Medical Hypotheses 85 (2015) 359–364

Contents lists available at ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

Sleep: The hebbian reinforcement of the local inhibitory synapses

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ARTICLE INFO

Article history:
Received 20 February 2015
Accepted 17 June 2015

ABSTRACT

Sleep is ubiquitous among the animal realm, and represents about 30% of our lives. Despite numerous efforts, the reason behind our need for sleep is still unknown. The Theory of neuronal Cognition (TnC) proposes that sleep is the period of time during which the local inhibitory synapses (in particular the cortical ones) are replenished. Indeed, as long as the active brain stays awake, hebbian learning guarantees that efficient inhibitory synapses lose their efficiency – just because they are efficient at avoiding the activation of the targeted neurons. Since hebbian learning is the only known mechanism of synapse modification, it follows that to replenish the inhibitory synapses' efficiency, source and targeted neurons must be activated together. This is achieved by a local depolarization that may travel (wave). The period of time during which such slow waves are experienced has been named the "slow-wave sleep" (SWS). It is cut into several pieces by shorter periods of paradoxical sleep (REM) which activity resembles that of the awake state. Indeed, SWS – because it only allows local neural activation – decreases the excitatory long distance connections strength. To avoid losing the associations built during the awake state, these long distance activations are played again during the REM sleep. REM and SWS sleeps act together to guarantee that when the subject awakes again, his inhibitory synaptic efficiency is restored and his (excitatory) long distance associations are still there.

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Introduction

All creatures with a brain are able to learn – and must sleep. Indeed, sleep evidence has been found in most species studied, including cubomedusan jellyfish [1], tree frogs [2], lizards [3], drosophilas [4,5], zebrafish [6], birds [7], and all mammals [8] (starting with the platypus [9]). Sleep may take curious forms such as for the dolphin [10] whose sleep implies only one hemisphere at a time. This happens because breathing is a voluntary act, and requires an awake state. Therefore the dolphin exhibits a two hours sleep in one hemisphere, one hour of awake state for both hemispheres followed by two hours of sleep in the other hemisphere, all over the twelve hours of night. During their migration, several species of birds fly for several days, and sleep while flying a few seconds at a time [11], or one hemisphere at a time.

Sleep seems ubiquitous in the animal realm, and occurs as soon as the species exhibits learning abilities. Only a few (primitive) species seem to be deprived of sleep [8]. Since sleep puts the subject in a dangerous situation with greatly reduced sensory responsiveness (and therefore increased possibility to be eaten by predators), one could conclude that if the process of natural selection has not banished sleep from the behavior repertoire [12], then it must be mandatory. It looks like sleep is a necessary component of learning – even an integral part of it. It is the opinion of most sleep researchers [13–18].

The duration of sleep varies between species, and also among members of a same species. Humans sleep an average of 8 h per day, but a few of us are satisfied with only three hours of sleep, while others demand ten hours [8]. Also, sleep requirements varies with the age. Toddlers sleep more than children, who sleep much longer than elderly people [8]. It is acknowledged that young subjects learn more during the day than elderly ones [19], which emphasizes again the relation between learning and sleeping. Since both actions are intrinsically connected, the explanation of sleep must lay in the learning process.

Learning in the brain

At a neuronal level, learning obeys the hebbian rule, the only known rule of synapse efficiency modification. It was formulated in 1942 by Donald Hebb [20], confirmed by electrophysiology several decades later [21], and since that time, no other proposal has shown up in order to explain learning at a neuronal level. Hebb's rule states that "neurons who fire together get reinforced", and neurons which do not (fire together) see their connection efficiency diminish.

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http://dx.doi.org/10.1016/j.mehy.2015.06.013
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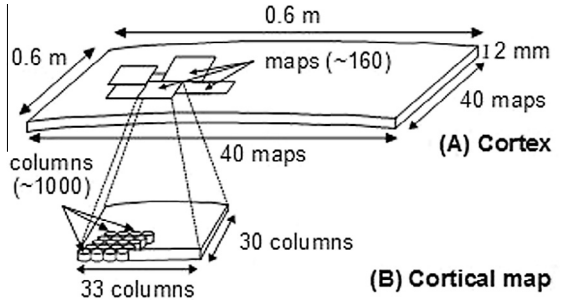

Fig. 1. (a) Following the proposal of T. Kohonen [27], the TnC models the cortex as an homogeneous organization of cortical columns. Each column is a set of about 1000 minicolumns, each minicolumn containing about 110 neurons (in the human cortex [25,28]). (b) The columns belong to one of the estimated 160 cortical maps, each map is specific to a certain kind of information. The maps belonging to the primary and secondary cortex represent dimensions of the sensory events. Maps of higher level of abstraction combine and fuse information from lower level maps [29]. This fact prohibits an easy interpretation of the information they represent (see Tanaka [30] for examples of high level information representations). The cortical map columns prevent their neighbor activations through lateral inhibition [31,32] – but are potentially excitatorily connected to any other columns of the cortex [23] (adapted from [22]).

