Revisión

Téllez-López Arnoldo,
Sánchez-Jáuregui Teresa [1,2]

[1]*Centro de Investigación y Desarrollo en Ciencias de la Salud, Universidad Autónoma de Nuevo León*
[2] *Facultad de Psicología, Universidad Autónoma de Nuevo León*

Neurociencia del soñar

Neuroscience of dreaming

Resumen

Tradicionalmente la neuropsicología se ha enfocado a la identificación de los mecanismos cerebrales de determinados procesos psicológicos tales como la atención, el movimiento, la percepción, la memoria, el lenguaje, la conciencia, así como sus respectivas alteraciones. Sin embargo hay procesos psicológicos que han recibido poca atención dentro de este campo, entre ellos está el soñar. Este trabajo revisa las investigaciones neuropsicológicos clínicos y experimentales más relevantes en relación al soñar, desde la alteración de los sueños en pacientes con lesión cerebral, hasta la actividad cerebral funcional durante el sueño MOR usando diferentes métodos de imagen. Estos hallazgos son analizados dentro del modelo de bloques cerebrales de Luria y se hace una propuesta para explicar algunas de las características esenciales del soñar. Se describe cómo durante el soñar se produce una activación del bloque 1 que comprende la formación reticular del tallo cerebral, la activación del bloque 2, formado por los lóbulos parietal, occipital y temporal y el bloque L, formado por el sistema límbico, junto a una inhibición simultánea del bloque 3 es decir la inhibición de la actividad del lóbulo frontal. La actividad anteriormente descrita, produce la percepción de imágenes alucinatorias de diferente modalidad sensorial, así como una desinhibición, un pensamiento no reflexivo, una falta de planeación y dirección de tales imágenes oníricas. Se plantea al soñar como un tipo de confabulación natural, parecida a la que ocurre en pacientes con daño en lóbulo frontal o la esquizofrenia. Además se propone el carácter confabulatorio, bizarro e impulsivo del soñar tiene una función de homeostasis cognitivo-emocional, que ayuda al buen funcionamiento del cerebro durante el día.

Palabras clave.

Bloques funcionales, neuropsicología, soñar, sueño.

Abstract

Traditionally, neuropsychology has been focused on the identification of brain mechanisms of specific psychological processes, such as attention, motor skills, perception, memory, language, and consciousness, as well as their corresponding disorders. However, there are psychological processes that have received little attention in this field, such as dreaming. This work examined the clinical and experimental neuropsychological research that is more relevant to dreaming, ranging from sleep disorders in patients with brain damage to brain functional activity during REM sleep using different methods of images. These findings were analyzed into the frame of Luria's Three Unit Model of Brain functioning, and a proposal was made to explain certain of the essential characteristics of dreaming. This explanation describes how during dreaming an activation of the first unit occurs, comprising the reticular formation of the brainstem, activating, in turn, the Second Unit, which is formed by the parietal, occipital, and temporal lobes and Unit L, which is comprised by the limbic system, as well as a simultaneous hypofunctioning of the Third Unit (frontal lobe). This activity produces a perception of hallucinatory images of various sensory modes, as well as a lack of inhibition, a non-self-reflexive thought process, and a lack of planning and direction of such oneiric images. Dreaming is considered a type of natural confabulation, similar to the one that occurs in patients with frontal lobe damage or schizophrenia. It also proposes that the confabulatory, bizarre, and impulsive nature of dreaming has a function in the cognitive-emotional homeostasis that aids proper brain function throughout the day.

