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PHYSIOLOGY OF SLEEP

Sleep is defined as unconsciousness from which a person can be aroused by sensory or other stimuli. It is to be distinguished from coma, which is unconsciousness from which a person cannot be aroused. A person's state of consciousness is defined in two ways:

- Behavior, covering the spectrum from maximum attentiveness to comatose;
- Pattern of brain activity that can be recorded electrically.

This record, known as the **electroencephalogram (EEG)**, portrays the electrical potential difference between different points on the surface of the scalp. The EEG is such a useful tool in identifying the different states of consciousness. EEG patterns are largely due to synchronous graded potentials. The majority of the electrical signal recorded in the EEG originates in the pyramidal cells of the cortex. The processes of these large cells lie close to and perpendicular to the surface of the brain, and the EEG records postsynaptic potentials in their dendrites. EEG patterns are complex waveforms with large variations in both amplitude and frequency.

The wave's amplitude, measured in microvolts (μV), indicates how much electrical activity is occurring beneath the recording electrodes at any given time. A large amplitude indicates that many neurons are being activated simultaneously. In other words, it indicates the degree of synchronous firing of the neurons that are generating the synaptic activity. On the other hand, a small amplitude indicates that these neurons are less activated or are firing asynchronously.

The frequency of the wave indicates how often it cycles from the maximal to the minimal amplitude and back. The frequency is measured in hertz (Hz, or cycles per second) and may vary from 0.5 to 40 Hz or higher. In general, lower EEG frequencies indicate less responsive states, such as sleep, whereas higher frequencies indicate increased alertness. As we will see, one stage of sleep is an exception to this general relationship.

The EEG pattern changes profoundly in sleep. As a person becomes increasingly drowsy, his or her wave pattern transitions from a beta rhythm of >12 Hz (high frequency, small amplitude) to a predominantly alpha rhythm of 8 to 12 Hz (low frequency, larger amplitude). When sleep actually occurs, the EEG shifts toward lower-frequency, larger-amplitude wave patterns known

as the theta rhythm (4–8 Hz) and the delta rhythm (slower than 4 Hz). Relaxation of posture, decreased ease of arousal, increased threshold for sensory stimuli, and decreased motor neuron output accompany these EEG changes.

PHASES OF SLEEP

There are two phases of sleep, the names of which depend on whether or not the eyes move behind the closed eyelids:

- NREM (non–rapid eye movement) sleep, also known as, slow-wave sleep (SWS).
- REM (rapid eye movement) sleep.

NREM (non–rapid eye movement) Sleep

The initial phase of sleep—NREM sleep—is subdivided into three stages. Each successive stage is characterized by an EEG pattern with a lower frequency and larger amplitude than the preceding one. In stage N1 sleep, theta waves begin to be interspersed among the alpha pattern. In stage N2, high frequency bursts called sleep spindles and large-amplitude K complexes occasionally interrupt the theta rhythm. Delta waves first appear along with the theta rhythm in stage N3 sleep; as this stage continues, the dominant pattern becomes a delta rhythm, sometimes referred to as slow-wave sleep. Sleep begins with the progression from stage N1 to stage N3 of NREM sleep, which normally takes 30 to 45 min. This sleep is exceedingly restful and is associated with decreases in both peripheral vascular tone and many other vegetative functions of the body. For instance, 10 to 30 percent decreases occur in blood pressure, respiratory rate, and basal metabolic rate. Although slow-wave sleep is frequently called “dreamless sleep,” dreams and sometimes even nightmares do occur during slow-wave sleep. However, dreams of slow-wave sleep are usually not remembered because consolidation of the dreams in memory does not occur. Most sleep during each night is of the slow wave (NREM) variety.

