. (1988)	R. M, 64y M	L. med. occip. temp. (thrombosis)‖	+	Global cessation
Solms (1997)	Patient 8, 10y M	R. med. par. temp. meningioma‖	_	Global cessation
	Patient 17, 36y M	R. par. temp. stab wound with intracerebral hemorrhage‖	_	Global cessation

Solms (1997)	Patient 23, 28y F	L. par. temp. limbic infarct following subarachnoid hemorrhage (L. mid. cereb. art. aneurysm)‖	_	Global cessation
	Patient 27, 29y F	L. par. front. depressed fracture with abscess formation (assault)‖	_	Global cessation with recovery within 1 year
	Patient 43, 22y F	L. par. infarct (L. int. carotid. art. traumatic occlusion)‖	_	Global cessation
	Patient 44, 44y F	R. par. front. meningioma (with R. mid. cereb. art. infarction)‖	_	Global cessation for at least 2 years
	Patient 48, 42y M	L. par. temp. arachnoid cyst‖	_	Global cessation with recovery within 1 year
	Patient 49, 29y M	R. lat. par. temp. front. chronic subdural hemorrhage (MVA)‖	_	Global cessation for at least 2 years
	Patient 57, 29y M	R. lat. par. front. chronic subdural hemorrhage (assault)‖	_	Global cessation
	Patient 71, 42y M	L. deep par. temp. cyst (cysticercosis)‖	_	Global cessation
	Patient 86, 46y F	L. par. front. infarct following subarachnoid hemorrhage (L. mid. cereb. art. aneurysm)‖	_	Global cessation
	Patient 87, 60y M	L. par. front. limbic depressed fracture with abscess formation (assault)‖	_	Global cessation
	Patient 99, 34y M	L. lat. par. front. temp. acute subdural hemorrhage with L. front. intracerebral extension‖	_	Global cessation
	Patient 119, 14y F	R. deep par. temp. limbic astrocytoma‖	_	Global cessation with recovery within 1 year
	Patient 122, 37y M	Bilat. occip. subdural and cerebellar extradural hemorrhages (assault)	_	Global cessation
	Patient 130, 56y F	R. par. front. meningioma (with cortical infiltration)‖	_	Global cessation with recovery within 1 year
	Patient 134, 54y F	R. lat. par. temp. meningioma‖	_	Global cessation
	Patient 143, 27y M	R. lat par infarct (R. mid. cereb. art. thrombosis)‖	_	Global cessation
	Patient 157, 40y F	L. lat. par. occip. infarct following subarachnoid and intracerebral hemorrhage (L. mid. cereb. art. aneurysm)‖	_	Global cessation with recovery within 1 year
	Patient 162, 73y M	R. par. occip. intracerebreal hemorrhage (hypertention)‖	_	Global cessation
	Patient 168, 35y M	L. par. temp. limbic astrocytoma	_	Global cessation

Continued

Table 34.2

Continued

Source	Case	Lesion	Irreminiscence	Dreams
Solms (1997)	Patient 173, 34y M	Par. temp. limbic intracerebral hemorrhage (AVM fed by R. mid. cereb. art)‖	_	Global cessation with recovery within 1 year
.	Patient 174, 29y M	Vertex and L. temp stab wounds with medial L. hemispheric subarachnoid and temp. intracerebral hemorrhages (pericallosal traumatic aneurysm)	_	Global cessation
	Patient 186, 65y F	L. lat par. temp. meningioma‖	_	Global cessation
	Patient 187, 17y F[f]	R. med. par. meningioma‖	_	Global cessation
	Patient 189, 49y F	Bilat. med. occip. temp multiple lacunar infarctions (region of post. cereb. art)	_	Global cessation
	Patient 191, 33y F	Bilat. par. front. and L. temp. multiple intracerebral hemorrhage (lupus erythematosus)‖	_	Global cessation
	Patient 217, 42y F	Pontine cerebellar (acoustic neuroma); postsurgery R. occip-temp. intracerebral hemorrhage	_	Global cessation
	Patient 224 39y F	R. lat. par. front. occip. infarct (R. mid. cereb. art. thrombosis)‖	_	Global cessation
	Patient 234, 77y M	R. deep par. occip. temp. limbic glioma‖	_	Global cessation
	Patient 238, 63y F	R. med. par. front. limbic meningioma‖	_	Global cessation
	Patient 241, 18y M	Bilat. lat. par. front. occip. dermoid cyst with R. front. chronic subdural hemorrhage‖	_	Global cessation
	Patient 244, 77y M	L. deep par. occip. Metastatic carcinoma‖	_	Global cessation
	Patient 263, 43y F	L. med. occip. temp. limbic meningioma	_	Global cessation
	Patient 268, 67y M	R. lat. par. front. subdural empyema‖	_	Global cessation
	Patient 273, 27y M	L. par. front. open head injury (MVA)‖	_	Global cessation with recovery within 1 year
	Patient 284, 23y M	R. par. open head injury (stab wound)‖	_	Global cessation
	Patient 285, 18y M	L. lat. par. front. open head injury (gunshot wound)‖	_	Global cessation
	Patient 286, 31y F	L. par. open head injury (stab wound)‖	_	Global cessation with recovery within 1 year

	Patient 288, 67y F	L. front. temp. subdural and intracerebral hemorrhages (assault)	_	Global cessation
	Patient 289, 35y M	R. temp. and cerebellar intracerebral hemorrhages and shrapnel/bone (bomb blast)	_	Global cessation
	Patient 299, 54y M	L. occip. temp. limbic glioma (astrocytoma)	_	Global cessation
	Patient 302, 32y F	L. lat. par. front. infarct (L. mid. cereb. art. thrombosis)‖	_	Global cessation
	Patient 303, 25y M	L. lat. par. front. acute subdural hemorrhage (MVA)‖	_	Global cessation with recovery within 1 year
	Patient 305, 58y M	R. lat. par. temp. front. infarct (L. mid. cereb. art. thrombosis)‖	_	Global cessation
	Patient 306, 33y F	L. lat. par. temp. infarct (L. mid. cereb. art. thrombosis)‖	_	Global cessation
	Patient 312, 59y M	R. lat. par. temp. front. infarct (R. mid. cereb. art. thrombosis)‖	_	Global cessation with recovery within 1 year
	Patient 321, 35y M	R. lat par. temp. front. infarct (mid. cereb. art) following subarachnoid hemorrhage (ant. comm. art. aneurysm)‖	_	Global cessation for at least 2 years
	Patient 327, 44y F	R. deep par. intracerebral hemorrhage‖	_	Global cessation
	Patient 336, 77y M	L. lat. par. front. metastatic carcinoma	_	Global cessation
	Patient 338, 54y M	L. deep par. astrocytoma‖	_	Global cessation
	Patient 341, 18y M	L. lat. par. front. acute subdural hemorrhage (MVA)‖	_	Global cessation
	Patient 345, 43y M	R. lat. par. front. acute subdural hemorrhage (MVA)‖	_	Global cessation
	Patient 349, 63y F	L. med. par. front. meningioma‖	_	Global cessation
	Patient 353, 50y M	R. lat. par. front. subdural-extradural hemorrhage (assault)‖	_	Global cessation
Bischof and Bassetti (2004)	72y F	Bilat. med. occ. R. thalam. (bilat post. cereb. art. thrombosis)‖	_	Global cessation with gradual recovery after 14 weeks; residual reduction in frequency
Poza and Marti (2006)	24y M	L. temp. occip. hemorrhage‖	_	Global cessation

y, year; F, female; M, male; AVM, arteriovenous malformation; MVA, motor vehicle accident.
*Autopsy-confirmed localization.
†Clinical localization or diagnosis.
‡Unless otherwise indicated, it is assumed that the authors comprehensively investigated and reported the neuropsychological status of their cases.
§Localization or diagnosis based on intraoperative observation.
¶Localization or diagnosis based on *in vivo* imaging techniques.
¶Left-handed or ambidextrous patient.

