Neurobiology of conscious and unconscious processes during waking and sleep

Abstract

Waking mind functioning comprises conscious and unconscious processes, with the latter being experimentally demonstrated by parapraxes and recent findings showing the active suppression of unwanted memories. According to psychoanalytic theory, these repression phenomena involve a censorship process. Today, neurobiological results show that this process seems to occur during waking rather than during the dreaming sleep stage.

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The study of the differences in mental functioning between the sleep and waking stages, and the identification of neurobiological bases for each stage, has provided crucial knowledge regarding the support of consciousness and has partially opened doors onto the neurobiological basis of unconscious processes. Kandel (1999) distinguishes between three kinds of unconscious processes: (1) repressed or dynamic ones, that is, those described in psychoanalysis; (2) procedural unconscious ones, which are not repressed but rather remain out of awareness and which concern habits, perceptual and motor skills; and (3) preconscious-unconscious ones, which "refer to almost all mental activities, to most thoughts and all memories that enter consciousness." This short paper will be mainly devoted to the neurobiological basis of the conscious and repressed unconscious processes.

Waking Consciousness

Waking is characterized by vigilance. This physiological property supports the ability to focus attention (psychological property) and to interpret environmental stimuli (perception categorization). Waking mentation is characterized by both explicit and implicit activities (Lotstra, 2007). Among the latter are all the unconscious and preconscious processes underlying behavior as considered by psychoanalysis. Psychoanalysis posits that through the action of several defense mechanisms, unwanted memories of events, wishes, and drives are actively prevented from invading the consciousness in order to exclude unpleasant feelings that could disturb adaptation to the environment. Today, such suppression ("repression"(Freud,1900)) of unwanted memories is demonstrated by think/no think paradigms (Anderson & Green, 2001; Anderson, Ochsner, Kuhl, Cooper, Robertson, Gabrielli

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et al., 2004; Depue, Banich, & Curran, 2006; Depue, Curran, & Banich, 2007), this phenomenon being reinforced by sleep (Rauchs, Feyers, Maquet, & Collette, 2008) and reduced in older people (Levy & Anderson, 2008) These results confirm old findings showing that unconscious thoughts can incidentally burst in during waking through parapraxes like forgetting, slips of the tongue, misreading, slips of the pen, bungled actions, and mislaying (Freud, 1901).

Neurobiological Basis of Conscious and Unconscious Waking Processes

The disruption of vigilance leads to pathological states like confusion and coma. Usually, numerous neurobiological processes intervene to support normal waking mentation. The first identified criterion for waking mentation was provided by electroencephalography (EEG). Indeed, Hans Berger and Loomis's team (Berger, 1909; Loomis, Harvey, & Hobart, 1938) were able to distinguish waking from sleep cortical activities; these differences were subsequently confirmed by animal research (Bremer, 1936; Moruzzi & Magoun, 1949). It was shown soon thereafter that the low voltage EEG activity seen during waking corresponds to cortical activation (Hubel, 1959; Steriade, 1996). This approach, involving spontaneous cerebral activity, was more recently enriched by the discovery of the synchronized gamma rhythm, which is observed both in animals (Bouyer, Montaron, & Rougeul, 1981; Ferster, 1988) during attentive behavior as well as in humans, where it is decreased in Alzheimer disease (Ribary, Ionnides, Singh, Hasson, Bolton, Lado et al., 1991). Finally, blood flow level, which is studied by neuroimaging techniques and which is a criterion for cerebral activation, was shown to be high in the forebrain during waking (Braun, Balkin, Wesensten, Carson, Varga, Baldwin et al., 1997; Maquet, Peters, Aerts, Delfiore, Degueldre, & Luxen, 1996; Nofzinger, Mintun, Wiseman, & Kupfer, 1997).

That the forebrain, and mainly the cortex, must be activated for the appearance of mentation is obviously essential. However, the discovery of inhibitory processes in the forebrain represented a major finding. Although already identified during the 19th century at the cortical level via suppression of behavioral criteria (Bubnoff & Heidenhain, 1881), and more recently confirmed by neuron recordings (Creutzfeldt, Baumgartner, & Schoen, 1956) and gamma aminobutyric acid (GABA) application (Krnjevic, Randic, & Straughan, 1966), two studies provided significant results on this topic with respect to sleep-waking processes. First, it was shown that the pyramidal cells of monkeys fire regularly and at a high rate during waking, but fire more slowly and irregularly after sleep onset (Evarts, 1964); the author hypothesized that an inhibitory control process that regulates waking discharges is decreased during sleep. The second study was carried out by a Romanian group which, by analyzing thalamocortical responsiveness in cats, showed that both activating and inhibitory influences act at the cortical level during waking (Demetrescu, Demetrescu, & Iosif, 1966).

