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MATRIC NUMBER: 17/MHS01/232

COURSE CODE: NEUROPHYSIOLOGY

DEPARTMENT: MEDICINE AND SURGERY

LEVEL: 300

Question 1: **Write on the physiology of sleep.**

Sleep is defined as unconsciousness from which a person can be aroused by sensory or other stimuli. Each night, a person goes through two major types/stages of sleep that alternate with each other.

Stages of sleep

- Slow wave sleep and
- Rapid eye movement sleep (REM)

RAPID EYE MOVEMENT SLEEP (PARADOXICAL, DESYNCHRONIZED) SLEEP

In a normal night of sleep, bouts of rapid eye movement (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.

Characteristics of REM sleep are:

1) It is an active form of sleep usually associated with dreaming and active bodily muscle movements.

2) The person is even more difficult to arouse by sensory stimuli than during seep slow-wave sleep and yet people usually awaken spontaneously in the morning during an episode of REM sleep.

3) Muscle tone throughout the body is exceedingly depressed, indicating strong inhibition of the spinal muscle control areas.

4) Heart rate and respiratory rate usually become irregular, which is characteristic of the dream state.

5) Despite the extreme inhibition of the peripheral muscles, irregular muscle movements do occur in addition to the rapid movements of the eyes.

6) The brain is highly active in REM sleep and overall brain metabolism may be increased as much as 20%. An electroencephalogram (EEG) shows a pattern of brain waves similar to those that occur during wakefulness.

This type of sleep is also paradoxical sleep because it is a paradox, that a person can still be asleep despite the presence of marked activity in the brain.

Possible Cause of REM sleep

Drugs that mimic the action of acetylcholine (Ach) increase the occurrence of REM sleep. Therefore, it has been postulated that the large Ach-secreting neurons in the upper brain stem reticular formation might, through their extensive efferent fibers, activate many portions of the brain.

SLOW-WAVE SLEEP

This stage of sleep occurs after a person has been kept awake for more than 24 hours and it is exceedingly restful and is associated with decreases in both peripheral vascular tone and many other vegetative functions of the body.

Although slow-wave sleep is frequently called dreamless sleep, dream and sometimes even nightmare do occur during slow-wave sleep. In slow-wave sleep, the dreams are usually not remembered because consolidation of dreams in memory does not occur.

Stimulation of several specific areas of the brain can produce sleep, these areas are:

a) The stimulation of the raphe nuclei in the lower half of the pons and in the medulla almost causes natural sleep. The nerve fibers from these nuclei spread locally in the brain stem reticular formation and also upward into the thalamus, hypothalamus, most areas of the limbic system and even the neocortex of the

cerebrum. Many nerve endings of fibers from these raphe neurons secrete **serotonin** (which aids in sleep).

When a drug that blocks the formation of serotonin is administered to an animal, the animal often cannot sleep for the next few days.

b) Stimulation of some areas in the nucleus if the tractus solitarius can also cause sleep. This nucleus is the termination in the medulla and pons for visceral sensory signals entering by way of the vagus and glossopharyngeal nerves.

c) Sleep can be promoted by stimulation of the:

- Rostral part of the hypothalamus, mainly in the suprachiasmal area AND
- An occasional area in the diffuse nuclei of the thalamus.

Discrete lesions in the raphe nuclei leads to a high state of wakefulness. The phenomenon is also true of bilateral lesions in the medial rostral suprachiasmal area in the anterior hypothalamus.

Physiological Functions of Sleep

1) Sleep has the principal valve to restore natural balnces among the neuronal centers.

2) Facilitation of learning or memory.

3) Cognition.

4) Clearance of metabolic waste products generated by neural activity in the awake brain.