Table 34.3

Global cessation or suppression of dreaming in clinical series of prefrontal leukotomy

Source	Series	Dreams
Frank (1946)	100 cases, 22–26 years, 100 M, 100 F	Global cessation and decreased frequency or narrative complexity (description ambiguous) in an unspecified number of cases
Frank (1950)	300+ cases	Global cessation or decreased frequency (description ambiguous) and decreased narrative complexity and emotional intensity in an unspecified number of cases
Piehler (1950)	58 cases, 21 M, 37 F	Global cessation in 11 cases; recovery over 1 year with residual reduction in frequency, vivacity, narrative complexity, and emotional intensity in 8 of these
Partridge (1950)	300 cases	Global cessation or gross reduction in frequency (description ambiguous) in several cases; recovery over 6 months–1 year in an unspecified number of cases
Slater (cited in Humphrey and Zangwill, 1951)	"A number of patients"	Global cessation or reduction in frequency (description ambiguous)
Schindler (1953)	150+ cases	Global cessation in 134 cases; recovery several months to 1 year with residual reduction in narrative complexity in an unspecified number of cases
Jus et al. (1973)	13 cases, 31–63 years, 1 M, 12 F; 13 matched (schizophrenic) controls	Global cessation in 9 cases (sleep lab confirmed in 7 of these); global cessation claimed by 3 controls (not confirmed in lab)
Freeman (cited in Jus et al., 1973)	No information	Global cessation or reduction in frequency (description ambiguous)

M, male; F, female.

It is noteworthy that the psychotropic medications that replaced prefrontal leukotomy as the treatment of choice for psychotic disorders block dopamine (DA) transmission in a mesial forebrain pathway that projects primarily to the nucleus accumbens. Probably related to this is the observation that both prefrontal leukotomy in general and cessation of dreaming in particular, due to lesions in this general area, are associated with reduced motivational incentive (Solms, 1997), as indeed are most antipsychotic medications (Kapur, 2003). Also of interest in this connection is the observation by Piehler (1950) and Schindler (1953) to the effect that early recovery of dreaming after prefrontal leukotomy typically coincides with psychiatric relapse, suggesting that absence of dreaming could serve as an index of the clinical success of the operation. Dreaming is, after all, a psychotic state.

Effects of pontine brainstem lesions

Despite the longstanding assumption that dreaming is caused by – if not identical with – effects on the sleeping forebrain of the cyclical, spontaneous activation of cholinergic (ACh) cells in the mesopontine tegmentum during the REM state, together with reciprocal inhibition of serotonergic (5-HT) and noradrenergic (NA) cells in the dorsal raphe and locus coeruleus complex (Hobson et al., 1975, 1986), cessation of dreaming following circumscribed pontine lesions, with or without cessation of REM sleep, has never been demonstrated (see Solms 1997, 2000, for reviews). Consciousness in general is of course frequently compromised by pontine lesions, but at least 8 cases with cessation or near-cessation of REM have been reported in which patients were capable of communicating meaningfully about their dreams (Feldman, 1971; Markand and Dyken, 1976; Osorio and Daroff, 1980; Lavie et al., 1984). Indeed, one such patient did actually report loss of dreaming (Feldman, 1971), but the lesion – caused by ruptured traumatic aneurysm of the basilar artery – probably extended beyond the pontine brainstem and included the visual cortical areas discussed above. Even this isolated case therefore does not support the old equation of pontine brainstem mechanisms with dream generation. (The relationship between dreaming and REM sleep is discussed further below.)

Table 34.4

Cases of global cessation of dreaming with frontal lobe lesions

Source	Case	Lesion	Irreminiscence	Dreams
Piehler (1950)	Case 1, 49y M	Bilat. deep front. (leukotomy)†	_	Global cessation with recovery after approx. 6 weeks; global cessation again after 2nd op. with recovery after approx. 2 years; residual reduction in frequency, narrative complexity, and intensity
	Case 2, 36y M	Bilat. deep front. (leukotomy)†	_	Global cessation with gradual recovery after approx. 4 months
	Case 3, 34y F	Bilat. deep front. (leukotomy)†	_	Global cessation with recovery after approx. 3 months; residual reduction in frequency
	Case 4, 33y F	Bilat. deep front. (leukotomy)†	_	Global cessation with recovery after approx. 3 months
Gloning and Sternbach (1953)	(p.318)	Bilat. deep front. (leukotomy)†	(No information)*	Global cessation
	(pp. 318–319)	Bilat. deep front. (leukotomy)†	(No information)	Global cessation
Schindler (1953)	Case 89	Bilat. deep front. (leukotomy)†	(No information)	Global cessation of imagery (dream feeling without imagery)
	Case 111	Bilat. deep front. (leukotomy)†	(No information)	Global cessation of imagery (dream atmosphere without imagery)
Solms (1997)	Patient 9, 13y F	Bilat. deep front. thalam. glioma (glioblastoma multiforne)†‡	_	Global cessation
	Patient 129, 14y M	Bilat. deep frontal Abscess‡	_	Global cessation
	Patient 150, 58y F	Suprasellar pituitary tumour with med. front. limbic-ventricular extension (prolactinoma-macroadenoma)†‡	_	Global cessation for at least 12 weeks
	Patient 181, 35y M	L. deep front.abscess and intracerebral hemorrhage; R med. front. intracerebral hemorrhage and subdural empyema‡	_	Global cessation

Continued

Table 34.4

Continued

Source	Case	Lesion	Irreminiscence	Dreams
Solms (1997)	Patient 214, 18y M	L. basal front. intracerebral hemorrhage; R. carotid-cavernous fistula‡	_	Global cessation
	Patient 261, 62y M	Bilat. med. frontal meningioma†‡	_	Global cessation
	Patient 280, 63y F	Bilat. calloso-front. limbic glioma (glioblastoma multiforme)†‡	_	Global cessation
	Patient 281, 44y F	Bilat. med. front. limbic subarachnoid and intracerebral hemorrhage (ant. comm. art. aneurysm)‡	_	Global cessation for at least 10 weeks
	Patient 291, 21y F	L. med. front. limbic diencephalic abscess (stab wound)‡	_	Global cessation

M, male; F, female.
*Unless otherwise indicated, it is assumed that the authors comprehensively investigated and reported the neuropsychological status of their cases.
†Localization or diagnosis based on intraoperative observation.
‡Localization or diagnosis based on *in vivo* imaging techniques.

Excesses of dreaming

Solms (1997) loosely grouped together 12 case reports in the literature and 10 of his own cases that reported excesses of dreaming, ranging from increased frequency and/or vivacity of dreams to intrusions of dreaming and dream-like thinking into waking cognition (Table 34.5). The principal justification for collecting these cases together under a single nosological heading seems to be that the lesions are frequently located in the transitional zone between the anterior diencephalon and basal forebrain. Kindred phenomena are, however, observed with visual deafferentation, peduncular hallucinosis, delirium, Parkinson's disease and related disorders, and a variety of toxic and metabolic abnormalities. The common denominator in these cases may therefore simply be degradation of controls and constraints on consciousness.

Recurring nightmares

Nocturnal seizures (and complex partial seizures in particular) sometimes present as recurring nightmares. Solms (1997) identified 24 cases of this type in the literature and 9 in his own series (Table 34.6). Of theoretical interest is the fact that such nightmares typically occur during non-REM sleep, and that complex partial seizures preclude recruitment of pontine brainstem mechanisms. In such cases, the content of the recurring nightmares frequently coincides with that of the patient's typical aura or "dreamy state" seizures. Penfield was able artificially to generate a waking aura resembling the recurring nightmare in 1 case by stimulating exposed cortex in the region of the epileptogenic focus (Penfield, 1938; Penfield and Erickson, 1941; Penfield and Rasmussen, 1955). Successful pharmacological or surgical treatment of the seizure disorder invariably results in disappearance of the recurring nightmares. These facts support the interpretation of the nightmares as seizure equivalents (and indeed as non-REM phenomena). Seizure disorders may also be associated with increased frequency of nightmares in general (Solms, 1997). However, as with other "excess of dreaming" described above, increased frequency of nightmares is associated with a wide range of toxic and metabolic abnormalities. The common



Fig. 34.1. Combined facsimile of scans in 9 cases with global cessation of dreaming caused by deep frontal lesions, illustrating the strong involvement of the white matter surrounding the frontal horns of the lateral ventricles. (Reproduced from Solms (1997).)

denominator here may therefore simply be nocturnal cerebral irritability.