Recently, in human studies, the prepulse inhibition experimental paradigm (the modern appellation for the recovery cycle of responsiveness studied by Demetrescu , Demetrescu and Iosif (1966)) showed that the response to the second (test) stimulus was decreased in normal subjects but not in schizophrenics, indicating the presence of abnormal disinhibitory processes in this disease (Kisley, Olincy A., Robbins E., Polk S.D., Adler L.E., Waldo M.C. et al., 2003). These inhibitory influences, acting at the cortical level, were later confirmed by neurochemical findings. Indeed, brainstem noradrenergic and serotonergic

neurons, which show their highest activity during waking (Aston-Jones & Bloom, 1981; Hobson, McCarley, & Wyzinski, 1975; McGinty & Harper, 1976; McGinty, Harper, & Fairbanks, 1974; Rasmussen, Heym, & Jacobs, 1984), give rise to cortical terminals, and both neuromodulators (so-called because of mainly diffuse release at the varicose instead of the synaptic level) inhibit most cortical neurons (Araneda & Andrade, 1991; Foote, Freedman, & Oliver, 1975; Krnjevic & Phillis, 1963; Reader, Ferron, Descarries, & Jasper, 1979). This is also the case, at least partly, with dopaminergic (Giulledge & Stuart, 2005; Krnjevic & Phillis, 1963; Levy, Reves, & Aoki, 2006; Nelson, Hoffer, Chu, & Bloom, 1973; Otani, Blond, Desce, & Crepel, 1998; Reader, Ferron, Descarries et al., 1979) and cholinergic neuron terminals (Giulledge & Stuart, 2005; Levy, Reves, & Aoki, 2006; Nelson, Hoffer, Chu et al., 1973). All of these inhibitory influences promote brain performance because they increase the neuronal signal-to-noise ratio of incoming information.

Consequently, waking mentation is regulated by both antagonistic and complementary activating and inhibitory processes acting at the cortical level, with the collaboration of related subcortical structures. This double functioning that regulates conscious mentation and related behavior also supports the control of repressed unconscious processes. Thus, during waking, the cooperation of different neurobiological processes is now beginning to explain conscious, as well as unconscious, mind functioning all the more, since the increase of the brain molecule (CaMKII) has been recently shown to not only disturb immediate memory fixation (Wang, Feng, Wang, Li, Cao & Tsien, 2008) but also inhibits memory retrieval as well (Cao et al., 2008). Still more recently, it was shown in humans that β-noradrenergic antagonists induce the same suppressing effects (Kindt, Soeter, & Verliet, 2009). The prefrontal BA_{10} area is seemingly mainly involved in these processes of repressed access to consciousness (Depue, Curran, & Ganich, 2007).

Sleep

The sleep which follows waking first comprises slow wave sleep, with four EEG stages (I to IV- the latter being the deepest). Beginning after about 90 minutes, three to five rapid eye movement (REM) sleep periods of progressively increasing duration occur each night, with the final one, taking place prior to morning awakening, lasting up to 50 minutes.

Slow Wave Sleep Consciousness

The brain is never psychologically silent. “There is no sleep of mind. Consciousness ignores sleep”(Hervey de Saint Denys, 1867, p. 165). Indeed, although “The first period following sleep onset is almost always free of dreaming” (p. 163), psychological activity is encountered during slow wave sleep. Foulkes (1962) noticed in this mental activity “considerable and consistent qualitative differences” (p. 22) compared to dreaming. When compared to dreaming, the reports are “less often visual and have a higher degree of correspondence with reality” (p. 23). The “thought-like” content of slow wave sleep instead corresponds to Freud’s “secondary process” ((Freud, 1895), p. 324) involving the “reality principle” (Freud, 1911, p. 219) . The content “has more of the secondary process characteristics which are assigned to preconscious mentation than does REM sleep mentation”