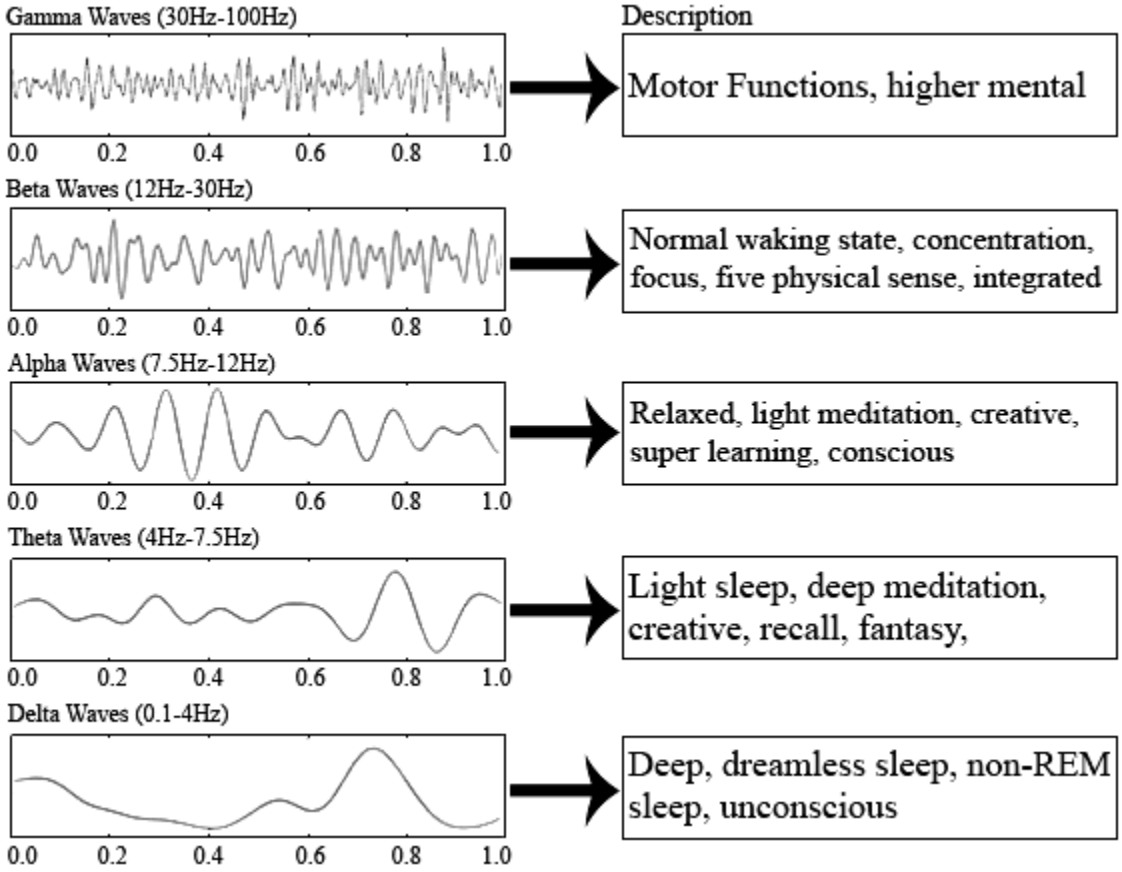
5) Conservation of metabolic energy.

BRAIN WAVES

Electrical recordings from the surface of the brain or even from the outer surface of the brain or even from the outer surface of head demonstrate that there is continuous electrical activity in the brain. Both the intensity and the patterns of this electrical activity are determined by the level of excitation of different parts of the brain resulting from sleep, wakefulness or brain disorders such as epilepsy or even psychoses.

The record of brain waves is called **Electroencephalogram (EEG)**. The intensities of brain waves recorded from the surface of the scalp range from 0 to 200 microvolts and their frequencies range from once every few seconds to 50 or more per second.

In healthy people, most waves in the EEG can be classified as alpha, beta, theta and delta waves.



i) Alpha waves: they are rhythmical waves that occur at frequencies between 8 and 13 cycles\sec and are found in the EEGs of almost all healthy adults when they are awake and in a quiet, resting state of cerebration.
These waves occur most intensely in the occipital region but can also be recorded from the parietal and frontal regions of the scalp. During deep sleep, the alpha waves disappear.

ii) Beta waves: They occur at frequencies greater than 14 cycles/sec and as high as 80 cycles/sec. They are recorded mainly from parietal and frontal regions during specific activation of these parts of the brain. Beta waves appear when the person awake attention is directed to some specific types of mental activity.

iii) Theta waves have frequencies between 4 and 7 cycles/sec. They occur normally in the parietal and temporal regions in children, but they also occur during emotional stress in some adults, particularly during disappointment and frustration. Theta waves also occur in many brain disorders, often in degenerative brain states.

iv) Delta waves include all the waves of the EEG with frequencies less than 3.5 cycles/sec, and they often have voltages two or four times greater than most other types of brain waves. They occur in very deep sleep, in infancy and in persons with serious organic brain disease.
Delta waves can occur strictly in the cortex independently of activities in lower regions of the brain.

Changes in the Electroencephalogram (EEG) at different stages of wakefulness and sleep

Alert wakefulness is characterized by high-frequency beta waves, whereas quiet wakefulness is usually associated with alpha waves. Slow-wave sleep is divide into 4 stages. In the first stage, this is a stage of light sleep, the voltage of the EEG waves becomes low. This stage is broken into sleep spindles also.

In stages 2, 3 and 4 of slow-wave sleep, the frequency of the EEG becomes progressively slower until it reaches a frequency of only one to three waves per second in Stage 4, these waves are Delta waves.