Keywords

Dream, dreaming, functional units, neuropsychology

Correspondencia:
Arnoldo Téllez López, Unidad de Psicología de la Salud (CIDICS). Av. Carlos Canseco s/n esquina con Av. Gonzalitos, Colonia Mitras Centro,
Monterrey, N.L., México. C.P. 64460.
Tel; 01(81)13-40-43-70 ext.1768.
Correo electrónico: atellez50@hotmail.com

Introducción

Since the beginning, neuropsychology has focused on identifying the brain functions of psychological processes known as *"higher processes"*, such as attention, motor skills, perception, memory, language, and conscience, as well as all their corresponding disorders (e.g., inattention, apraxia, agnosia, aphasias). However, there are psychological processes that have received little attention in this field, and among them is the process of dreaming. In the last few decades, interesting neuropsychological findings have started to surface on the relationship between the production and recollection of the oneiric processes. Such research has been examined here. We can start by asking ourselves: Are dreaming or dreams a subject of study for neuropsychology? Although there is not a definition of neuropsychology that has been thoroughly accepted, Alexander R. Luria,[1] considered the founder of modern neuropsychology, defined this field as: *"A new branch of scientific knowledge, which sole particular aim is to investigate the role of specific brain systems, in the complex forms of mental activity."* According to this definition we can suggest the following question: Is dreaming a complex form of mental activity?

To answer this question, it would be of great help to describe the phenomenology of dreaming. Dreaming is a psychophysiological active process that involves the presence of perceptive hallucinatory images during sleep (i.e., the visual, auditory, tactile, kinesthetic, and linguistic kind) and the cognitive activity of emotional content of variable intensity that has been generated internally.[2] The content of the dream is bizarre by nature, defining bizarre as incongruities and discontinuities in time, space and the characters that appear in it.[3] Furthermore, there is a lack of control over the course of the scenes, in which there are often infringements on the laws of physics. All of this is due to a lack of critical thinking that can evaluate the coherence or the lack thereof in what is happening, so there is a passive and uncritical acceptance of everything that happens.[3] Epstein[4] says that *"dream formation involves a complex psychological activity that integrates memory, language and thinking itself."*

If, after this, we assume that reality training is a complex form of mental activity, the next questions would be: Which are the particular brain systems involved in this process? What are the differences between the neuropsychological systems involved in the conscious processes of wakefulness and the ones involved in dreaming?

It is evident that dreams have always interested and captivated humanity since ancient times. It was not until 1900, however, that Freud [5] published his book *"The Interpretation of Dreams"*, which included the first scientific approach to the subject from a purely psychological point of view. The first approach and start of the psychobiological scientific research on the subject of dreaming happened in 1953 when Aserinsky and Kleitman[6] from the University of Chicago published their research, stating that sleep with rapid eye movement, also known as REM sleep, is frequently associated with dream recalling. Since then, we have learned that human sleep is made up of two phases: REM sleep, which is generally associated with dreaming, and non-REM sleep, or sleeping without rapid eye movement, in which very few dreams are recalled. In the REM phase, the eyes move rapidly in all directions, and upon waking up, people frequently report having dreamt. During this phase, there is also an increase of electroencephalographic [7] and cerebral metabolic activity, which is equal to or greater than the activity during wakefulness.[8-11] Penis erection also occurs in males,[14] as well as increases in heart rate.[12,13] All this intense psychophysiological activity is accompanied by muscle atonia,[15] the function of which some authors have mentioned is to avoid the translation of the dream into action.[14] On the other hand, opposite of REM sleep, we find non-REM sleep, which is characterized by a decrease in the psychophysiological activity in general.[16]

Luria´s theory of brain functioning: Three Functional Units Model

As it has been previously mentioned, dreaming is a psychophysiological process as active as wakefulness; however, little is known about the neuropsychological systems implied. Therefore, the objective of this article was to present a proposal of a neuropsychological model of dreaming based on the most relevant clinical, experimental, psychophysiological and neuropsychological research. To present the proposal about the generation and bizarre content of a dream, we took, as a general frame, Luria's Three Functional Units Model,[1] which attempts to explain the neuropsychological functioning of human beings during wakefulness.