FUNCTIONAL NEUROIMAGING FINDINGS

For technical reasons, functional brain imaging of dreaming sleep has relied mainly upon positron emission tomography (PET). Due to limitations of temporal resolution in PET, "dreaming sleep" has typically been operationalized as REM sleep in these studies. Since dreaming is approximately three times more common in REM than non-REM sleep, it is reasonable to assume that PET images of the REM state should at least include images of the dreaming brain. In other words, PET at least enables us to visualize the pattern of regional cerebral activation in the combined dreaming and REM state. The canonical studies (Maquet et al., 1996, 1997; Braun et al., 1997, 1998; Nofzinger et al., 1997) are summarized in Maquet (2000).

If the well-known brainstem correlates of REM activation are set aside for the methodological reasons just mentioned, the pattern of regional cerebral activation in the available PET studies of dreaming sleep

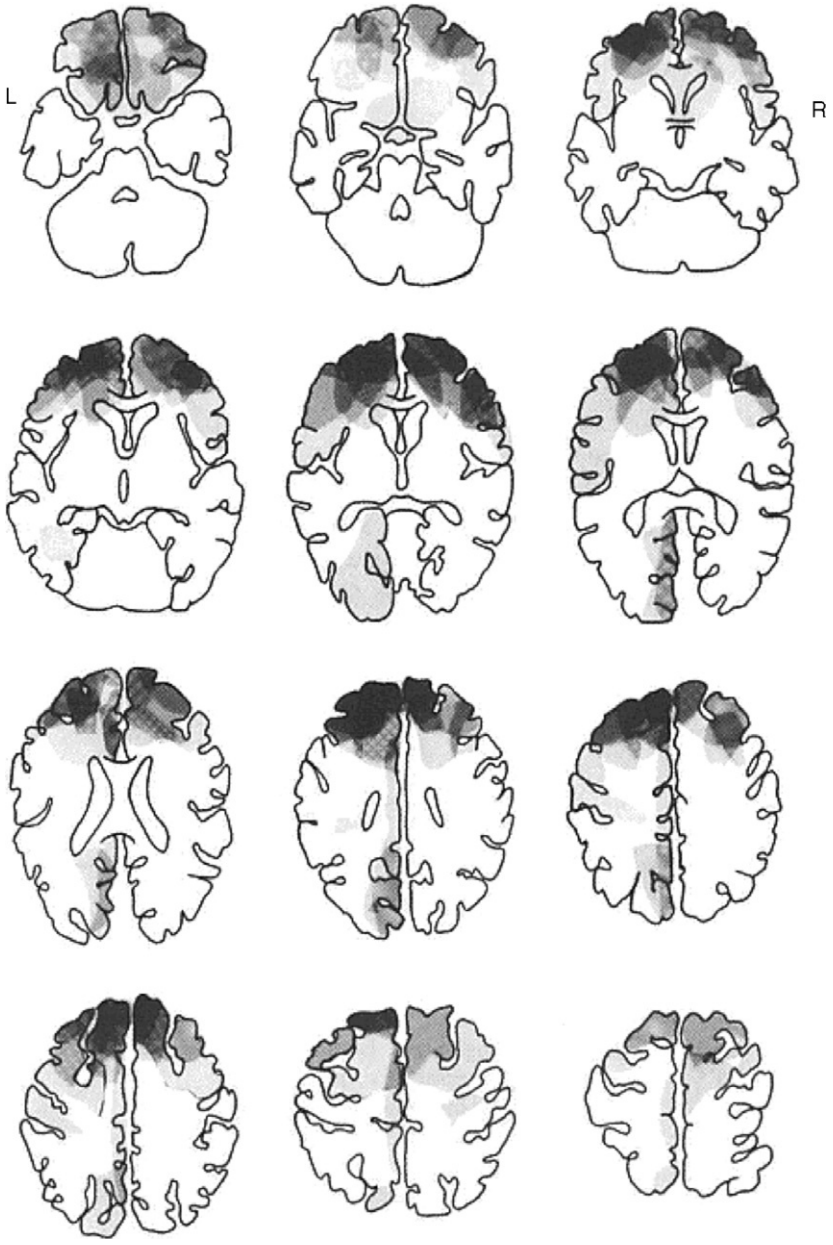


Fig. 34.2. Combined facsimile of scans in 14 cases with preserved dreaming with bifrontal lesions, illustrating the relative preponderance of cortical convexity involvement. (Reproduced from Solms (1997).)

(compared to other sleep stages and waking) is strikingly convergent with the clinicoanatomical findings summarized in previous sections (Pace-Schott and Hobson, 1998). The emerging picture seems to be: (1) activation of limbic and paralimbic structures and basal ganglia; (2) activation of modality-specific posterior cortices (including specifically areas 19, 22, and 37; see above); and (3) deactivation of the dorsolateral prefrontal convexity (Figure 34.4).

In general, the brain regions implicated in dream generation by lesion studies are relatively activated in the PET studies, and those not implicated by the lesion studies are deactivated in the PET studies. Particularly impressive in this respect is the precise distinction between early visual projection cortices, where lesions spare visual dreaming and PET shows reduced metabolic activity, and later visual association cortices, where lesions impair visual dreaming and PET shows increased activity (Solms, 1997; Braun et al., 1998).

The strong convergence between the lesion and functional neuroimaging findings has prompted wide

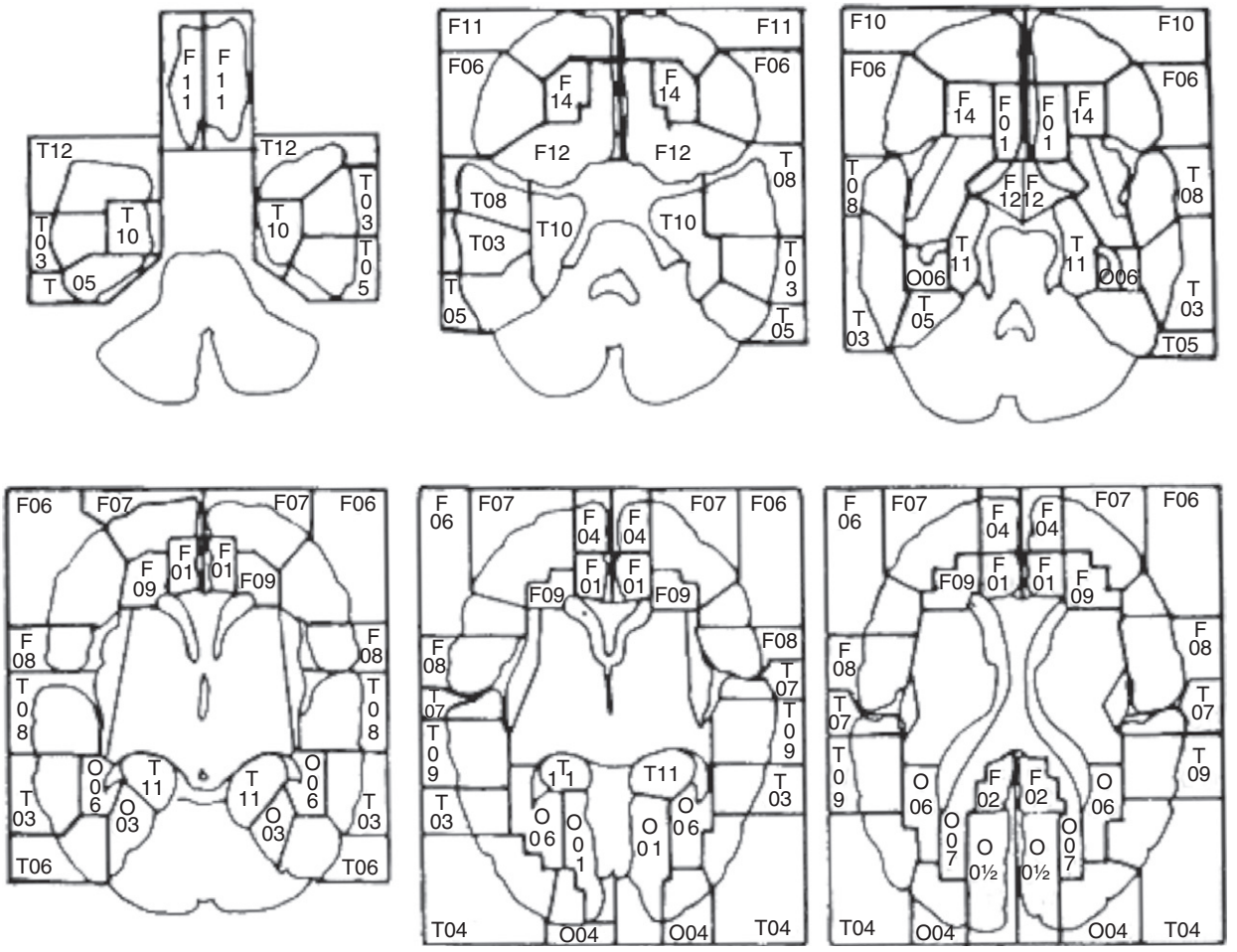


Fig. 34.3. The classification of Damasio and Damasio (1989).