In Rapid Eye Movement sleep, in EEG, the waves are irregular and of high frequency, which are normally suggestive of desynchronized neurons activity as found in the awake state. Therefore, REM sleep is frequently called desynchronized sleep because there is lack of synchrony in the firing of neurons despite significant brain activity.

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.

CLINICALS

Some factors affects the brain activity and can lead to some abnormalities, some of these abnormalities include:

1) Seizures: They are temporary distruption of brain function caused by uncontrolled excessive neuronal activity. Depending on the distribution of neuronal discharges, seizures manifestation can range from experimental phenomena that are barely noticeable to dramatic convulsions. These temporary symptomatic seizures usually do not persist if the underlying disorder is corrected.

2) Epilepsy: this is a chronic condition of recurrent seizures that can also vary from brief and nearly undetectable symptoms to periods of vigorous shaking and convulsions.

Epilepsy clinical symptoms are heterogenous and have multiple underlying causes and pathophysiological mechanisms that causes cerebral dysfunction and injury such as trauma, tumors, infection or degenerative changes. At a basic level, an epileptic seizure is caused by a distruption of the normal balance between

inhibitory and excitatory currents or transmission in one or more regions of the brains.

Epileptic seizures can be classified into two major types:

- Focal/Partial epileptic seizures
- Generalized seizures.

1) Focal Seizure begin in a small localized region of the cerebral cortex or deeper structures of the cerebrum and brainstem and have clinical manifestations that reflect the function of the affected brain area. Most often, focal epilepsy results from some localized organic lesion or functional abnormality such as scar tissue in the brain the pulls on the adjacent neuronal tissue, a tumor that compresses an area of the brain, a destroyed area of brain tissue or congenitally deranged local circuitry.

Focal seizures can spread locally from a focus or more remotely to the contralateral cortex and subcortical areas of the brain through projections to the thalamus, which has widespread connections to both hemisphere. When such a wave of excitation spreads over the motor cortex, it causes a progressive march of muscle contractions throughout the opposite side of the body, beginning most characteristically in the mouth region and marching progressively downwards to the legs but at other times marching in the opposite direction. This phenomenon is called JACKSONIAM MARCH.

Focal seizures are often classified as simple partial when there is no major change in consciousness or as complex partial when consciousness is impaired.

2) Generalized Seizures are characterized by diffuse, excessive and uncontrolled neuronal discharges that at the onset spread rapidly and stimultaneously to both cerebral hemispheres through interconnections between the thalamus and cortex. During this seizure, often the person bites or swallows his/her tongue and may have difficulty breathing, sometimes to the extent that cyanosis occurs.

Question 2: **Discuss the role of basal ganglia in co-ordinating movement**.

The basal ganglia (or basal nuclei) are large masses of grey matter situated in the cerebral hemispheres. Like the cerebellum, constitute another accessory motor system that functions usually not by itself but in close association with the cerebral cortex and corticospinal motor control system.

Anatomically, it develops from the telencephalon and has parts:

- Caudate nucleus
- Lentiform Nucleus, which consists of 2 distinct parts, the putamen and the globus pallidus.
- Amygdaloid nuclear complex
- Claustrum

Functionally, the basal nuclei comprises of structures which include:

- Corpus striatum
- Subthalamic nucleus
- Substantia Nigra (midbrain)
- Some masses of grey matter found just below the corpus striatum are described as the Ventral Striatum.

Physiologically, on each side of the brain, these ganglia consists of the Caudate nucleus, putamen, globus pallidus, substantia nigra and subthalamic nucleus. Almost all motor and sensory nerve fibers connecting the cerebral cortex and spinal cord pass through the space that lies between the major masses of basal ganglia, the caudate nucleus and the putamen. This space is called the **Internal Capsule of the Brain.**