A) The First Unit is made up by the structures of the brainstem, specifically, the reticular formation, which functions to activate and keep the general cortical tone needed to produce the activation of the cerebral cortex, generating a state of alertness.[17]

B) The Second Unit is formed by the parietal, occipital and temporal lobes and is responsible for obtaining, processing, integrating, and storing sensory information from the environment.

C) The Third Unit is formed by the frontal lobe, which is in charge of the selection, planning, execution, and direction of the behavior programs, as well as their evaluation.[1,18]
Although Luria does not explicitly mention it, we believe it is convenient to incorporate the limbic system as a Fourth Unit:

D) Unit L, which includes the hippocampus, amygdala, and fornix, comprising the limbic system, as well as paralimbic structures, such as the cingulate gyrus and the parahippocampal and orbitofrontal regions. This Unit is responsible for emotional responses and the consolidation of the memory.[19]

The Three Unit Model during REM Sleep

During REM sleep, there is an activation of the First Unit similar to the one in the state of wakefulness that manifests with an increase of the electroencephalographic and metabolic activity in most regions of the brain. Hobson [20] found that during REM sleep, there is an activation of the brainstem that starts in the cholinergic system on a pontine level. Studies with positron emission computerized tomography (PET) have confirmed an increase in the brainstem's metabolism,8 generating electroencephalographic and metabolic activation, as well as stimulation of the posterior cortical and subcortical areas, especially the limbic-emotional system.

However, the activation of the First Unit during dreaming is not completely the same as in wakefulness because a cortical motor inhibition occurs, producing motor paralysis.[14,15]

The activation of the First Unit is also different from that in wakefulness, as it produces a bigger stimulation of the structures in Unit L that produce emotional activation.[21] This has been confirmed by experimental studies in animals and humans. For example, it has been reported that the stimulation of the cingulate gyrus in humans causes complex hallucinatory phenomena, emotional changes, rapid eye movement, and oneiric sensation. Similar changes are reported following the stimulation of the hippocampus and amygdala.[21] It can be inferred that any variable that increases limbic system activity during dreams can cause differences to occur in emotional intensity, ranging from little emotional content to nightmares. As an example, the cases of patients with areas of epileptogenic activity in the limbic and paralimbic regions (Unit L), as in the case of epilepsy of the temporal lobe, show a higher incidence in recalling their dreams in contrast with patients with generalized tonic-clonic seizures and normal people.[4,10]

As a consequence of the activation of Unit 1, Unit 2 is stimulated, generating activation in visual perceptive-imaginative, auditory, linguistic, spatial and tactile functions.

Studies with PET have found that the visual and auditory secondary areas are especially metabolically active during REM sleep, even above levels found in wakefulness.[8,9] The activation of the visual system is manifested through PGO spikes (bioelectric activity that comes in a synchronized way from the pons, lateral geniculate body, and the occipital cortex) that are associated with rapid eye movement, giving way to dreams with visual predominance.[21, 22] Several studies agree that lesions in the areas involved in visuospatial processing and representation in Unit 2 bring as a consequence a reduction or elimination of dreaming, a neuropsychological syndrome called *"anoneria"*. These disturbances in dreaming are positively correlated with the appearance of some type of agnosia.[23-26] For example, Peña-Casanova et al.[26] reported a case of a patient with a lesion in the left temporo- occipital region due to a cerebrovascular accident. This patient lost the ability to dream and also showed optic aphasia, optic apraxia, aphasia without agraphia, and color agnosia.