agreement that "frontal deactivation must play a key role in any neurocognitive theory of dreaming" (Pace-Schott, 2003, pp. 340–341). The hypothesis that sleep involves disengagement of prefrontal systems from posterior perceptual and deeper memory and emotional systems holds great value for explaining dream phenomenology.

NEUROCHEMICAL AND PSYCHOPHARMACOLOGICAL FINDINGS

The chemical and pharmacological evidence is difficult to interpret. This is due partly to the dynamic interactions that characterize neurotransmitter systems, and partly to the paucity of rigorous pharmacological studies (Hobson, 2001). Mention will be made here of recent findings which seem particularly relevant to understanding dream generation and the distinction between dreaming and REM sleep.

The neurochemical signature of the REM state is well established: autochthonous activation of ascending pontine ACh cells,which is thought to produce characteristic pontine-geniculate-occipital waves, and reciprocal inhibition of pontine aminergic (5-HT and NA) cells, which is thought to demodulate the dreaming forebrain (Hobson et al., 2000). Equally well established is the fact that non-REM sleep has the opposite pattern. Less widely known is the fact that, unlike other aminergic brainstem cells, the source cells in the ventral tegmental area of the mesocortical DA pathway described above continue to fire at equivalent rates during sleeping and waking (Miller et al., 1983; Trulson and Preussler, 1984). These cells also fire with greater interspike variability during REM than non-REM sleep (Miller et al., 1983). This possibly indicates increased burst activity in the REM state, which would result in greater terminal DA release. Whatever the mechanism, the result is maximal delivery of DA to the nucleus accumbens during REM sleep (Lena et al., 2005). The REM state is also characterized by minimal prefrontal glutamate release (Lena et al., 2005).

Table 34.5

Cases of increased frequency and vivacity of dreaming

Source	Case	Lesion	Relevant symptoms and signs	Dreams
Grünstein (1924)	Patient N., 21y M	Bilat. occip. (trauma)	Cortical blindness	Increased vivacity, recovering gradually over approx. 3 weeks; residual increased frequency
Gloning and Sternbach (1953)	M. Johanna, 27y F	Hypothalam. (tumor)	Blindness	Increased vivacity
	H. Therese, 27y F	Bilat. med. front. (meningioma)	Disinhibition	Increased vivacity (with nightmares), gradually decreasing in frequency over 2 years
	S. Josef, 54y M	Hypothalam. thalam. (craniopharyngioma)	Disinhibition	Increased frequency and vivacity (with nightmares), recovery after approx. 1 year
	E. Gustav, 44y M	R. thalam. (thrombosis)	Disinhibition	Increased frequency and vivacity
	K. Rudolf, 50y M	R. thalam. (thrombosis)	Disinhibition	Increased vivacity
Whitty and Lewin (1957)	Case 1, 58y M	Bilat. med. front. (cingulectomy)	Disinhibition	Continuous dreaming with increased vivacity, blurred fantasy/reality distinction; recovery over approx. 4 days
	Case 2, 31y F	Bilat. med. front. (cingulectomy)	Disinhibition	Continuous dreaming with increased vivacity, blurred fantasy/reality distinction; recovery over approx. 2 days
	Case 3, 60y M	Bilat. med. front. (cingulectomy)	Disinhibition	Continuous dreaming with increased vivacity, blurred fantasy/reality distinction; recovery over approx. 4 days

Lugaresi et al. (1986); Gallassi et al. (1992)	S.S., 53y M	Bilat. thalam. (atrophy)	Disinhibition	Increased vivacity with enactment, gradually increasing in frequency over approx. 6 months, becoming continuous)
Gallassi et al. (1992)	L.S., 29y F	No information		Increased vivacity, gradually increasing in frequency
Morris et al. (1992)	S.J., 51y M	R. hypothalam. (atrocytoma)	Disinhibition; confabulatory amnesia	Increased vivacity; blurred fantasy/reality distinction
Sacks (1995)	Franco Magnani, 54y M	No information (febrile illness)	Blindness	Increased frequency (continuous dreaming?) and increased vivacity; blurred fantasy/reality distinction
Solms (1997)	Case 17, 62y M	Bilat. med. front. cingulate g., basal forebrain; R. lat. front. temp. par. (ant. comm. aneurysmal rupture with ant. and mid. cereb. spasm)	Disorientation, confabulatory amnesia, disinhibition, fluctuating affect, reduplicative paramnesia, neglect, reduced insight	Increased reality in context of global fantasy/reality breakdown
	Case 18, 32y M	Bilat. cingulate g., postcentral, precentral and premotor g., R. lat. par. occip. (open head injury)	Disinhibition, euphoria/ placidity, visual hallucination, neglect, anosodiaphoria	Increased reality and frequency with circumscribed fantasy/ reality breakdown (and with repetitive nightmares)
	Case 19, 30y M	R. med. front. cingulate g., basal forebrain (ant. comm. aneurysmal rupture with ant. cereb. spasm)	Disorientation, confabulatory amnesia, disinhibition, fluctuating affect, reduplicative paramnesia, anosognosia	Increased reality in context of global fantasy/reality breakdown
	Case 20, 30y M	R. med. thalamic astrocytoma	Torpor/stupor, disorientation, confabulatory amnesia, paranoia, neglect, reduced insight	Increased reality in context of global fantasy/reality breakdown

Continued

Table 34.5

Continued

Source	Case	Lesion	Relevant symptoms and signs	Dreams
Solms (1997)	Case 21, 61y F	Hypothalam. (suprasellar pituitary adenoma)	Disinhibition, fluctuating affect, reduced insight	Increased reality and frequency (with increased frequency of nightmares)
	Case 22, 44y F	L. med. front., bilat. cingulate g., basal forebrain (ant. comm. aneurysmal rupture with front. intracerebral hemorrhage extension)	Amnesia, visual hallucination, reduplicative paramnesia, neglect	Increased reality in context of global fantasy/reality breakdown and recovery
	Case 23, 27y M	Bilat. med. thalam. (colloid cyst of the third ventricle)	Torpor, amnesia, irritability	Increased reality and frequency with circumscribed fantasy/ reality breakdown
	Case 24, 44y F	Hypothalam, bilat. med. thalam. (craniopharyngioma)	Disorientation, amnesia, fluctuating affect, anosognosia	Increased reality (and frequency of nightmares)
	Case 25, 32y F	Bilat. med. frontal, cingulated g. (open head injury with ?R. ant. cereb. infarct)	Torpor, fluctuating affect, paranoia	Increased reality and frequency with circumscribed fantasy/ reality breakdown (with repetitive nightmares) and recovery