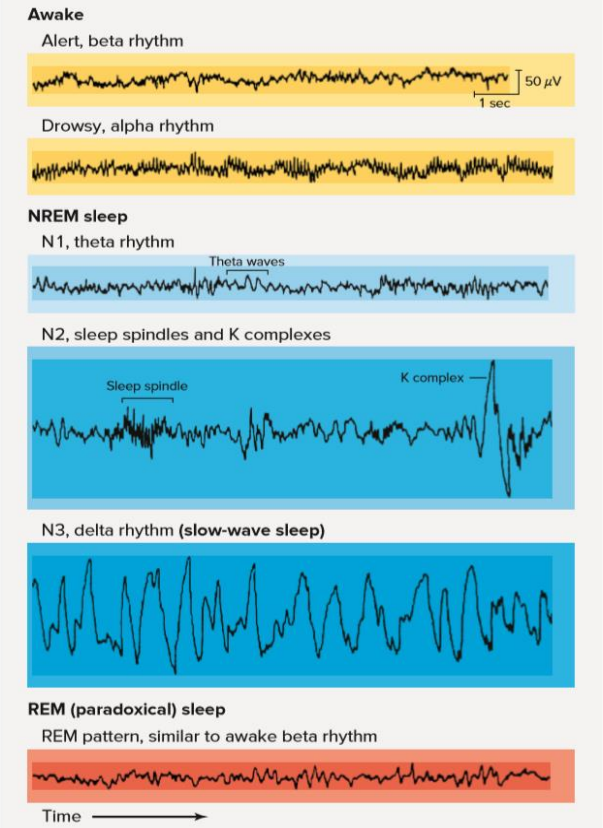
REM (rapid eye movement) Sleep

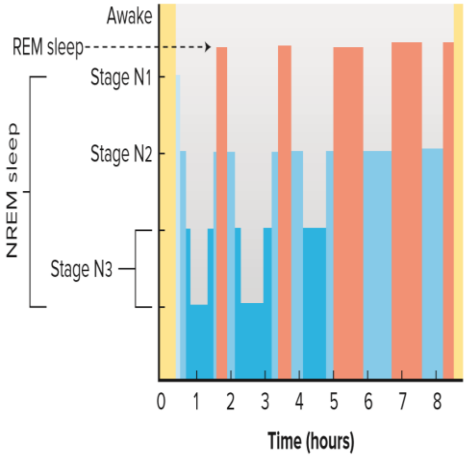
REM sleep is also called paradoxical sleep, because even though a person is asleep and difficult to arouse, his or her EEG pattern shows intense activity that is similar to that observed in the alert, awake state. In fact, brain O2 consumption is higher during REM sleep than during the NREM or awake states. This type of sleep is not so restful, and it is often associated with vivid dreaming. In a normal night of sleep, bouts of REM sleep lasting 5 to 30 minutes usually appear on average every 90 minutes in young adults. When a person is extremely sleepy, each bout of REM sleep is short and may even be absent. As the person becomes more rested through the night, the durations of the REM bouts increase. REM sleep occurs in episodes that occupy about 25 percent of the sleep time in young adults.

Sleep Cycle

There is an orderly progression of sleep stages and states during a typical sleep period. When an individual falls asleep, the N1 stage of SWS sleep is entered first. During next hour or so the person passes progressively in deeper stages of sleep until deep sleep is reached. After about 15 minutes of deep sleep, depth of sleep starts decreasing and continues to do so until the person re-enters the N1 stage of sleep about 90 minutes after the start of the first sleep cycle. At this point, individual passes from SWS to REM sleep. This cycle repeats itself about 5 times during night. After the second cycle, the intervals between periods of REM sleep become shorter and the duration of each of period of REM sleep becomes longer. As morning approaches, an individual spends less time in the deeper stages of SWS and periodically awakens. This sleep cycle is typical of adult.

During infancy, about 16 hours of every day are spent asleep. Th is decreases to 10 hours during childhood and 7 hours during adulthood. In elderly people sleep becomes 6 hours/day.

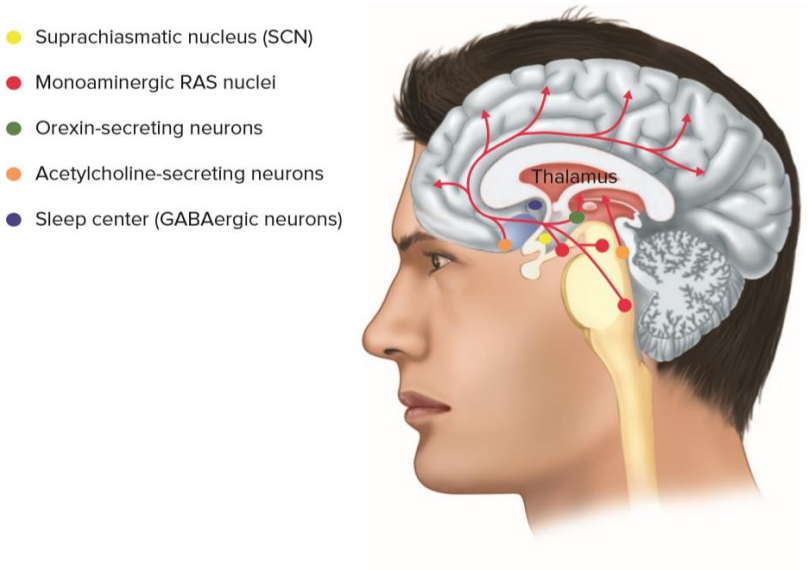




Basic Theories of Sleep

Mechanism of sleep is not very clear and has been explained by several theories:

1. *Passive theory of sleep*: It was thought earlier that excitatory areas of reticular activating system send signals to cortex and are responsible for waking state. Fatigue of reticular activating system makes it inactive, and causes sleep. This is the passive theory of sleep.
2. *Active theory of sleep*: Some centers located in mid pons actively cause sleep by inhibiting other parts of the brain.
3. *Stimulation of raphe nuclei in the lower half of the pons and in the medulla causes sleep*: Nerve fibers from these nuclei are spread widely in the reticular formation, upward into the thalamus, neocortex, hypothalamus and most of the areas of limbic system. Fibers also run downwards in the spinal cord terminating on posterior horn cells to inhibit incoming pain signals. Most of these endings secrete serotonin which is probably the major neurotransmitter associated with production of sleep. Excitation of raphe nuclei stimulates some areas in nucleus tractus solitarius for promoting sleep through serotonergic system (stimulation of certain areas of tractus solitarius also produces sleep). Stimulation of several regions in the diencephalon, e.g. suprachiasmatic nucleus of the hypothalamus, diff use nuclei of thalamus also produces sleep. Discrete lesions of raphe nucleus produces a state of wakefulness. Other possible transmitters related to sleep are muramyl peptide and non-apeptide which accumulate in blood and CSF when animal is kept awake for several days. When they are injected in the third ventricle, they cause sleep. Therefore, it is possible that prolonged wakefulness causes progressive accumulation of a sleep factor in the brain.



Importance of Sleep

Although we spend about one-third of our lives sleeping, the functions of sleep are not completely understood. Many lines of research, however, suggest that sleep is a fundamental necessity of a complex nervous system. Studies of sleep deprivation in humans and other animals suggest that sleep is a homeostatic requirement, similar to the need for food and water. Deprivation of sleep impairs the immune system, causes cognitive and memory deficits, and ultimately leads to psychosis and even death. Part of the restorative mechanism of sleep may arise from removal of protein fragments, waste products, and neurotransmitters that accumulate from brain activity in the awake state. During sleep, the space between neurons increases more than 60% due to transient shrinking of glial cells, allowing a significant increase in the flow of cerebrospinal fluid between neurons.

Stage	Behavior	EEG (See Figures 8.3 and 8.4)
Alert wakefulness	Awake, alert with eyes open.	Beta rhythm (greater than 12 Hz).
Relaxed wakefulness	Awake, relaxed with eyes closed.	Mainly alpha rhythm (8–12 Hz) over the parietal and occipital lobes. Changes to beta rhythm in response to internal or external stimuli.
Relaxed drowsiness	Fatigued, tired, or bored; eyelids may narrow and close; head may start to droop; momentary lapses of attention and alertness. Sleepy but not asleep.	Decrease in alpha-wave amplitude and frequency.
NREM (slow-wave) sleep		
Stage N1	Light sleep; easily aroused by moderate stimuli or even by neck muscle jerks triggered by muscle stretch receptors as head nods; continuous lack of awareness.	Alpha waves reduced in frequency, amplitude, and percentage of time present; gaps in alpha rhythm filled with theta (4–8 Hz) and delta (slower than 4 Hz) activity.
Stage N2	Further lack of sensitivity to activation and arousal.	Alpha waves replaced by random waves of greater amplitude.
Stage N3	Deep sleep; in stage N3, activation and arousal occur only with vigorous stimulation.	Much theta and delta activity; progressive increase in amount of delta.
REM (paradoxical) sleep	Greatest muscle relaxation and difficulty of arousal; begins 50–90 min after sleep onset, episodes repeated every 60–90 min, each episode lasting about 10 min; dreaming frequently occurs, rapid eye movements behind closed eyelids; marked increase in brain O_2 consumption.	EEG resembles that of alert awake state.

Clinical Correlates

Sleep Apnea: In one form of a disease known as sleep apnea, stimulation of the respiratory muscles temporarily ceases, sometimes hundreds of times during a night. The resulting decreases in oxygen levels repeatedly awaken the apnea sufferer, who is deprived of both slow-wave and REM sleep. As a result, this disease is associated with excessive—and sometimes dangerous—sleepiness during the day.