As stated by the Theory of neuronal Cognition (TnC), the brain does not process information – but represents it [52]. Therefore, it is most important that world regularities are associated to identifiable sets of neurons. The neural localization of a world event is an identification *per se*, and allows to act consequently. Neural localization is obtained thanks to inhibitory connections which limit the number of excited neurons at any given time. The proportion of inhibitory connections in the human brain is about 40% of all connections [23].

In the case of the human species, 22% of the neurons belong to the cortex [24]. The (human) cortex is composed of about 160,000 cortical columns [25] (a column is a set of about 100,000 neurons), each column belonging to one of the estimated 160 cortical maps that form the cortex (Fig. 1). The columnar architecture of the cortex is genetically determined, but the fine-tuning of its functioning relies on neuroplasticity [26].

Inhibitory synapse efficiency

Wakefulness functioning requires inhibition to induce localization of the information at the cost of suppressing neighbor activities (in order to end up with only one locally activated column). The inhibitory connections are plastic and obey hebbian learning [33–39]. Since local inhibitory connections are efficient at suppressing the targeted neurons' activity, these inhibitory connections are never reinforced, and their strength diminishes as the day progresses.

On the contrary, excitatory long distance connections linking two or more columns belonging to separated cortical maps are reinforced as soon as they are activated in the same time frame (Hebb rule again) and are constitutive of the learned experience. As advocated by the TnC, such long distance excitatory connections are representative of «high level» information extraction, and may even account for our flashes of «intelligence» [40].

There is no secret that as the day progresses, we are less and less cognitively efficient. In case of sleep deprivation, the subjects thinking process becomes fuzzy, prone to hallucinations, etc. Van Dongen et al. [41] provide evidences that the build-up of neurobehavioral deficits is not caused by reduction of sleep time *per se*, but rather by excessive wakefulness beyond a maximum period during which stable neurobehavioral functioning could be maintained. "Excess wakefulness" is all waking time beyond a hypothetical critical period. They provide results showing a near-linear relationship linking each consecutive hour of wake extension (*i.e.*, excess wakefulness) to an increase in lapses of behavioral alertness (Fig. 2).

Our hypothesis is that the reduced alertness is the result of Hebb learning onto the inhibitory inter-column connections. Less inhibitory efficiency means that instead of a unique activated column in a given location, there may be two, or more, which induces a less precise representation of the event. If this event is a world situation perceived by the body senses, it may require more time to be recognized (lapse of behavioral alertness). In some cases, it may even be wrongly recognized (confusion, hallucination).

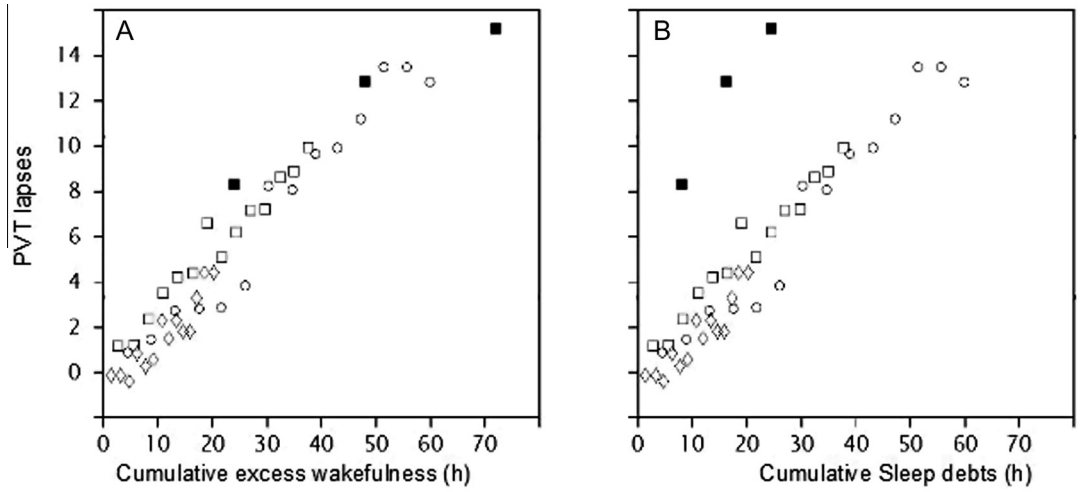

Fig. 2. The panels show behavioral alertness, as measured by psychomotor vigilance task (PVT) performance lapses (relative to baseline), plotted as a function of cumulative excess wakefulness (panel A) and as a function of cumulative sleep debt (panel B). Cumulative wake extension (*i.e.*, excess wakefulness – A), rather than cumulative loss of sleep (*i.e.*, sleep debt – B), is the primary cause of progressively reduced behavioral alertness both across days of chronic sleep restriction and across days of total sleep deprivation. Subjects in all four experimental conditions (sleep restriction: 8 h (diamond), 6 h (light square), 4 h (circle) and 0 h (black square) appeared to experience the same cumulative "cost" (*i.e.*, increase in lapses of behavioral alertness) for each consecutive hour they extended their wake periods (near-linear relationship, A), but if considered from the perspective of sleep debt (B), the response to total sleep deprivation (black square) is fundamentally different from the chronic sleep restriction (adapted from [41]).

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