(Rechtschaffen, Boger, & Shaikun, 1963, p. 546). Some dreams, however, have been described during slow wave sleep (Bosinelli, 1995; Cavallero, 1992, 2003; Tracy & Tracy, 1974). Although in the 1950s, Dement and Kleitman (1957) suggested that dreams occurring during slow wave sleep didn't really occur then at all, instead they are delayed reports made by the dreamer of dreams occurring in the previous REM sleep period, recent research strongly suggests that dreaming can occur on a neurobiological background of "covert" REM sleep (Nielsen, 2000; Takeuchi, Miyasita, Inugami, & Yamamoto, 2001; Takeuchi, Ogilvie, Ferrelli, Murphy, Yamamoto, & Inugami, 1999). This process can appear outside of the defined REM sleep periods and the fact that atonia can appear in these stages (II to IV) (Werth, Achermann, & Borbely 2002) could indicate that these periods represent covert REM sleep. Further, directly at sleep onset, during stage I, the occurrence of true episodes of dreaming is now established. This clearly confirms Nielsen's theory (2003). Indeed, during this stage there is low voltage EEG, similar to during REM sleep, and "slow pendular swings of the eye balls from one side of the orbit to the other" (Dement, 1964); both of these criteria are also observed during REM sleep. Moreover, "during descending stage I lasting one-half to 5 minutes… there is a regressive content (characteristic of REM sleep mentation) and there is partial loss of reality contact. During descending stage II there is a paradoxical return to nonregressive content and complete loss of contact with external reality. There is a relatively destructuralizated 'ego' (during descending stage I) and a relatively restruturalized ego (during descending stage II)" (Vogel, Goulkes, & Trosman, 1966, pp. 241-242)(for reference see Gottesmann, 2005).

A particular case concerns arousal from night terrors (*parvor nocturnus*), which occur during stage IV delta waves, principally in teenagers (Gastaut & Broughton, 1964). As noted by Kales & Jacobson (1967), the subject "is usually unable to orient himself to his immediate environment or recall any specific dream other than the general feeling of fear" (p. 88). Today, results have confirmed that "the individual is unaware of the fullness of their surroundings and is totally focused in their concern or activity" (Crisp, 1996, p. 599) . Night terrors are "associated with limited or no mental content during the events and this feature is commonly used to distinguish them clinically from true nightmares" (Hartman, Crisp, Sedwick, & Borrow, 2001, p. 246) occurring during REM sleep. In these episodes, which are often associated with sleep-talking and which are characterized by a "terrified scream accompanied by intense autonomic discharge" (Hartman et al., p. 245) , "a history of major psychological trauma exists in only a minority of adult patients"(p. 244). It could be an index of a dissociative defense process for "excluding painful memories from awareness" (p. 248).

Finally, at the junction of ascending stage II (following stage IV) and REM sleep, there is a short electrophysiological stage that displays criteria of stage II (spindles and K complexes) interspersed with a feature of REM sleep (low-voltage EEG without eye movements) (Goldsteinas, Guennoc, & Vidal, 1966; Lairy, 1966; Lairy, Barros-ferreira, & Goldsteinas, 1968; Salzarulo, Vidal, & de Barros-Ferreira, 1968). During this "intermediate phase," even when the individual is behaviorally awakened, it is difficult to establish psychological contact. Finally, verbal reports do not reveal visual content but a "feeling of indefinable discomfort, anxious perplexity and harrowing worry" (Lairy, Barros-Ferreira, & Goldsteinas, 1968, p. 279). The data seem "to point to a reduction in reportable mentation and in efficient cognitive reactivity at the point of transition from non-REM (slow wave sleep) to REM sleep" (Larson & Foulkes, 1969, p. 552).