THE ROLE OF BASAL GANGLIA IN COORDINATING MOVEMENT

Basal ganglia are the group of neurons at the base of cerebral cortex and the brainstem. They include;

1. Caudate nucleus
2. Putamen
3. Globus pallidus
4. Substantia nigra (midbrain)
5. Subthalamic nucleus (diencephalon)

The basal ganglia have cognitive, emotional and movement related functions. However, they are best known for their role in movement. The caudate nucleus and putamen are collectively called corpus striatum. Putamen and globus pallidus together form a bean-shaped lenticular nucleus. Caudate nucleus has a tail and head. Between caudate nucleus and putamen lies the anterior limb of internal capsule. The Substantia nigra is located within the midbrain while the Subthalamic nucleus is located posterior to the thalamus within the diencephalon.

The basal ganglia receive information from the cerebral cortex through the caudate nucleus or the putamen. These are the main input structures of the basal ganglia. The globus pallidus and the substantia nigra are the major output structures and they send information to the cerebral cortex (mostly through the thalamus) and nuclei in the brainstem. The basal ganglia on its own (i.e. activity in the basal ganglia alone) does not initiate movement but they control the structures that initiate movement through the thalamus.

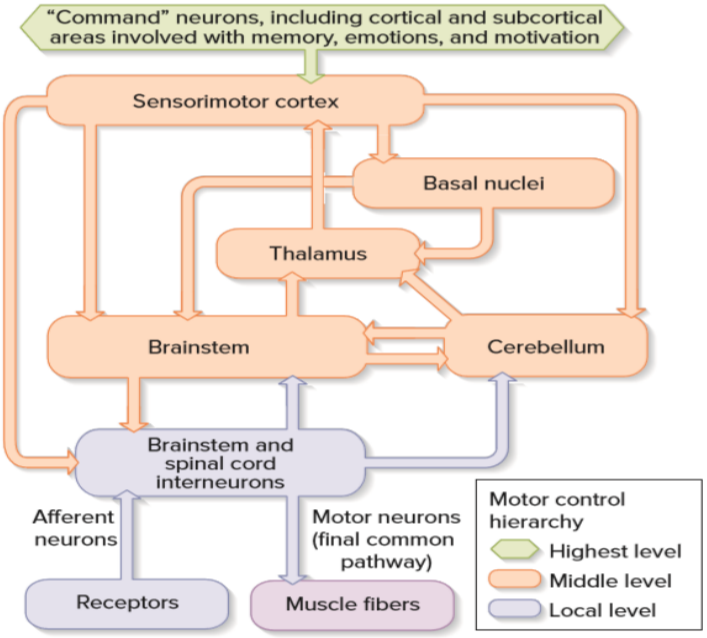


FIG. 1 organization of the neural systems controlling body movement

Role of the Basal Ganglia in Motor Control

Most of the motor actions occur as a consequence of thoughts generated in the mind. This process is known as cognitive control of motor activity. Basal ganglia play role because caudate nucleus extends into all lobes of cerebrum and it receives large amount of input from the association areas of the cerebral cortex. Association areas are the areas which integrate different types of sensory and motor information into thought patterns. After the signals pass from cerebral cortex to caudate nucleus, they pass to globus pallidus and then through ventroanterior and ventrolateral relay nuclei of thalamus, they return back to prefrontal, premotor and supplementary motor areas of cortex. Due to these connections basal ganglia help in cognitive control of motor activity. Two major circuits within the basal ganglia are responsible for this. They are;

- Putamen Circuit: This circuit functions in executing patterns of motor activity. This circuit functions in association with the corticospinal system to control complex patterns of motor activity. An example is the writing of letters of the alphabet. This circuit begins mainly in the premotor and supplementary areas of the motor cortex and in the somatosensory areas of the sensory cortex. Next they pass to the putamen (mainly bypassing the caudate nucleus), then to the internal portion of the globus pallidus, and next to the ventroanterior and ventrolateral relay nuclei of the thalamus, and they finally return to the cerebral primary motor cortex and to portions of the premotor and supplementary cerebral areas closely associated with the primary motor cortex.