Table 34.6

Cases of recurring nightmares associated with seizures

Source	Case	Lesion/focus	Dreams
Clarke (1915)	Case 3, 24y M	No information	Repetitive nightmares, with subsequent development of manifest epilepsy associated with recurrent dreams which sometimes culminated in seizures
	Case 8, 35y M	No information	Recurring nightmare, disappearing with onset of manifest epilepsy
Kardiner (1932)	Case 4 (A.R.) 13y F	No information	Repetitive nightmares of sudden onset, with subsequent development of manifest epilepsy
	Case 5, 21y F	No information	Repetitive nightmares, sometimes continuous, with subsequent development of manifest epilepsy
Naville and Brantmay (1935)	Case 18, 11y F	No information	Frequent nightmares associated with onset of epilepsy
Penfield (1938)	J.V., 14y M	R. lat. temp. occip. (hemorrhage)	Recurring nightmare, later becoming hallucinatory aura; reproducible by R. temp. stimulation
Penfield and Erickson (1941)	C. Ft., 27y M	R. deep temp. (glioma)	Repetitive series of dreams, typically culminating in nocturnal seizure; reproducible by R. temp. stimulation
Rodin et al. (1955)	Case 3, 23y F	Bilat. temp. EEG foci, R.>L.	Dreams of recurring (unpleasant) form, typically culminating in nocturnal seizure
Ostow (1954)	Case 3, 36y F	R. temp. EEG focus	Recurring (unpleasant) dreams, related in content to compulsive waking thoughts and seizure automatisms
	Case 4, 38y M	Bilat. temp. EEG slowing, L.>R.	Nightmares of recurring form, related in content to compulsive waking thoughts
Epstein and Ervin (1956)	H.D. 35y F	R. temp. EEG focus	Recurring nightmares, related in content to compulsive waking thoughts and seizure automatisms
	L.E., 22y F	R. bilat. temp EEG slowing	Repetitive nightmares, being nocturnal seizures, related in content to waking complex partial seizures
Snyder (1958)	A.M, 9y M	R. par. EEG focus	Nightmares of sudden onset, with confusion or complaints of headache on awakening, controlled by anticonvulsant medication
Epstein (1964)	38y M	L. temp. EEG focus	Recurring nightmare, later becoming hallucinatory seizure or aura (description ambiguous)
	23y F	R. temp. EEG focus	Recurring nightmares, gradually becoming more frequent and vivid, later becoming nocturnal seizure or aura with blurred fantasy/reality distinction; controlled by anticonvulsant medication

Continued

Table 34.6

Continued

Source	Case	Lesion/focus	Dreams
Epstein and Hill (1966)	35y F	R. temp. focus (encephalitis)	Recurring nightmares, typically culminating in or being nocturnal seizure; associated with R. temp. spiking during REM sleep
Epstein (1967)	Case 1, 41y F	Bilat. temp. EEG foci, R.>L.	Nightmares of recurring form, typically being nocturnal seizures
	Case 2, 20y F	R. par. EEG focus	Recurring nightmare, disappearing with onset of manifest epilepsy, later becoming hallucinatory aura
Boller et al. (1975)	65y M	R. temp. (thrombosis)	Frequent nightmares with enactment; controlled by anticonvulsant medication
Epstein (1979)	Patient 2, 27y M	R. temp. EEG focus	Recurring nightmares, with same content as compulsive waking thoughts and aura
	Patient 3, 35y F	Bilat. temp. EEG foci; hemangiomatosis	Recurring nightmare, sometimes being or culminating in nocturnal seizure; associated with spiking during REM sleep
	Patient 4, 27y F	L. temp. EEG focus	Recurring nightmare, sometimes culminating in nocturnal seizure; associated with spiking during REM sleep
	Patient 5, 30y F	Bilat. temp. EEG foci	Recurring nightmares, typically being nocturnal seizures; associated with R. temp. occip. spiking during REM sleep
Epstein and Freeman (1981)	38y M	R. temp. (gliosis)	Recurring nightmares, with content related to seizure aura; partially controlled by anticonvulsant medication
Solms (1997)	Case 9, 33y F	R. temp. focus on EEG (idiopathic)	Frequent nightmares with repetitive theme
	Case 18, 32y M	Bilat. paracentr. front. par., R. occip. par., bilat. cingulate g. (open head injury)	Increased reality and frequency with circumscribed fantasy/reality breakdown and repetitive nightmares
	Case 25, 32y F	Bilat. med. frontal/ant. cingulate g. (open head injury)	Increased reality and frequency with circumscribed fantasy/reality breakdown and repetitive nightmares
	Case 27, 24y M	Bitemporal foci on EEG (anoxic episode)	Recurring stereotypical nightmare incorporating epileptogenic hallucinatory imagery
	Case 28, 25y F	Bitemporal foci on EEG, L.>R. (closed head injury)	Recurring stereotypical nightmare
	Case 29, 47y F	L. ant. basal temp. (oligodendroglioma)	Recurring incorporation of epileptogenic olfactory image
	Case 30, 12y F	Nonspecific ABN on EEG (closed head injury)	Recurring nightmare incorporating epileptogenic hallucinatory imagery and aura

Continued

Table 34.6

Continued

Source	Case	Lesion/focus	Dreams
Solms (1997)	Case 31, 46y M	R. lat. front. temp. (aneurysmal rupture)	Recurring stereotypical dream
	Case 32, 50y M	L. deep par. temp. (glioblastoma multiforme)	Recurring stereotypical nightmare (with increased frequency and vivacity)

EEG, electroencephalogram; REM, rapid eye movment; ABN, abnormality.

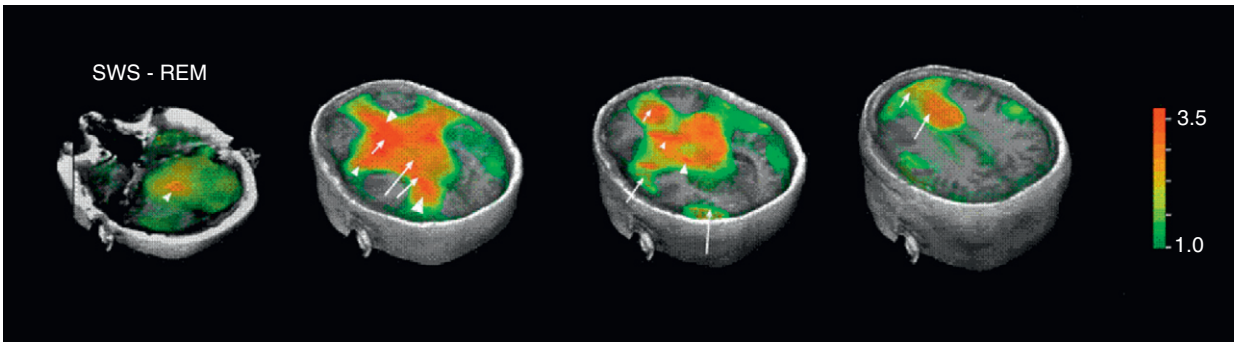


Fig. 34.4. Regional cerebral blood flow in rapid eye movement (REM) sleep. SWS, slow-wave sleep. (Reproduced from Braun et al. (1997).)

The chemical signature of the REM state, as regards the neurotransmitter interactions underlying the observed regional patterns of forebrain activation and deactivation, is certainly more complex than was previously assumed (Gottesmann, 2004).

This complexity is underscored by the impenetrable thicket of psychopharmacological evidence. Of particular value is any evidence that could clarify the pathophysiology of dream cessation following deep ventromesial frontal lesions. Since the sleep cycle is unaffected by such lesions (Jus et al., 1973) it is reasonable to assume that they impair a mechanism which is specific to dream generation (as opposed to REM generation). Two competing hypotheses have been advanced to account for dream cessation following deep ventromesial frontal lesions (and commensurate activation of this region in PET imaging of dreaming sleep). The first hypothesis is that it reflects activation of ACh cells in the basal forebrain; the second is that it reflects activation of DA cells in the ventral tegmental area.

Against the former hypothesis is the observation that ACh antagonists (like scopolamine), rather than suppressing dreaming and dream-like thinking, have the opposite effect (Cartwright, 1966). In fact, anticholinergic drugs mirror the effects of lesions in ACh basal forebrain nuclei (Damasio et al., 1985; Table 34.5). These and other considerations led Braun (1999) to observe that activation of these nuclei during REM sleep may actually reflect inhibition of forebrain ACh in that state.

In favor of the latter hypothesis is the observation that DA agonists (like L-dopa) increase dream bizarreness, vivacity, complexity, and emotionality without having any commensurate effects on REM sleep (Hartmann et al., 1980). Systematic studies of the effects on dreaming of DA antagonists have not yet been performed. However, a preliminary study by Yu (2007) of the effects on dreaming of antipsychotic medications recently found significant dream-suppressing effects.