Neurobiological Basis of Slow Wave Sleep Consciousness

Activating brainstem ascending influences (Moruzzi, 1972) progressively decrease during the deepening of slow wave sleep because of active influences inducing sleep (Gallopin, Fort, Eggermann, Caul, Luppi, Roissier et al., 2000). This can be shown by the progressive increase in the reticular arousal threshold (Piallat & Gottesmann, 1995), the decrease in reticular and cortical neuron firing (Arduini, Berlucchi, & Strata, 1963 ; Evarts, 1964; Steriade, 1996), and the decrease in thalamocortical responsiveness (Demetrescu, Demetrescu, & Iosif, 1966; Gandolfo, Arnaud, & Gottesmann, 1980; Okuma & Fujimori, 1963; Rossi, Palestini, Pisano, & Rosadini, 1965; Steriade, 1970). This lowering of forebrain activation is also evidenced by forebrain blood flow, which "decreases dramatically as a function of δ(delta waves of stages III and IV) and spindle activity, reflecting the disfacilitation and active inhibition of thalamocortical neurons that occur during slow wave sleep and possibly underlie the loss of consciousness and sensory awareness characteristic of that state" (Hofle et al., p. 4806). Interestingly, the visual and secondary auditory cortices maintain some activation. This global cerebral deactivation has also been observed by other authors (Maquet et al., 1997). Finally, acetylcholine, which is preferentially facilitatory, is released at a lower level than it is during waking (Jasper & Tessier, 1971; Marrosu, Portas, Mascia, Casu, Fa, Giagheddu et al., 1995). This continuous decrease in cortical activation is accompanied by a simultaneously and progressive decrease in inhibitory processes. This was first shown by unicellular discharges, which begin to become irregular (Evarts, 1964), and by the recovery cycle of radiation-cortical and thalamocortical evoked potentials which becomes shorter (Allison, 1968; Demetrescu, Demetrescu, & Iosif, 1966; Rossi, Palestini, Pisano et al., 1965). The disinhibitory processes are also evidenced by the decrease in dopaminergic, noradrenergic, and serotonergic neuron firings and the related lowering of cortical release (Cespuglio, Houdouin, Oulerich, El Mansari, & Jouvet, 1992; Léna, Parrot, Deschaux, Muffat, Sauvinet, Renaud et al., 2005).

To conclude, the decrease in both facilitating and inhibitory influences acting at the cortical level, particularly during stage II (which occupies the major part of slow wave sleep at night), is able to explain the maintenance of some mentation of waking quality, that is, following the reality principle but occurring to a lower extent. The further decrease of both of these cortical influences during deep slow wave stage IV sleep could release rudimentary impulsive acting-out like manifestations of night terrors and induce brutal awakening. Finally, the fully unusual, poor, and anguished mental content of the intermediate phase is the understandable consequence of the nearly isolated (disconnected) forebrain (physiological "cerveau isolé" like preparation) (Gottesmann, 1964, 1996, 1999; 2008 ; Gottesmann, Gandolfo, & Zernicki, 1984; Gottesmann, User, & Gioanni, 1980), that is, without the usual brainstem-originating regulatory influences.

REM Sleep Dreaming Sleep Stage

Consciousness

Dreaming is the characteristic mental activity of REM sleep. Dreaming can be considered from two non-exclusive points of view, as already believed by Freud ((Freud, 1900). On the one hand, dreaming is characterized by "sensory hallucinations, bizarre imagery… diminished reflective awareness, orientational instability… intensification of emotion, instinctual behaviors" (Hobson, Stickgold, & Pace-Schott, 1998, p. R2), features strongly reminiscent of schizophrenia. Numerous philosophers, writers, and neuropsychiatrists have underlined the similarity between the two mental activities. Thus, Hughlings Jackson could predict, "Find out all about dreams and you will find out about insanity" (for references see Gottesmann, 2004, 2005, 2006).

On the other hand, the Talmud said, "an uninterpreted dream is like an unread letter "(Fromm, 1953, p. 1) , an idea beautifully developed by Freud in *The Interpretation of Dreams* (1900). For him, dreams have a "safety-wave" function to preserve sleep and are primarily the manifestation of unconscious wish-fulfillments. Such wishes or drive representations , however, cannot enter the dreamer's consciousness in their crude form. The dream content must thus be changed by a disguise-censorship to avoid anxiety which would induce awakening because of psychologically unacceptable feelings. A dream-work process would transform the original latent dream content into more acceptable figurative content that follows the primary pleasure principle, leading to a reduction in emotional tension. The dream-work modification of mental content carried out by the censorship process would act through two mechanisms: first, by "condensation", transforming several ideas or feelings into a unique element or representation. The second is "displacement": "an idea's emphasis, interest or intensity is liable to be detached from it and to pass on to other ideas, which were originally of little intensity but which are related to the first idea by a change of associations" (Laplanche & Pontalis, 2006, p. 121) . An additional important assertion of Freud was that "The forgetting of dreams remains inexplicable unless the power of the psychical censorship is taken into account"(1900, p. 517).

