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**NEUROPHYSIOLOGY ASSIGNMENT**

**Question 1; Discuss the Physiology of sleep**

Far from a simple absence of wakefulness, sleep is an active, regulated, and metabolically distinct state, essential for health and well-being.

The cortical activation necessary to maintain wakefulness is supported by an extensive network of subcortical structures and pathways. Major neurochemicals of this “ascending arousal system” include excitatory norepinephrine arising from the locus ceruleus (LC), serotonin from the midline raphe nuclei, histamine from the tuberomammillary nucleus, dopamine from the ventral periacqueductal gray matter, acetylcholine from the pedunculopontine tegmentum, and the laterodorsal tegmentum of the pons and orexin from the perifornical area. Despite their apparent redundancy, normal behavioral functioning may require all of these arousing systems. For example, it is now clear that narcolepsy results from a selective loss of orexin-releasing neurons in the forebrain, accounting for the excessive daytime sleepiness, fragmented sleep, and cataplexy (sudden muscle weakness without loss of consciousness) associated with this disorder.

Initiation and maintenance of sleep require suppression of activity in the ascending arousal systems. This is accomplished by inhibitory neurons of the ventrolateral pre-optic area (VLPO) which remain active throughout sleep. The molecular “triggers” that activate the VLPO and initiate sleep onset have not been fully defined, but a substantial body of evidence points to extracellular adenosine as a candidate. Adenosine accumulates in basal forebrain during wakefulness and diminishes with ongoing sleep. Adenosine receptors are expressed in the VLPO and adenosine activates VLPO neurons in vivo making it a reasonable candidate for the “sleep switch.” Caffeine and theophylline are potent adenosine receptor antagonists, which may form the basis for their well-known alerting effects. Despite this evidence, it is almost certain that other molecules also play important signaling roles controlling the initiation and maintenance of sleep.

Sleep itself is not a homogenous process. There exist two fundamentally distinct types of sleep: rapid eye movement (REM) sleep, which is associated with active dreaming, and non–rapid eye movement (NREM) sleep. Switches between NREM and REM sleep appear to be controlled by reciprocal inhibition between monoaminergic neurons and a specific subset of cholinergic neurons within the brainstem. These “REM-on” cholinergic neurons exhibit reciprocal inhibitory connections to noradrenergic (LC) and serotonergic (raphe) neurons. When REM sleep is triggered, REM-on cholinergic neurons become maximally active, while noradrenergic and serotonergic neurons become virtually silent. The switching between activity and inhibition of these neurons results in a characteristic cycling between NREM and REM during the sleep period.

**Measurement and Quantification of Sleep and Wake States**

Assessment of sleep/wake states can be made by behavioral observation, physiological monitoring, or a combination of the two. Behaviorally, sleep in adults is characterized by loss of consciousness and by relative immobility in a recumbent posture with the eyes closed. During NREM sleep, there is reduced tonus of large skeletal muscles that progresses to complete or near-complete atonia with a transition to REM sleep. Throughout sleep, there is a relative sparing of activity among respiratory pump muscles. Visual, olfactory, auditory, somatosensory, and even nociceptive sensory responses all are diminished but not eliminated during sleep. Furthermore, many sensory responses exhibit differing characteristics during NREM versus REM sleep.

Physiologically, the gold standard for assessment of sleep and wake states is the laboratory polysomnogram (PSG). To conduct a PSG, numerous noninvasive sensors are attached to a subject. These sensors include multiple skin electrodes, which record brain activity (electroencephalogram [EEG]), eye movements, submental muscle tone, leg movements, and electrocardiogram (ECG). Thoracic and abdominal strain gauges, oral and nasal airflow sensors, and a finger probe to measure arterial oxygen saturation are also attached to the subject to help monitor respiration during sleep.

**Endocrine Manifestations of Sleep and Wake States**

Plasma levels of most hormones exhibit significant 24-hour rhythms, pointing to the importance of both the circadian clock and sleep-specific influences on their release and/or metabolism. Some hormones are little influenced by sleep versus wakefulness, including adrenocortotropic hormone, cortisol, and melatonin; some are strongly influenced by sleep, such as thyroid-stimulating hormone (TSH) and prolactin; and some are affected by particular sleep stages, such as growth hormone.