In the meantime, the Unit 3, or frontal lobe, simultaneously suffers an inhibition of some of its regions and an activation of others. The frontal lobe can be divided into two regions: the motor region (Brodmann areas 4, 6, and 8) and the non-motor region or prefrontal lobe (Areas 9, 10, 11, 44, 45, 46 and 47). The prefrontal lobe is divided into three regions:
1) The dorso-medial region, which is associated with executing functions like the formulation of goals, working memory, planning, execution, and the self-regulation of behavior; 2) the orbital frontal region, which is related to the inhibition and control of impulses and social tact; and 3) the medial region, which has been related to motivation 18 and the process of thinking what another person is thinking, also known as mentalization, which is a second order process of representation, relevant to social skills.[27] In general, the prefrontal lobe has been associated with selection functions, programming and direction of the behavioral plan, and impulse inhibition, as well as critical and reflexive thought.[1,18,28] *(Figure 1)*

It has been proven through PET and functional magnetic resonance imaging (fMRI) that during dreaming, there is an activation of the primary and supplementary motor areas, such as the frontal ocular area (Brodmann's area 8), which is activated by the Unit1 and then collaborates in the production of the rapid eye movements of REM sleep.[22] The activation of the supplementary motor (Brodmann's area 6) and primary motor area (Brodmann's area 4) produce a programming and activation of a sequence of corporal movements during the oneiric content, except that said activation remains on a representation level, because an inhibition occurs in the caudal region of locus coeruleus located in the pons of the brain stem (Unit 1) due to hyperpolarization of the motoneurons in the spinal cord, which generates a general muscle paralysis (with the exception of ocular movement), preventing the dream from becoming an action.[15,29] Other areas that are activated are the prefrontal medial region and the part that corresponds to the anterior region of the cingulate gyrus.[8,30] These structures have a connection with Unit L.

Moreover, the dorsolateral region of the prefrontal lobe (Brodmann's areas 9, 10, 45, 46, 47) and the orbital frontal region (Brodmann's areas 11 and 12) show an inhibition during dreaming. Madsen[9] using PET found a drop in metabolism in the orbitofrontal region during REM sleep. Using the same technique, Maquet and Jean-Marie[10] found a decrease in the activity of the frontal lobes and an increase in the amygdaloid complex. It is well-established that lesions or dysfunction in this area in neuropsychological patients results in uninhibited, impulsive, and bizarre behavior.

Years later, using PET with radioactive tracers H2 150, which is the most suitable for sleep research, Braun et al.[8] found low metabolism in the orbitofrontal and dorsolateral regions of the prefrontal lobe during REM sleep, as well as in the inferior parietal association, and simultaneously, an increase in metabolism in the visual and auditory association areas of Unit 2. Doricchi and Violani[23] and Murri et al.[25] found that frontal lesions were the only ones that did not affect dreaming, indirectly confirming the previously mentioned PET findings.

This also supports the hypothesis that states that Unit 3 is inactive and is not necessary for the dreaming process.

Other indicators that the prefrontal lobe is hypofunctioning during dreaming come from comparing studies of interhemispheric and intrahemispheric electroencephalographic correlation during wakefulness, REM and non-REM sleep. Corsi-Cabrera et al.[3] found an absence of electroencephalographic correlation between the frontal and perceptual regions, as well as an increased correlation among the perceptual regions. These researchers suggested that this temporary dissociation between the executive and perceptual areas is the cause of the characteristic bizarreness of dreams.

That being said, it can be expected that upon the activation of Unit L and a simultaneous decrease in the functioning of the prefrontal lobe during wakefulness, any person could behave in an uninhibited, impulsive or aggressive way, with difficulties in planning and self-regulation. Such can be the case of people with schizophrenia,[31,32] major depression[33] and 75% of criminals that exhibit low metabolism in the prefrontal lobe during wakefulness.[32,34] During REM sleep in normal people, there is an increase in the activity of Unit L and a decrease in Unit 3; however, we cannot observe the behavioral effects due to the activation of the cerebral mechanisms that produce the muscle paralysis that comes with this type of sleep, preventing the body from acting out dreams. On the contrary, experiments with cats that had the caereleus alfa nucleus damaged, which seems to be responsible for the motor paralysis during REM sleep, has caused these animals to translate their *"dreams"* into behavior, which is generally manifested in rapid behavioral sequences of, for instance, attack, rage, and grooming. These behaviors are not directed towards an objective, because when a piece of meat or a mouse is placed near them, they do not seem to notice them, and they continue with their stereotypical behavior. Furthermore, the pupils display miosis and are covered by the nictitating membranes the same way it happens during REM sleep. This phenomenon has been called *"oneiric behavior"*.[29] Jouvet et al.[29] make an interesting observation about animal behavior: *"Some cats that exhibited friendly behavior during daytime, showed a large incidence rate of aggressive behavior during REM sleep."* This can be interpreted as the result of a broad activation of Unit L without a cortical regulation.[30]