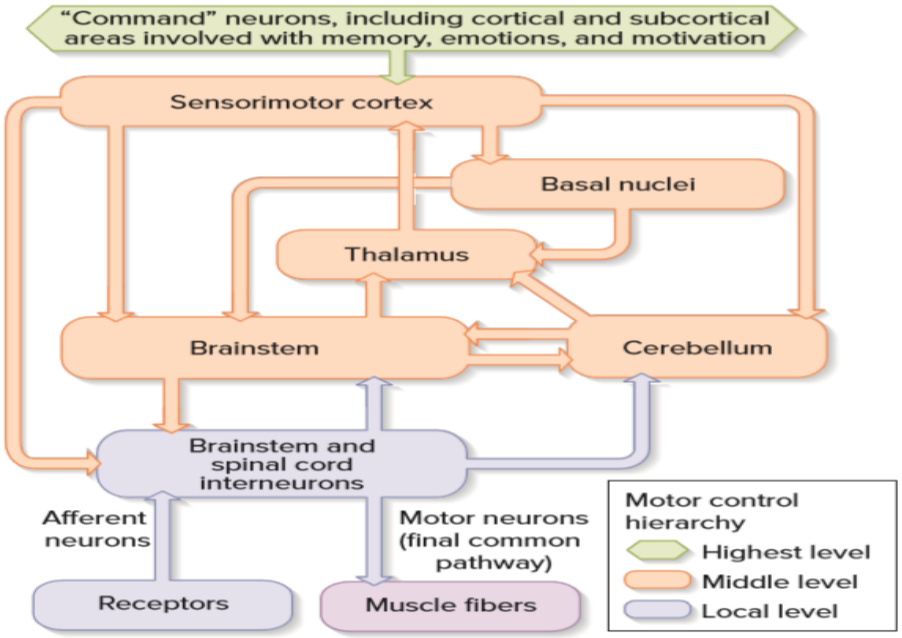
Neuronal Circuitry of the Basal Ganglia

One of the principal roles of the basal ganglia in motor control is to function in association with the corticospinal system to control complex patterns of motor activity. The idea for motor activity comes from various parts of the brain culminating in the prefrontal cortex. The prefrontal cortex orchestrates the thoughts and actions in accordance with the desired goals.

The impulses from the prefrontal cortex, along with those from emotional centers of limbic region and memory area of temporal lobe, go to the sensory association areas of the brain. The association areas project to the premotor and motor areas for the intended activity.

The premotor and motor areas activate the basal nuclei loop for postural adjustment and cerebellar loop for fine co-ordination by sending information to these centers about their motor plan. The corticospinal pathway executes the movement through the lower motor neurons.

When there is a damage to the basal ganglia, the cortical system of motor control can no longer provide these patterns.



Organizing the neural system controlling body movement

Role of the basal ganglia in co-ordinating movement.

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.

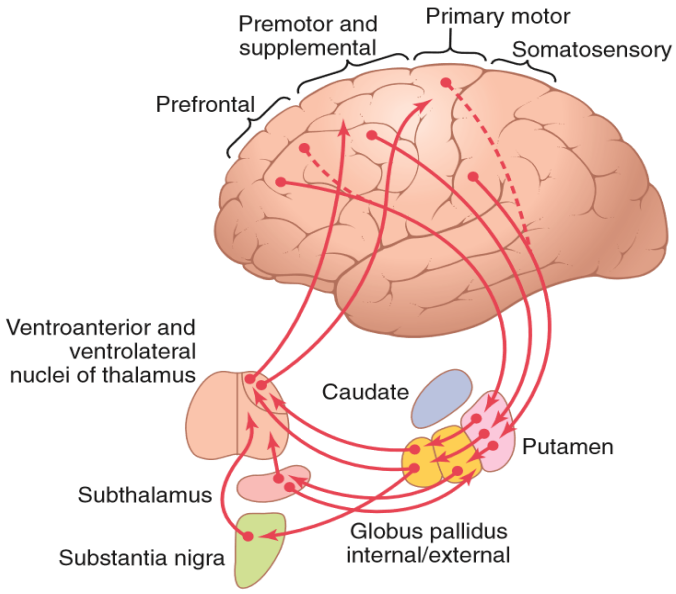


FIG. 2 Putamen circuit

- 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.

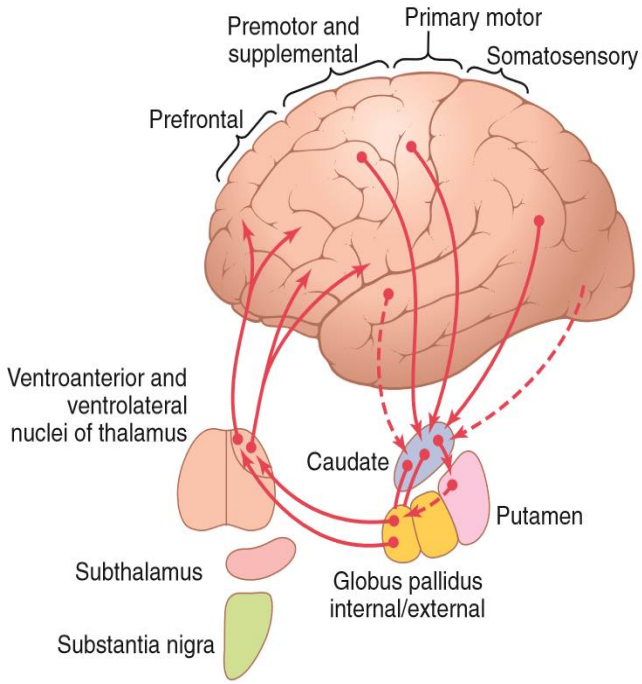


FIG. 3 Caudate circuit

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.