Neurobiological Basis of Dreaming

REM sleep is characterized by strong cortical activation. This was first shown with low voltage rapid cortical field activity, which was observed both in animals (Dement, 1958; Jouvet, Michel, & Courjon, 1959; Klaue, 1937) and humans (Aserinsky & Kleitman, 1953; Loomis, Harvey, & Hobart, 1937). It was further demonstrated by the observation of a high rate of cortical neuron firing (Arduini, Berlucchi, & Strata, 1963; Evarts, 1962, 1964), related to depolarization as shown by a negative steady potential in the cortex (Kawamura & Sawyer, 1964 ; Wurtz, 1965) and high thalamocortical responsiveness (Favale, Loeb, & Manfredi, 1963 ; Okuma & Fujimori, 1963 ; Rossi, Palestini, Pisano et al., 1965). More recent findings have confirmed strong cortical (and more generally forebrain) activation, as evidenced by increased blood flow (Braun, Balkin, Wesensten et al., 1997 ; Madsen, Holm, Vorstrup, Friberg, Lassen, & Wildschiodtz, 1991; Maquet, Peters, Aerts et al., 1996; Maquet, Ruby, Schw artz, Laurey, Albouy, Dang-Vu et al., 2004; Nofzinger, Mintun, Wiseman et al., 1997).

This cortical activation increases during the eye movement bursts (concomitant to ponto-geniculo-occipital, or PGO, waves), as shown by different criteria (Hong, Gillin, Dow, Wu, & Buschbaum, 1995; Kiyono & Iwama, 1965; Satoh, 1971). Consequently, in this activated forebrain it is understandable that there is (generally vivid) mental activity.

There is, however, major forebrain dysfunction during REM sleep, which can explain the abnormal mental activity encountered in this sleep stage. First, there is a decrease in the release of dopamine, noradrenaline, serotonin, and acetylcholine in the cortex (Cespuglio, Houdouin, Oulerich et al., 1992; Léna, Parrot, Deschaux et al., 2005; Marrosu, Portas, Mascia et al., 1995), and disturbances in the function of these four neuromodulators has been described as favoring schizophrenic symptoms (Abi-Dargham & Moore, 2003; Cartwright, 1966; Collerton, Perry, & McKeith, 2005 ; Friedman, Adler, & Davis, 1999; Huang, Li, Ichikawa, Dai, & Meltzer, 2006 ; Linner, Wiker, Wadenberg, Schalling, & Svensson, 2002; Okubo, Suhara, Kobahashi, Inoue, Terasaki, Someya et al., 1997; Silver, Barash, Aharon, Kaplan, & Poyurovsky, 2000; Van Hes, Smid, Stroomer, Tipker, Tulp, Van der Heyden et al., 2003). Another major index of neurochemical disturbance is provided by the neurochemical functioning of the nucleus accumbens. Indeed, dopamine is maximally released during REM sleep in this structure (Léna, Parrot, Deschaux et al., 2005); this has long been considered a characteristic of schizophrenia (MacKay, Iversen, Rossor, Spokes, Bird, Arregui et al., 1982). In addition, the level of glutamate release is the lowest of all the sleep-waking stages (Léna, Parrot, Deschaux et al., 2005), another criterion that is now considered to be the main characteristic of schizophrenia (Grace, 2000; Heresco-Levy, 2000). It is of interest that, as shown by psychotropic compounds, both the increase in dopamine and the decrease in glutamate induce not only psychotic symptoms but vivid dreaming (Larsen & Tandberg, 2001; Perry & Piggott, 2003; Reeves, Lindholm, Myles, Fletcher, & Hunt, 2001). Finally, whereas glutamate functioning is reduced in the nucleus accumbens in both schizophrenia and during dreaming, it is worth mentioning that glutamate function at the cortical level is unchanged in both states (Lauriat, Dracheva, Chin, Schmeidler, McInnes, & Haroutunian, 2005; Léna, Parrot, Deschaux et al., 2005).