In relation to humans, it is interesting to find the existence of a clinical sleep disorder that is similar to *"oneiric behavior"* experimentally induced in cats. This disorder is named *"REM sleep behavior disorder"* (RBD) and is characterized by the absence of a muscle paralysis, which is customary during this stage of sleep, due to neurological related disorders. In contrast to sleepwalking, which occurs during the slow-wave stage of non-REM sleep and in people who generally behave in a peaceful way, patients with RBD frequently have accidents and physical and verbal assaults on other people during these episodes.[16,35,36] This behavior is very similar to that of the Jouvet et al.[29] cats and is the result of the activation of Units 1, 2, and especially L, along with the simultaneous inhibition of the prefrontal lobe. RBD seems to be an early warning sign of Parkinson's disease, as some authors have noted that 65% of patients with this sleep disorder develop the disease on a nine-year average after RBD shows up,[37] indicating the degenerative etiology of this disorder. Thus, RBD also represents an etiological model for the study of oneiric behavior.

The Model of the Four Units of Dreaming: A Proposal

The previous information allows us to suggest that during dreaming, it is the inhibition of the prefrontal lobe in the dorsolateral region that is in charge of the executive functions and in its orbitofrontal region that is related to the regulation of limbic impulses. The parietal-temporal-occipital (PTO) region is responsible for the nature of the oneiric content; that is, the lack of planning and control of critical and coherent thought toward what is dreamt, as well as the ease in which emotional and motivational impulses emerge in comparison with wakefulness. Thus, the main proposal of this model, is that these characteristics of the oneiric content basically correspond to the increase in the activity of Unit1, 2 (with the exception of the PTO region), L, and the medial region of the prefrontal lobe that occurs simultaneously with the inhibition of the dorsolateral and orbital regions of Unit 3 *(table 1)*. This leads us to think of the oneiric process as basically a process of confabulation, suggesting that dreaming is a type of normal confabulation that happens every night in a cyclic way but that does not differ much from the confabulatory thoughts of patients with frontal lobe damage. Luria 1 mentioned that the confabulations of these patients are similar to the oneiric states in terms of the loss of the selectivity of mental processes, which is typical of the normal conscious life *(Figure 1)*. Meanwhile, Koukkou and Lehman 38 have suggested that the cerebral state of an adult during dreaming corresponds functionally to the state of wakefulness during childhood based on the similarity of the electroencephalographic activity of the different phases of sleep and in the human development phases. They suggest that every time we dream there is an age regression on the psychophysiological functioning that causes us to have access to cognitive memories and strategies of that age. They also suggested that these cognitive strategies during dreaming in adults are equivalent to the processes of fantasy and are far from the reality thought of a young child during wakefulness (Piaget's preoperational stage). Some data indicate that the prefrontal lobe does not reach maturity until between the ages of 10 to 12 years.[39] Then, according to this hypothesis, a functional regression on the cognitive activity in