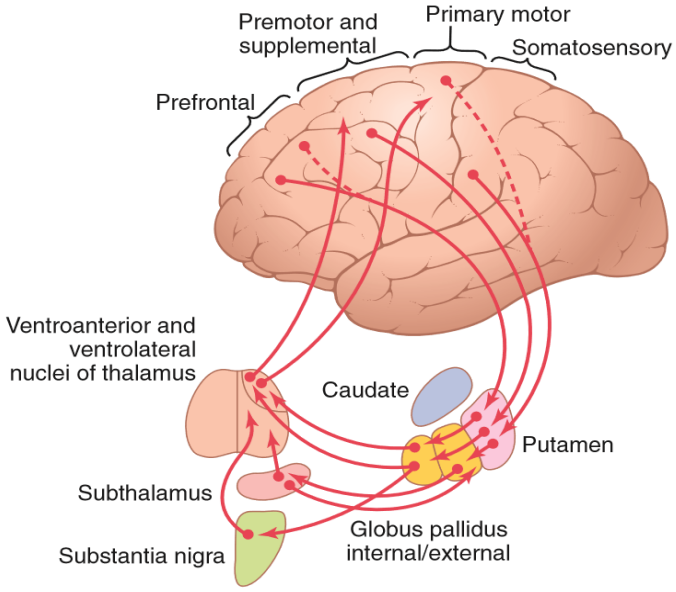


FIG. 2 Putamen circuit

- Caudate Circuit: The caudate circuit plays the role of cognitive control of sequences of motor patterns. The caudate nucleus receives large amounts of its input from the association areas of the cerebral cortex overlying the caudate nucleus, mainly areas that also integrate the different types of sensory and motor information into usable thought patterns. After the signals pass from the cerebral cortex to the caudate nucleus, they are transmitted to the internal globus pallidus, then to the relay nuclei of the ventroanterior and ventrolateral thalamus, and finally back to the prefrontal, premotor, and supplementary motor areas of the cerebral cortex, but with almost none of the returning signals passing directly to the primary motor cortex. Instead, the returning signals go to the accessory motor regions in the premotor and supplementary motor areas that are concerned with putting together sequential patterns of movement lasting 5 or more seconds instead of exciting individual muscle movements. Thus, cognitive control of motor activity determines subconsciously, and within seconds, which patterns of movement will be used together to achieve a complex goal that might itself last for many seconds.

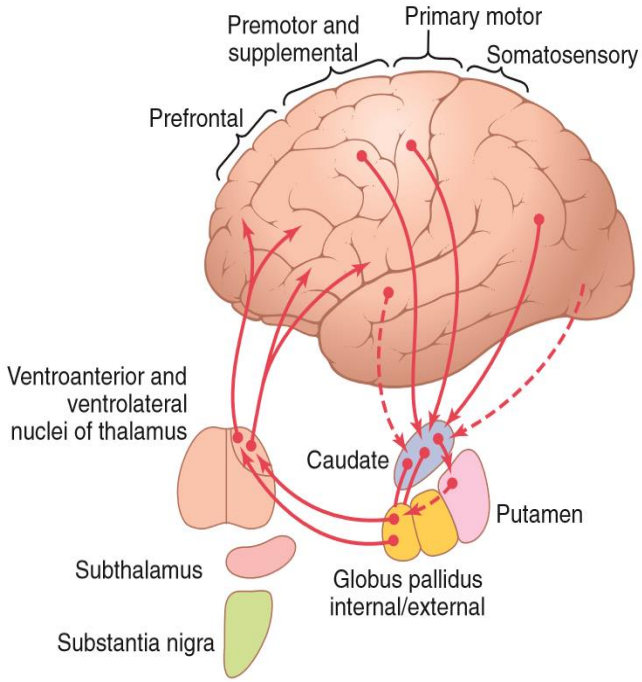


FIG. 3 Caudate circuit

Two important capabilities of the brain in controlling movement are to (1) determine how rapidly the movement is to be performed and (2) control how large the movement will be. In absence of basal ganglia timing and scaling functions become very poor. Because the caudate circuit of the basal ganglia system functions mainly with association areas of the cerebral cortex, it is believed that the timing and scaling of movements are functions of the caudate cognitive motor control circuit. However, there is still much to learn about how the basal ganglia works to achieve these complex movements.

Other functions of the basal ganglia includes;

1. Semiautomatic movements: Swinging of arms while walking are carried out subconsciously at the level of basal ganglia. Crude movements of facial expression that accompany emotion are controlled by basal ganglia. By subconscious control of ordinary activities, basal ganglia relieve cortex from routine acts so that cortex can be free to plan its action.
2. Control of reflex muscular activity: Basal ganglia exert inhibitory effect on spinal reflexes and regulate activity of muscles which maintain posture.
3. Control of muscle tone: Gamma motor neurons, muscle spindle and therefore the muscle tone are controlled by basal ganglia, especially substantia nigra. In lesion of basal ganglia muscle tone increases.

Clinical Correlates

Parkinson's disease: This is a syndrome whereby dopaminergic neurons of the substantia nigra degenerate. When this happens, the ability of the basal ganglia to promote or inhibit movement is affect. This causes difficulty in initiating movement. Symptoms include rigidity, slow movement tremor, postural instability. Thinking and behavioral problems occur in the advance stage. The cause is unknown although it is believed to be genetic and environmental. There is no cure. Treatment involves medications such as levodopa, dopamine agonists.