Under normal conditions, prolactin levels are low during the daytime and high during sleep at night. Studies using daytime naps or sudden changes in sleep schedule have shown that sleep onset, regardless of time of day, is associated with a stimulation of prolactin release. It has been suggested that a negative association exists between EEG delta activity and pulsatile prolactin release.

Growth hormone also exhibits a strong sleep-dependent rhythm. Cortisol also exhibits a significant 24-hour pattern, but, unlike growth hormone, this appears to be primarily a result of circadian influences rather than of sleep per se. Also in contrast to growth hormone, which peaks early during the sleep period, cortisol is at its nadir early during a nocturnal sleep period and peaks toward the end of sleep or in the early morning hours.

TSH exhibits low daytime values, increasing during the evening and peaking around the time of sleep onset.

**Question 2; Discuss the role of basal ganglia in coordinating movement**

The basal ganglia are a set of brain structures located beneath the **cerebral cortex** that receive information from the cortex, transmit it to the motor centers, and return it to the part of the cerebral cortex that is in charge of motion planning.

The basal ganglia represent a
second, auxiliary motor system that functions independently, just like the
cerebellum, and is closely related to the cerebral cortex and the
cortico-spinal motor system. The basal ganglia receive most of their input
signals from the cortex itself and also return almost all of their output
signals to the cortex.

The set of the basal ganglia consist of **nucleus caudatus**, putamen, globus palidus, nigra substance, and subtalamus nucleus. They are located mainly lateral to the thalamus, surrounding it so that they occupy large portions of the internal regions of both cerebral hemispheres.

The separate nuclei of the basal ganglia all have extensive roles of their own in the brain, but they also are interconnected with one another to form a network that is thought to be involved in a variety of cognitive, emotional, and movement-related functions. The basal ganglia are best-known, however, for their role in movement.

The contributions of the basal ganglia to movement are complex and still not completely understood. In fact, the basal ganglia probably have multiple movement-related functions, ranging from choosing actions that are likely to lead to positive consequences to avoiding things that might be aversive. But the basal ganglia are most often linked to the initiation and execution of movements. One popular hypothesis suggests that the basal ganglia act to facilitate desired movements and inhibit unwanted and/or competing movements.

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 nd 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.

According to the direct/indirect model, when a movement is desired, a signal to initiate the movement is sent from the cortex to the basal ganglia, typically arriving at the caudate or putamen (which are referred to collectively as the striatum). Then, the signal follows a circuit in the basal ganglia known as the **direct pathway**, which leads to the silencing of neurons in the globus pallidus and substantia nigra. This frees the thalamus from the inhibitory effects of the basal ganglia and allows movement to occur.

There is also a circuit within the basal ganglia called the **indirect pathway**, which involves the subthalamic nucleus and leads to the increased suppression of unwanted movements. It is thought that a balance between activity in these two pathways may facilitate smooth movement.

The greatest source of insight into the functions of the basal ganglia has come from the study of two neurological disorders, Parkinson’s disease and Huntington’s disease. For both of these disorders, the nature of the neural damage is well-understood and can be correlated with the resulting symptoms.

Parkinson’s disease involves the major loss of dopaminergic cells in the substantia nigra. Huntington’s disease involves the massive loss of medium spiny neurons in the striatum.

The symptoms of the two diseases are virtually opposite: Parkinson’s disease is characterized by a gradual loss of the ability to initiate movement, whereas Huntington’s disease is characterized by an inability to prevent parts of the body from moving unintentionally.

It is noteworthy that, although both diseases have cognitive symptoms, especially in their advanced stages, the most salient symptoms relate to the ability to initiate and control movement. Thus, both are classified primarily as movement disorders.

A different movement disorder, called hemiballismus, may result from damage restricted to the subthalamic nucleus. Hemiballismus is characterized by violent and uncontrollable flinging movements of the arms and legs.