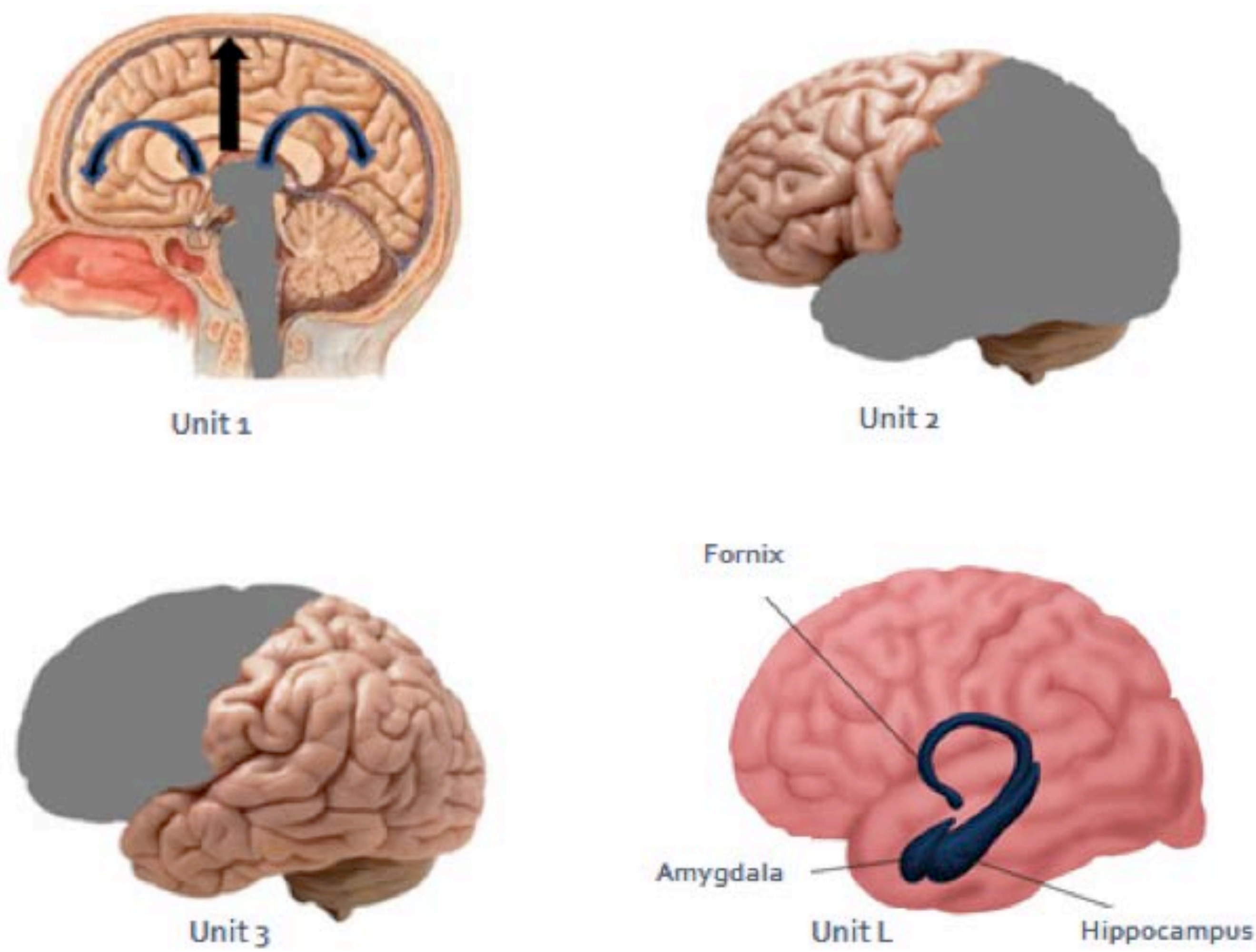

Figure 1. This picture shows the four Functional Units of the brain: Unit 1(reticular system), Unit 2 (parietal, occipital and temporal lobes), Unit 3 (Frontal lobe) and Unit L (limbic system).

dreaming would imply an incomplete functioning of the prefrontal lobe.