The available pharmacological evidence therefore supports the view that cessation of dreaming following ventromesial frontal lesions reflects interruption of DA rather than ACh pathways in this region (Solms, 2002).

THEORETICAL CONSIDERATIONS

Necessary and sufficient conditions for dreaming

The well-established fact that 25% of REM-like dreams occur during non-REM sleep (mainly, but not exclusively, at sleep onset and in the late morning), together

with the clinical evidence reviewed above, renders untenable the view that the REM state provides necessary and sufficient conditions for dream generation. The only way in which the old view could be salvaged would be to argue with Nielsen (2000) that non-REM dreams are somehow generated by "covert" REM states. This argument has limited empirical support at best (Bosinelli and Cicogna, 2000; Ogilvie et al., 2000; Perry and Piggott, 2000; Porte, 2000; Vogel, 2000; Takenchi et al., 2001; Suzuki et al., 2004), and is difficult to reconcile with the available clinicoanatomical evidence.

More plausible is the view that REM activation facilitates rather than causes dreaming. This allows for the empirical fact that dreaming is also facilitated by other states, such as sleep onset (descending stages 1 and 2), late-morning arousal (which is hormonally driven), epileptogenic activity (i.e., local forebrain events) and various toxic/metabolic influences (which do not have concomitant effects on REM sleep). The common denominator in all these states is relative cerebral activation. Although cerebral activation may be considered a necessary condition for dream generation (indeed, it is a necessary condition for any conscious state), it is not sufficient to generate the unique state of consciousness known as dreaming. This fact is demonstrated by the simple observation that patients with deep ventromesial frontal lesions do not dream despite preservation of REM (and sleep-onset and late-morning) activation. For dreaming to occur, something specific needs to be added to activation, coupled with the known deactivation of dorsolateral prefrontal convexity (which characterizes all sleep stages and is apparently mediated by reduced glutamate, coupled with reduced 5-HT and NA in the REM state), as well as other as yet unknown variables which vary independently of the classical sleep stages. The requisite "something specific" appears to involve the deep ventromesial frontal region. The converging findings reviewed above suggest that it is the relative increase in mesocortical-mesolimbic DA activity during sleep. This formulation takes for granted the view that dreaming also depends upon posterior cortical mechanisms for the generation of mental imagery.

Functional neuroanatomy of dreaming

Although it is therefore not yet possible to provide a definitive account of the functional neuroanatomy of dreaming, the following model may be advanced on the basis of the converging evidence outlined above. The dream process is initiated by a paradoxical conjunction of sleep with relative forebrain activation. From the functional neuroanatomical viewpoint, the first element in this conjunction is characterized above all by cortical deactivation (with special emphasis on dorsolateral frontal deactivation). Under normal conditions, the most regular source of the second element is REM sleep activation. But REM (i.e., mesopontine) activation is by no means the exclusive source of forebrain activation causing dreaming (c.f., sleep onset, the late-morning effect); and even intrinsic forebrain activation is effective (e.g., complex partial seizures).

The above-described paradoxical conjunction, by itself, is necessary but not sufficient to support dreaming. This is demonstrated by the fact that specific forebrain lesions prevent dreaming, despite preservation of normal sleep with REM activation. The available evidence suggests that the additional specific variable which causes dreaming is the activation of certain limbic forebrain structures. The precise delimitation of this final common pathway to dreaming remains controversial, but networks associated with motivational incentive and/or emotional salience are strongly implicated by the clinicoanatomical, functional neuroimaging, and neurochemical evidence.

The dream process concludes with activation of posterior cortical structures associated with perceptual (especially visual) imagery and memory.

Function of dreaming

Taken together, these considerations suggest that dreaming is: (1) a state of consciousness, characterized by (2) reduced constraints and controls on (3) memory and perceptual imagery with (4) motivational incentive and emotional salience. The occurrence of this hallucinatory mental state during normal sleep probably requires no further explanation than that motivated behavior is precluded during sleep. An adaptive function for dreaming has, however, not been empirically demonstrated.

References

Adler A (1944). Disintegration and restoration of optic recognition in visual agnosia: analysis of a case. Arch Neurol Psychiatry 51: 243–259.

Adler A (1950). Course and outcome of visual agnosia. J Nerv Ment Dis 111: 41–51.

Anan'ev B (1960). Psixologija Cuvstvennogo Poznanija [The psychology of learning through experience.] Academy of Pedagogical Science, Moscow.

Aserinsky E, Kleitman N (1953). Regularly occurring periods of eye motility and concurrent phenomena during sleep. Science 118: 273–274.

Aserinsy E, Kleitman N (1955). Two types of ocular motility during sleep. J Appl Physiol 8: 1–10.

Basso A, Bisiach E, Luzzatti C (1980). Loss of mental imagery: a case study. Neuropsychologia 18: 435–442.

Benson DF, Greenberg J (1969). Visual form agnosia: a specific defect in visual discrimination. Arch Neurol 20: 82–89.

Bischof M, Bassetti CL (2004). Total dream loss: a distinct neuropsychological dysfunction after bilateral PCA stroke. Ann Neurol 56: 583–586.

Boller F, Wright D, Cavalieri R et al. (1975). Paroxysmal "nightmares": sequel of a stroke responsive to diphenylhydantoin. Neurology 25: 1026–1028.

Bosinelli M, Cicogna PC (2000). REM and NREM mentation: Nielsen's model once again supports the supremacy of REM. Behav Brain Sci 23: 913–914.

Botez M, Olvier M, Vézina J-L et al. (1985). Defective revisualization: dissociation between cognitive and imagistic thought. Case report and short review of the literature. Cortex 21: 375–389.

Boyle J, Nielsen J (1954). Visual agnosia and loss of recall. Bull Los Angel Neuro Soc 19: 39–42.

Brain R (1950). The cerebral basis of consciousness. Brain 73: 465–479.

Brain R (1954). Loss of visualization. Proc R Soc Med 47: 288–290.

Braun AR (1999). The new neuropsychology of sleep. Neuro-Psychoanalysis 1: 196–201.

Braun AR, Balkin TJ, Wesenten NJ et al. (1997). Regional cerebral blood flow throughout the sleep–wake cycle – an (H_2O)-O-15 PET study. Brain 120: 1173–1197.

Braun AR, Balkin TJ, Wesenten NJ et al. (1998). Dissociated pattern of activity in visual cortices and their projections during human rapid eye movement sleep. Science 279: 91–95.

Brown JW (1972). Aphasia, Apraxia, Agnosia: Clinical and Theoretical Aspects. Thomas, Springfield, IL.

Cartwright R (1966). Dream and drug-induced fantasy behaviour: a comparative study. Arch Gen Psychiatry 15: 7–15.

Cathala H, Laffont F, Siksou M et al. (1983). Sommeil et rêve chez des patients de lesions parietals et frontales. [Sleep and dreams in patients with parietal and frontal lesions.] Rev Neurol (Paris) 139: 497–508.

Charcot J-M (1883). Un cas de suppression brusque et isolée de la vision mentale des signes et des objets (formes et couleurs). [On a case of sudden isolated suppression of the mental vision of signs and objects (forms and colours).] Progr Méd 11: 568–571.

Charcot J-M (1889). Clinical Lectures on Diseases of the Nervous System, vol. 3. (T. Savill, trans.). New Sydenham Society, London. (Original work published 1883.)

Clarke LP (1915). The nature and pathogenesis of epilepsy. New York Medical Journal 101: 522, 567–573, 623–628.

Critchley M (1953). The parietal lobes. Edward Arnold, London.

Damasio H, Damasio A (1989). Lesion Analysis in Neuropsychology. Oxford University Press, New York.

Damasio A, Graff-Radford N, Eslinger P et al. (1985). Amnesia following basal forebrain lesions. Arch Neurol 42: 263–271.

Dement W, Kleitman N (1957a). Cyclic variations in EEG during sleep and their relation to eye movements, bodily motility and dreaming. Electroencephalogr Clin Neurophysio 9: 673–690.

Dement W, Kleitman N (1957b). The relation of eye movements during sleep to dream activity: an objective method for the study of dreaming. J Exp Psychol 53: 89–97.

