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**Physiology of sleep**

Sleep is a physiological and behavioral process that an individual requires to carry out his daily functions. This process is completed in a regular and continuous manner every night.

As a part of biological rhythm, human brain has a healthy functioning by differentiating dark and day hours of the day. From controlling hormone levels to muscle tone, from regulating pace of breathing to contents of our thought; sleep influences all bodily and mental functions. It is not surprising that sleep can make these changes happen in the body because sleep causes significant changes in the electrical activity of the brain as a whole. Sleep characterizes itself by not responding to one’s surroundings and by drifting away from perception; yet it is a reversible behavior. During 1940–1950, physiologists believed that sleep was initiated as a result of tiredness that developed during the day and by a slowing down in the activation of the fore brain from weakening in the activation of the reticular activating system. Later, based on transection studies, brain stem was shown to be responsible for generating sleep especially studies in cats; where total sections performed on pontine tegmentum induce sleeplessness.

During sleep, the activity of RAS stops and the transmission of sensory information through thalamus is blocked and the stimulation of cortex is prevented. Anatomic structures responsible for the hypothalamic control of sleep and wakefulness: for wakefulness, stimuli originating from rostral pons and caudal midbrain regions reach paramedian midbrain in diencephalon and here the signals divide into two paths aiming to reach thalamus and hypothalamus. Main structures projecting to thalamus are PedunculoPontine Tegmental (PPT) and LateroDorsal Tegmental (LDT) nuclei that are of cholinergic nature.

The structure that initiates sleep is thought to be the ventrolateral preoptic nucleus (VLPO) located on the anterior part of the hypothalamus. VLPO nucleus suppresses the activities of brain stem, pons and locus coeruleus, dorsal raphe nucleus, laterodorsal tegmental pedunculopontine tegmental nucleus via GABA and galanin neurotransmitters. Suprachiasmatic Nucleus (SCN) is known as the light sensitive circadian pacemaker. Throughout daytime light stimulus is transmitted from retina to hypothalamus through neural pathways and results in secretion of melatonin from the pineal gland. It is an anatomical structure that has a central role in maintaining the day-night rhythm . Neurotransmitters controlling sleep and wakefulness can be listed as: “Glutamate, Acetylcholine, Histamine, Norepinephrine and GABA”. Reticular activating system stimulates the cortex by using glutamate while ponto-mesencephalic tegmental neurons do the same job by using acetylcholine. Neurons at locus coeruleus use mostly norepinephrine, these extend from the brain stem to the cerebral cortex by including the fore brain, and they activate the stimulation of the cortex and contribute to maintaining sleep. Cholinergic neuronal network results in wakefulness in two types of cortexes: (1) It projects to laterodorsal tegmental and pedunculopontine tegmental nuclei, midline and intralaminar thalamic nuclei and to a lesser degree to lateral hypothalamus and basal for brain. (2) Cholinergic neuron group starts from the basal for brain and has a widespread projection to cortex. This pontomesencephalic neuron group is part of the ascending reticular activating system; they not only play a part in the activation during wakefulness but also are actively involved in paradoxical sleep. Glutamate is another excitatory neurotransmitter; it acts as the primary neurotransmitter of the ascending reticular activating system. Glutamate is found at a very high concentration at the brain stem reticular formation. This neurotransmitter plays an active role in the wakeful brain and is secreted from the cortical cells to a significant degree throughout wakefulness. During slow wave sleep “burst discharges” appear due to the activation of special glutamate receptors. Histamine also plays an important role in wakefulness. Neurons containing histamine are found in tuberomammillary nuclei and in posterior hypothalamus. Noradrenergic neurons (locus coeruleus), have diffuse projections in the brain that extend to the cortex. Histaminergic neurons are associated with cortical activation during wakefulness whereas they are shut down during REM sleep. To sleep there needs to be a shift from sympathetic regulation to parasympathetic regulation. Parasympathetic centers of significance are found in “solitary tract nucleus neurons, anterior hypothalamus and preoptic fields”. Serotonergic raphe neurons facilitate the initiation of sleep while GABA-ergic neurons inhibit the activating system. These GABA-ergic neurons are selectively activated during slow wave sleep. As a result of this inhibition, brain stem, hypothalamus and nasal fore brain are suppressed and disfacilitation (inhibition) and hyperpolarization of thalamocortical system takes place. Thereby from the wakeful state where we see rapid, tonic discharges on EEG, the system shifts to sleep state we start recording sleep spindles and slow wave activity. Initiation and continuation of slow wave sleep is made possible by lengthening and strengthening the inhibition of the activating system with GABA-ergic system

Both phases of sleep are involved in memory consolidation. Very little new information is gained during sleep, but consolidation and maintenance of memory from experiences of the previous day is considerable. It is known that learning of visual information is improved during the first night of sleep and that sleep deprivation impairs recall of the information. Different types of sleep have a different effect on memory consolidation and retention of information. Retention is best if stage 3 and 4 non-REM sleep occurs in the first 2 hours of sleep and if the last 25% of sleep is REM sleep.

The type of sleep also affects the type of information which is consolidated by the brain. Learning of movement sequences is best if stage 2 non-REM sleep occurs late in the night, while learning of cognitive sequences occurs best if there is a cycle of REM and non-REM sleep.

Dreams are a manifestation of underlying brain activity and reflect the loose associative connections made during REM sleep. These loose associations are likely to result in increased creative mental activity and problem-solving abilities.

**Basal ganglia and movements**

The basal ganglia (or basal nuclei) are a group of nuclei of varied origin in the brains of vertebrates that act as a cohesive functional unit. They are situated at the base of the forebrain and are strongly connected with the cerebral cortex, thalamus, and other brain areas. The components of the basal ganglia include the striatum, pallidum, substantia nigra, and subthalamic nucleus. Each of these components has a complex internal anatomical and neurochemical organization.

The basal ganglia are associated with a variety of functions, including voluntary motor control, procedural learning relating to routine behaviors or habits such as bruxism, eye movements, and cognitive, emotional functions. Currently, popular theories implicate the basal ganglia primarily in action selection, that is, the decision of which several possible behaviors to execute at a given time. Experimental studies show that the basal ganglia exert an inhibitory influence on a number of motor systems and that a release of this inhibition permits a motor system to become active. The behavior switching that takes place within the basal ganglia is influenced by signals from many parts of the brain, including the prefrontal cortex, which plays a key role in executive functions.

The basal ganglia play a central role in a number of neurological conditions, including several movement disorders. The most notable are Parkinson’s disease, which involves degeneration of the melanin-pigmented dopamine-producing cells in the substantia nigra pars compacta (SNc), and Huntington’s disease, which primarily involves damage to the striatum.

The intricacies of how basal ganglia activity leads to the facilitation of movement are still a bit unclear, but one popular hypothesis suggests that there are different pathways in the basal ganglia that promote and inhibit movement, respectively. The direct/indirect model is centered around connections the basal ganglia (specifically the globus pallidus and substantia nigra) form with neurons in the thalamus. These thalamic neurons in turn project to the motor cortex (an area of the brain where many voluntary movements originate) and can stimulate movement via these connections. The basal ganglia, however, continuously inhibit the thalamic neurons, which stops them from communicating with the motor cortex—inhibiting movement in the process.