On the other hand, the memory of dreams becomes interesting. It is well known that dreams are difficult to remember in wakefulness.[14] This is possibly explained through the state-dependent theory.[38] According to the model that is presented in this research, dreams are difficult to remember precisely because of the lack of operation of the working memory due to the relative deactivation of the prefrontal lobe. It has been proven through fMRI that the activation of the frontal lobe and the parahippocampal region of the limbic system during the presentation of semantic and visual non-verbal information predicts its subsequent recall, showing the important role of these two structures in memory.[40,41] During dreaming, only the limbic region is activated, not the prefrontal, which produces a partial or absent memory of the oneiric content upon waking up in most people *(Figure 1)*.

We can arrive at the conclusion that dreams, as well as the cognitive activity in wakefulness, can come in various forms and contents. For example, there could be dreams with a very high emotional content due to an intense activity of Unit L, a high imaginative-visual content with an increase of activity in the right hemisphere of Unit 2 or a high narrative-linguistic content produced by the left hemisphere, but always partnered with an inhibition of the dorsolateral and basal regions of Unit 3. It has been shown that *"lucid"* dreams are characterized by *"being able to freely remember the circumstances of waking life, to think clearly, and to act deliberately upon reflection, all while experiencing a dream world that seems vividly real"*. [42] These are dreams where the control and direction of the oneiric process are maintained and the dreamer is aware that he is dreaming. This is the result of an exceptional and sudden reactivation of the functioning of the dorsolateral and medial regions of the left prefrontal lobe and the temporoparietal region during REM sleep. During wakefulness, complex information processing is subserved by these regions, but they are not active during non-lucid dreaming. This pattern of brain activity explains the recovery of the executive metacognitive abilities and voluntary control that characterizes lucid dreaming *(Figure 1)*.[43,44]

Foulkes,[45] whose studies were also based on Luria's work, suggested another model of brain functioning during dreaming. This author suggested that dreaming is generated verbally in the left frontal lobe, which remains functional during REM sleep and *"that competes with the basal affective and posterior associative systems that are left uninhibited during sleep."* However, this model has not been entirely confirmed by recent studies with PET.[8-10,30]

The Function of Dreaming: Cognitive-Emotional Homeostasis

We propose that the inhibition of prefrontal lobe functioning and the increase in activity of Unit L during REM sleep can have a cognitive and emotional homeostasis function that is important for good performance during wakefulness. This allows an increase in prefrontal lobe functioning and a decrease of limbic activity throughout the day, allowing better impulse control, planning direction, and self-regulation of behavior.

The statement above agrees with the *"Motivational Theory of REM Sleep"* from Vogel,[46] which suggests that the function of this phase of dreaming is to decrease the impulse-motivated behavior during wakefulness. This researcher proposes the prior in light of the observation that the selective deprivation of REM sleep in animals produces increases in aggressive, sexual and food-seeking behaviors. It was also noticed that the deprivation of REM sleep in patients experiencing endogenous behavior improves their symptomatology.[47] Meanwhile, Vogel[46] argued that the decrease in the amount of REM sleep was a consequence of the use of antidepressant drugs is caused by an increase in impulse-motivated behavior during wakefulness, and therefore, a clinical improvement of depression. In his new model to explain depression, Beck[33] affirms that in patients with depression, there is a hyperactivity of the amygdala that causes an excessive reactivity in the presence of negative events and hypoactivity of the prefrontal lobe that prevents a proper interpretation of the event and counteracts the high activity of the amygdala. In fact, the patients with depression show an increase in the metabolism of the dorsolateral region of the prefrontal lobe during REM sleep instead of a decrease as is observed in subjects without depression, and this causes a decrease in the dorsolateral region of the prefrontal lobe and an increase of activity of the limbic system, preventing the regulation and evaluation of social contexts and circumstances, sensations, and emotions in a suitable way during wakefulness.[48] As we have examined, the prefrontal lobe of the human being is extremely sensitive to sleep; its functioning is altered by sleep deprivation, and it benefits and recovers with a sleep of good quality and quantity (Muzur, Pace-Schott, et al., 2002). The usual effects of sleep deprivation on the prefrontal lobe's functions are well known and include irritability, lack of attention and concentration, working memory impairments, and lack of self-regulation skills.[50] Sleep deprivation makes us more sensible to emotional and stress-induced stimuli.[51]