The major cortical disinhibition revealed by neurochemistry has been reinforced by electrophysiological findings. Indeed, decades-old results already showed these crucial phenomena. First, the cortical pyramidal neurons show high frequency firing, but contrary to during waking, the firing occurs by intense bursts separated by long silences; this pattern reflects the suppression of inhibitory control processes that normally induce a regular discharge pattern (Evarts, 1964). Moreover, the recovery cycle of cortical responsiveness in animals (Allison, 1968 ; Demetrescu, Demetrescu, & Iosif, 1966; Rossi, Palestini, Pisano et al., 1965) and humans (Kisley, Olincy A., Robbins E. et al., 2003) is strongly reduced during REM sleep, indicating a reduction in the relative refractory process and thus a disinhibition. It is of the highest interest that the same cortical disinhibition is present during REM sleep and in schizophrenia (Kisley, Olincy A., Robbins E. et al., 2003). Another electrophysiological criterion of cortical dysfunction during REM sleep is the disturbance of the gamma rhythm. Although this rhythm is observed during both REM sleep and waking (Llinas & Ribary, 1993), its synchronization over cortical areas is lost during REM sleep (Corsi-Cabrera, Miro, del Rio Portilla, Perez-Garci, Villanueva, & Guevera, 2003; Perez-Garci, del Rio-Portilla, Guevara, Arce, & Corsi-cabrera, 2001); this is also the case for the hippocampo-cortical (Cantero, Atienza, Madsen, & Stickgold, 2004 ; Massimini, Ferrarelli, Huber, Esser, Singh, & Tononi, 2005) and intra-hippocampal relationships (Montgomery, Sirota, & Buzsaki, 2008). These intracerebral disconnections are well known indications of schizophrenia (Kubicki,

Styner, Gerig, Markant, Smith, MacCarley et al., 2008; Meyer-Lindenberg, Olsen, Kohn, Brown, Egan, Weinberber et al., 2005; Meyer-Lindenberg, Poline, Kohn, Holt, Egan, Weinberger et al., 2001; Peled, Geva, Kremen, Blankfeld, Esfandiarfard, & Nordahl, 2000; Tononi & Edelman, 2000; Young, Beach, Falkai, & Honer, 1998).

Finally, two last cortical criteria of REM sleep are also indexes of functional disturbances related to schizophrenia. First, there is a deactivation of the dorsolateral prefrontal cortex. This phylogenetically most recently appearing structure is involved in the highest integrated cognitive processes. In schizophrenics, in addition to the local deficit of dopamine (Abi-Dargham & Moore, 2003; Léna, Parrot, Deschaux et al., 2005), frontal glucose uptake is decreased (Buschbaum, Ingvar, Kessler, Waters, Cappelletti, Van Kammen et al., 1982), the dorsolateral prefrontal deactivation being particularly evident when the higher cognitive functions such as reflectiveness and working memory are disturbed (Berman, Doran, Pickar, & Weinberger, 1993 ; Weinberger, Berman, & Zec, 1986). It is of interest to mention that the posterior cingular cortex is also deactivated during REM sleep: When piano concertists are so involved in their playing that they "lose themselves," and are thus somewhat disconnected from the environment, there is a conjugate deactivation of both the dorsolateral prefrontal cortex and the posterior cingulate cortex (Parsons, Sergent, Hodges, & Fox, 2005). This deactivation associated with monoaminergic disinhibition is not only an index of a loss of function in the cortex, but also contributes to the nucleus accumbens-related disturbances, at least dopaminergic and glutamatergic, of REM sleep and schizophrenia (Brake, Flores, Francis, Meaney, Srivastava, & Gratton, 2000; Grace, 2000; Jackson, Frost, & Moghaddam, 2001).