Doricchi F, Violani C (1992). Dream recall in brain-damaged patients. A contribution to the neuropsychologyof dreaming through a review of the literature. In: J Antrobus, M Bertini (Eds.), The Neuropsychology of Sleep and Dreaming. Lawrence Erlbaum Associates, Hillsdale, NJ, pp. 99–140.

Efron R (1968). What is Perception? Boston Studies in the Philosophy of Science. Basic Books, New York.

Epstein A (1964). Recurrent dreams: their relationship to temporal lobe seizures. Arch Gen Psychiatry 10: 49–54.

Epstein A (1967). Body image alterations during seizures and dreams of epileptics. Arch Neurol 16: 613–619.

Epstein A (1979). Effect of certain cerebral hemispheric diseases on dreaming. Biol Psychiatry 14: 77–93.

Epstein A, Ervin F (1956). Psychodynamic significance of seizure content in psycho-motor epilepsy. Psychosom Med 18: 43–55.

Epstein A, Freeman N (1981). The uncinate focus and dreaming. Epilepsia 22: 603–605.

Epstein A, Hill W (1966). Ictal phenomena during REM sleep of a temporal lobe epileptic. Arch Neurol 15: 367–375.

Epstein A, Simmons N (1983). Aphasia with reported loss of dreaming. Am J Psychiatry 140: 108–109.

Ettlinger G, Warrington E, Zangwill O (1957). A further study of visual-spatial agnosia. Brain 80: 335–361.

Farah M, Levine D, Calvanio D (1988). A case study of mental imagery deficit. Brain Cogn 8: 147–164.

Farrell B (1969). Pat & Roald. Hutchinson, London.

Feinberg I (2000). REM sleep: Desperately seeking isomorphism. Behav Brain Sci 23: 931.

Feldman M (1971). Physiological observations in a chronic case of "locked-in" syndrome. Neurology 21: 459–478.

Frank J (1946). Clinical survey and results of 200 cases of prefrontal leukotomy. J Ment Sci 92: 497–508.

Frank J (1950). Some aspects of lobotomy (prefrontal leukotomy) under psychoanalytic scrutiny. Psychiatry 13: 35–42.

Gallassi R, Morreale A, Montagna P et al. (1992). Fatal familial insomnia: neuropsychological study of a disease with thalamic degeneration. Cortex 28: 175–187.

Gloning K, Sternbach I (1953). Über das Träumen bei zerebralen Herdläsionen. [On dreams with focal cerebral lesions.] Wiener Zeitschrif für die Nervenheilkunde 6: 302–329.

Gottesmann C (2004). Brain inhibitory mechanisms involved in basic and higher integrated sleep processes. Brain Res Rev 45: 230–249.

Grünstein A (1924). Die Erforschung der Träuma als eine Methode der topishen Diagnostik bei Groβhirnerkrankungen. [Investigation of dreams as a method of topical diagnosis in cerebral disease.] Zeitschrift für die gesamte Neurologie & Psychiatrie 93: 416–420.

Habib M, Sirigu A (1987). Pure topographical disorientation: a definition and anatomical basis. Cortex 23: 73–85.

Hartmann E, Russ D, Oldfield M et al. (1980). Dream content: effects of L-DOPA. Sleep Res 9: 153.

Hobson JA (2001). The Dream Drugstore: Chemically Altered States of Consciousness. Bradford, Cambridge, MA.

Hobson JA, McCarley R (1977). The brain as a dream-state generator: an activation-synthesis hypothesis of the dream process. Am J Psychiatry 134: 1335–1368.

Hobson JA, McCarley RW, Wyzinki PW (1975). Sleep cycle oscillation: reciprocal discharge by two brainstem neuronal groups. Science 189: 55–58.

Hobson JA, Lydic R, Baghdoyan H (1986). Evolving concepts of sleep cycle generation: from brain centers to neuronal populations. Behav Brain Sci 9: 371–448.

Hobson JA, Pace-Schott EF, Stickgold R (2000). Consciousness: its vicissitudes in waking and sleep – an integration of recent neurophysiological and neuropsychological evidence. In: M Gazzaniga (Ed.), The New Cognitive Neurosciences. 2nd edn. MIT Press, Cambridge, MA.

Humphrey M, Zangwill O (1951). Cessation of dreaming after brain injury. J Neurol Neurosurg Psychiatry 14: 322–325.

Jakobson R (1973). Towards a linguistic classification of aphasic impairments. In: H Goodglass, S Blumstein (Eds.), Psycholinguistics & Aphasia. Johns Hopkins University Press, Baltimore, pp. 29–47.

Jus A, Jus K, Villeneuve A et al. (1973). Studies on dream recall in chronic schizophrenic patients after prefrontal lobotomy. Biol Psychiatry 6: 275–293.

Kapur S (2003). Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. Am J Psychiatry 160: 13–23.

Kardiner A (1932). The bio-analysis of the epileptic reaction. Psychoanal Q 1: 375–483.

Kerr N, Foulkes D, Jurkovic G (1978). Reported absence of visual dream imagery in a normally sighted subject with Turner's syndrome. Journal of Mental Imagery 2: 247–264.

LaBerge S (2000). Lucid dreaming: evidence and methodology. Behav Brain Sci 23: 962–964.

Lavie P, Pratt H, Scharf B et al. (1984). Localized pontine lesion: nearly total absence of REM sleep. Neurology 34: 118–120.

Lena I, Parrot S, Deschaux O et al. (2005). Variations in extracellular levels of dopamine, noradrenaline, glutamate, and aspartate across the sleep–wake cycle in the medial prefrontal cortex and nucleus accumbens of freely moving rats. J Neurosci Res 81: 891–899.

Lissauer H (1890). Ein Fall von Seelenblindheit nebst einen Beitrage zur Theorie derselben. [A case of mind blindness with a contribution to theory.] Archiv für Psychiatrie 21: 222–270.

Lugaresi E, Medori R, Montagna P et al. (1986). Fatal familial insomnia and dysautonomia with selective degeneration of thalamic nuclei. N Engl J Med 315: 997–1003.

Lyman R, Kwan S, Chao W (1938). Left occipito-parietal tumour with observations on alexia and agraphia in Chinese and in English. Chin Med J 54: 491–516.

Macrae D, Trolle E (1956). The defect of function in visual agnosia. Brain 79: 94–110.

Maquet P (2000). Functional neuroimaging of normal human sleep by positron emission tomography. J Sleep Res 9: 207–231.

Maquet P, Peters J-M, Aerts J et al. (1996). Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. Nature 383: 163–166.

Maquet P, Degueldre C, Delfiore G et al. (1997). Functional neuroanatomy of human slow wave sleep. J Neurosci 17: 2807–2812.

Markand O, Dyken M (1976). Sleep abnormalities in patients with brain stem lesions. Neurology 26: 769–776.

Michel F, Sieroff E (1981). Une approche anatomo-clinique des deficits de l'imagerie oneirique, est-elle possible? [A clinico-anatomical approach to dream imagery deficits: Is it possible?] In: Sleep: Proceedings of an International Colloquium, Carlo Erba Farmitalia, Milan.

Miller JD, Farber J, Gatz P et al. (1983). Activity of mesencephalic dopamine and non-dopamine neurons across stages of sleep and waking in the rat. Brain Res 273: 133–141.

Morris M, Bowers D, Chatterjee A et al. (1992). Amnesia following a discrete basal forebrain lesion. Brain 115: 1827–1847.

Moss CS (1972). Recovery with Aphasia: The Aftermath of My Stroke. University of Illinois Press, Urbana.

Müller F (1892). Ein Beitrag zur Kenntniss der Seelenblindheit. [A contribution to the knowledge of mind-blindness.] Arch Psychiatr Nervenkr 24: 856–917.

Munk H (1878). Weitere Mittheilungen zur Physiologie des Grosshirnrinde. [Further contribution to the physiology of the cerebral cortex.] Archiv für Anatomie und Physiologie 2: 161–178.

Murri L, Arena R, Siciliano G et al. (1984). Dream recall in patients with focal cerebral lesions. Arch Neurol 41: 183–185.

Murri L, Massetani R, Siciliano G et al. (1985). Dream recall after sleep interruption in brain-injured patients. Sleep 8: 356–362.