This also favors the hypothesis that frontal hypoactivity and limbic hyperactivity during REM sleep is really homeostatic, meaning that the increase of the emotional and motivational activity works as an escape valve during the night without the logical, reasoned, and regulating activity of the prefrontal lobe, and that during the day, the limbic activity decreases, and the dorsolateral and orbital activity of the prefrontal lobe increases.

Schwartz and Maquet[52] suggested that the bizarre content of dreaming is similar to certain neuropsychological syndromes that produce visual and spatial agnosia. During REM sleep, the cerebral structures that are activated and deactivated are similar to the cerebral regions damaged in the former neuropsychological syndromes. In addition to the content of dreaming, there is an absence of control over the course of the scenes due to the lack of critical thinking that evaluates the coherence of what is happening; therefore, there is a passive and non-critic acceptance of what is occurring during the dream.[3]

That is the reason why Hobson[44] stated that dreaming represents a model for the explanation of schizophrenia because in both, cognitive and emotional similarities are present, such as the exaggeration of the emotional activity that contributes to the lack of selective attention and the deterioration of rationality and the direction of cognitive knowledge, which besides being grotesque, contains a great quantity of confabulations. Furthermore, both conditions show a similar neuropsychological functioning: a hypofunctioning of the frontal lobe and an activation of Unit L. These characteristics impede the schizophrenic patient and the dreamer from organizing their thoughts, integrating them with emotions, and turning them into appropriate actions. Schizophrenia studies with PET have shown diminishing frontal lobe functioning.

It can be said that dreaming is a state similar to a *"schizophrenic or frontal syndrome"*, but temporary, normal and healthy, so that the next day, it can carry out its homeostatic function and favor an optimal functioning of the dorsolateral and orbital region of the frontal lobe during wakefulness. In this way, the frontal lobe can carry out the functions of planning, execution, evaluation, attention, working memory, self-observation, a better impulse control, and proper decision making to be able to carry out a proper everyday life of social interaction.

Dresler et al. found that in lucid dreaming, the brain structures that are active are the ones that malfunction in schizophrenia, and this is what prevents patients from becoming aware of their pathological state.[53] We can state that *"the oneiric craziness of every night"* is a necessary escape valve to act in a sane way during the state of wakefulness. According to Solms,[54] a renowned researcher in the neuropsychology of dreaming, these data support the essential idea proposed by Freud,[5] who maintained that one of the functions of dreaming was to allow instinctive impulses to emerge (limbic) without the censor mechanism (dorsolateral and orbital prefrontal regions), allowing the attainment of repressed desires in a safe way.

Finally, we would like to mention several research questions that result from this article: How is the oneiric content in a patient with prefrontal damage? How can it be proven that the hypofunctioning of the prefrontal lobe and the limbic hyperfunctioning during dreaming is a homeostatic need for good psychological functioning during wakefulness?

There is a proven antidepressant effect of REM sleep deprivation.[47,48] Does it produce changes in the biochemical functioning of the prefrontal lobe and the limbic system? Answering these and other questions will allow the continuity of progress in this new and interesting field in the neurosciences: the neuropsychology of dreaming.

Conclusiones

Luria´s Model of the Brain's Functional Units can be used to explain the generation of dreams and their characteristics. The similarity of dreaming with frontal syndrome and schizophrenia are stressed, especially in terms of the confabulations, the lack of impulse control, and the lack of direction and monitoring that occurs in these disorders. In addition, the suggested hypothesis of the homeostatic character of REM sleep favors the working of the brain in an inverse way during the state of wakefulness to assist the better psychological functioning of the individual.

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