The second criteria is the specific deactivation of the primary visual cortex (Braun, Balkin, Wesensten, Gwardry, Carson, Varga et al., 1998); the extrastriate visual cortex, in contrast, is strongly activated (Braun, Balkin, Wesensten et al., 1998; Madsen, Holm, Vorstrup et al., 1991). This deactivation (although today questioned (Hong, Harris, Pearlson, Kim, Calhoun, Fallon et al., 2008) leads to a cortical restriction in the central processing of sensory afferent information. It is worth mentioning that, at the thalamic level, sensory afferent transmission is also blocked. Indeed, although postsynaptic responsiveness is increased, there is presynaptic depolarization, corresponding to inhibition, during the eye movement bursts during which the majority of dreams occur (Dagnino, Favale, Loeb, Manfredi, & Seitun, 1969; Gandolfo, Arnaud, & Gottesmann, 1980 ; Ghelarducci, Pisa, & Pompeiano, 1970; Iwama, Kawamoto, Sakkakura, & Kasamatsu, 1966; Steriade, 1970). Another argument for an increase in sensory gating control during REM sleep is that, contrary to during waking, external stimuli do not reset the gamma rhythm (Llinas & Ribary, 1993). All of these indices of decreases in sensory constraints favor the appearance of the hallucinatory activity typical of schizophrenia (Behrendt & Young, 2005). Moreover, a parallel can be drawn between this sensory deafferentation occurring during REM sleep and the analgesia observed during psychotic outbursts (Griffin & Tyrrell, 2003). Finally, also related to the integration of sensory afferents, at the end of REM sleep dreaming there is a lack of differentiation between self- and external stimulation (tickle), as is observed in schizophrenia (Blagrove, Blakemore, & Thayer, 2006)

Another issue which must be highlighted is the process that leads to the forgetting of dreams. Three main hypotheses have been offered for this well-known phenomenon. The first was proposed by Freud. According to him, the forgetting (see above) is the result of

repression by the censorship process. Its mechanism would be like the obliteration of writing observed in the children's game "mystic writing-pad"(Freud, 1925). The second, most frequently encountered hypothesis is that it prevents overloading of waking memory with, at the very least, useless mental content (Crick & Michison, 1983). The third hypothesis postulates a mental safety process: If dream content entered waking consciousness, then it could be taken as reality and induce schizophrenia (Kelly, 1998).

The forgetting of dreams upon arousal is either complete or progresses rapidly. Instead of a psychological censorship as postulated by Freud (1900), it is reasonable to presume that forebrain functioning is so disturbed during REM sleep that it is unlikely that an unconscious "super-ego" could control what should be repressed. We hypothesized a physiological censorship (Gottesmann, 2006; Gottesmann & Gottesman, 2007). Indeed, it is known that the noradrenergic neurons of the locus coeruleus begin to discharge again a few seconds before behavioral arousal (Aston-Jones & Bloom, 1981). A similar phenomenon could occur with dopaminergic, serotonergic and cholinergic neurons. Such a recovery of waking brain functioning processes prior to behavioral arousal could prevent dreams from being recorded into waking memory. Three other processes could also promote dream forgetting. First, at arousal, the recovery of prefrontal cortex efficiency occurs later than it does in other cortical areas (Balkin, Braun, Wesensten, Jeffries, Varga, Baldwin et al., 2002). Considering its crucial cognitive functions, this delayed recovery could contribute to the forgetting. Second, the release of cortisol in the early morning, near the occurrence of the longest REM sleep period, has long been known to have a negative influence on memorization (Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Payne & Nadel, 2004). Finally, it could be due to an endogenous increase in CaMKII synthesis at arousal. Indeed, this biochemical agent , once again, inhibits memory fixation (Wang, Feng, Wang, Li, Cao, & Tsien, 2008), and erases unwanted memories (Cao, Wang, Mei, An, Yin, Wang et al., 2008). Another possibility could be a transient β noradrenergic receptor desensitization (Kindt, Soeter, & Verliet, 2009).

Conclusion

The known neurobiological activities of the forebrain during waking are beginning to extensively explain the mechanisms underlying psychological activities. The antagonistic but complementary activating and inhibitory influences clearly provide the basis of the regulation of conscious as well as unconscious processes. This includes not only procedural unconscious processes (see Kandel, 1999) but repressed ones as well. Nowadays, experimental results confirm the intervention of defense-like mechanisms as hypothesized by Freud which suppress the entry of unwanted memories into waking consciousness. This is a major finding supporting the validity of some assertions of psychoanalysis.

During the different stages of slow wave sleep, the progressive and concomitant decrease of both kinds of influence is also beginning to explain the various levels of consciousness. During REM sleep, the current, more precise knowledge of the heavy neurobiological disturbances that take place in forebrain functioning can explain the psychotic-like mental functioning. This major disruption of function does not support the existence of Freud's advanced censorship process that would be able to transform unacceptable mental content into less emotional, acceptable figurative representations. It

seems that there is "more censorship in the waking analysis of dreams than during the dream itself" (Blechner, 2006, p. 17). For the same reasons, dream forgetting is better explained by neurobiological factors than by the intervention of a censorship mechanism.

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