Naville F, Brantmay H (1935). Contribution à l'étude des équivalents épileptiques chez les enfants. [Contribution to the study of epilepsy in infancy.] Archives Suisses de Neurologie et de Psychiatrie 35: 92–122.

Neal P, Deneut R (1988). As I Am. Century, London.

Nielsen J (1946). Agnosia, Apraxia, Aphasia: Their Value in Cerebral Localization. 2nd edn. Hoeber, New York.

Nielsen J (1955). Occipital lobes, dreams and psychosis. J Nerv Ment Dis 121: 50–52.

Nielsen TA (2000). A review of mentation in REM and NREM sleep: "covert" REM sleep as a possible reconciliation of two opposing models. Behav Brain Sci 23: 851–866.

Nofzinger EA, Mintum MA, Wiseman MB et al. (1997). Forebrain activation in REM sleep. An FDG PET study. Brain Res 770: 192–201.

Ogilvie RD, Takeuchi T, Murphy TI (2000). Expanding Nielsen's covert REM model, questioning Solm's approach to dreaming and REM sleep, and re-interpreting the Vertes & Eastman view of REM sleep and memory. Behav Brain Sci 23: 981–983.

Osorio L, Daroff R (1980). Absence of REM and altered NREM sleep in patients with spino-cerebellar degeneration and slow saccades. Ann Neurol 7: 277–280.

Ostow M (1954). Psychodynamic disturbances in patients with temporal lobe disorder. J Mt Sinai Hosp 20: 293–308.

Pace-Schott EF (2003). Postscript: recent findings on the neurobiology of sleep and dreaming. In: EF Pace-Schott, M Solms, M Blagrove et al. (Eds.), Sleep and Dreaming. University Press, Cambridge, pp. 335–350.

Pace-Schott EF, Hobson JA (1998). Review of Mark Solms (1997), The Neuropsychology of Dreams: A Clinico-Anatomical Study. Trends Cogn Sci 2: 199–200.

Partridge M (1950). Pre-Frontal Leukotomy: A Survey of 300 Cases Personally Followed for 1½–3 Years. Blackwell, Oxford.

Peña-Casanova J, Roig-Rovira T, Bermudez A et al. (1985). Optic aphasia, optic apraxia, and loss of dreaming. Brain Lang 26: 63–71.

Penfield W (1938). The cerebral cortex in man: I. The cerebral cortex and consciousness. Arch Neurol Psychiatry 40: 417–442.

Penfield W, Erickson T (1941). Epilepsy and Cerebral Localization. Thomas, Springfield, IL.

Penfield W, Rasmussen T (1955). The Cerebral Cortex of Man. MacMillan, New York.

Perry EK, Piggott MA (2000). Neurotransmitter mechanisms of dreaming: implications of modulatory systems based on dream intensity. BehavBrain Sci 23: 990–992.

Piehler R (1950). Über das Traumleben leukotomierter (Vorläufige Mitteilung). [On the dream-life of the leukotomized (preliminary communication).] Nervenärzt 21: 517–521.

Porte HS (2000). Neural constraints on cognition in sleep. Behav Brain Sci 23: 994–995.

Pötzl O (1928). Die Aphasielehre vom Standpunkt der klinischen Psychiatrie, I: Die Optisch-Agnostischen Storungen (die verschiedenen Formen der Seelenblindheit). [The aphasia doctrine from the standpoint of clinical psychiatry, I: Optic-agnosic disorders (the different forms of mind-blindness).] Deuticke, Leipzig.

Poza J, Marti J (2006). Total dream loss secondary to left temporo-occipital brain injury. Neurologia 21: 152–154.

Ritchie D (1959). Stroke: A Diary of Recovery. Faber & Faber, London.

Rodin E, Mulder D, Faucett R et al. (1955). Psychologic factors in convulsive disorders of focal origin. Arch Neurol 74: 365–374.

Sacks O (1985). The Man Who Mistook His Wife for a Hat. Duckworth, London.

Sacks O (1991). Neurological dreams. MD February: 29–32.

Sacks O (1995). An Anthropologist on Mars. Picador, London.

Sacks O, Wasserman R (1987). The case of the colorblind painter. New York Review of Books 34 (18): 25–34.

Schanfald D, Pearlman C, Greenberg R (1985). The capacity of stroke patients to report dreams. Cortex 21: 237–247.

Schindler R (1953). Das Traumleben der Leukomierten. [The dream-life of the leukotomized.] Wiener Zeitschrift für die Nervenheilkunde 6: 330.

Snyder H (1958). Epileptic equivalents in children. Pediatrics 18: 308–318.

Solms M (1991). Anoneira and the Neuropsychology of Dreams. Doctoral dissertation, University of the Witwatersrand, Johannesburg.

Solms M (1997). The Neuropsychology of Dreams. A Clinico-Anatomical Study. Erlbaum, New Jersey.

Solms M (2000). Dreaming and REM sleep are controlled by different brain mechanisms. Behav Brain Sci 23: 843–850.

Solms M (2002). The neurochemistry of dreaming: cholinergic and dopaminergic hypotheses. In: E Perry, H Ashton, A Young (Eds.), The Neurochemistry of Consciousness. Advances in Consciousness Research. John Benjamin's Publishing, Amsterdam, pp. 123–131.

Solms M, Kaplan-Solms K, Brown J (1996). Wilbrand's case of "mind-blindness." In: C Code, C-W Wallesch, Y Joanette et al. (Eds.), Classic Cases in Neuropsychology. Psychology Press, Hove, England, pp. 89–110.

Sparr S, Jay M, Drislane F et al. (1991). A historic case of visual agnosia. Revisited after 40 years. Brain 114: 789–900.

Suzuki H, Uchiyama M, Tagaya H et al. (2004). Dreaming during non-rapid eye movement sleep in the absence of prior rapid eye movement sleep. Sleep 27: 1486–1490.

Takenchi T, Miyasita A, Inugami M et al. (2001). Intrinsic dreams are not produced without REM sleep mechanisms: evidence through elicitation of sleep onset REM periods. J Sleep Res 10: 43–52.

Torda C (1969). Dreams of subjects with loss of memory for recent years. Psychophysiology 6: 358–365.

Trulson ME, Preussler DW (1984). Dopamine-containing ventral tegmental area neurons in freely moving cats: activity during the sleep–waking cycle and effects of stress. Exp Neurol 83: 367–377.

Tzavaras A (1967). Contribution á l'étude de l'agnosie des physiognomies. [Contribution to the study of agnosia for faces.] Unpublished doctoral dissertation, Faculté de Médecine de l' Université de Paris, Paris.

Vogel GW (2000). Critique of current dream theories. Behav Brain Sci 23: 1014–1016.

Walsh K (1994). Neuropsychology: A Clinical Approach. 3rd edn. Churchill Livingstone, Edinburgh.

Wapner W, Judd T, Gardener H (1978). Visual agnosia in an artist. Cortex 14: 343–364.

Whitty C, Lewin W (1957). Vivid day-dreaming: an usual form of confusion following anterior cingulectomy. Brain 80: 72–76.

Wilbrand H (1887). Die Seelenblindheit als Herderscheinung und ihre Beziehung zur Alexie und Agraphie. [Mind-blindness as a focal symptom and its relationship to alexia and agraphia.] Bergmann, Wiesbaden.

Wilbrand H (1892). Ein Fall von Seelenblindheit und Hemianopsie mit Sectionsbefund. [A case of mind-blindness

and hemianopia with autopsy results.] Deutsche Zeitschrift fur die Nervenheilkunde 2: 361–387.

Yu C (2001a). Neuroanatomical correlates of dreaming: the supramarginal gyrus controversy (dream work). Neuro-Psychoanalysis 30: 47–59.

Yu C (2001b). Neuroanatomical correlates of dreaming. II: The ventromesial frontal lesion controversy (dream investigation). Neuro-Psychoanalysis 30: 193–201.

Yu C (2007). Brain Mechanisms of Dreaming. Doctoral dissertation. University of Cape town, Cape town.

Zinkin N (1959). Psixologiceskaja Nauka vSSSR. [Psychological science in the USSR.] Academy of Pedagogical Science, Moscow.