CHANGES IN THE ELECTROENCEPHALOGRAM DURING HIGH-DOSE NARCOTIC ANAESTHESIA

ESSAY

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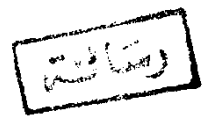

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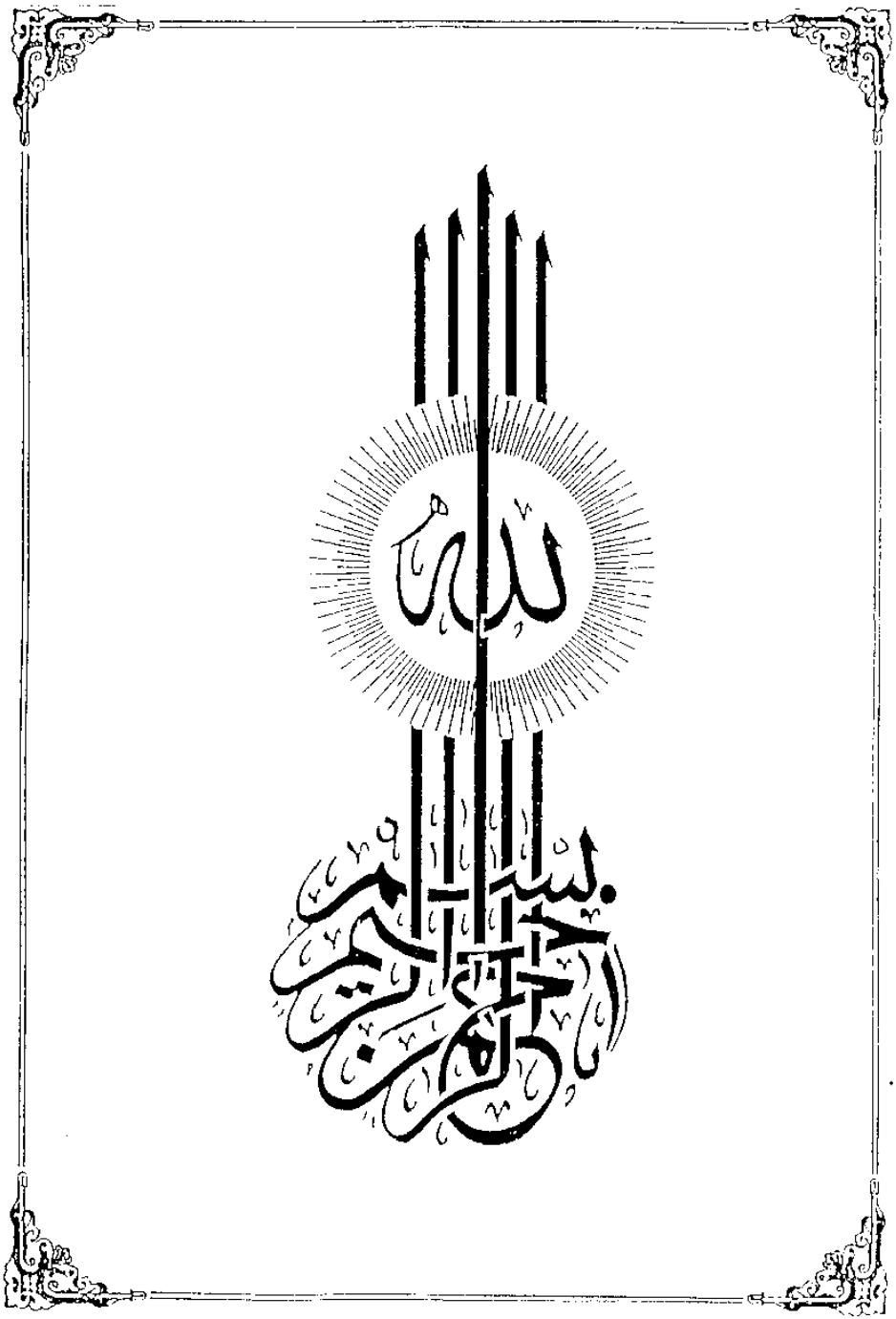

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Introduction

INTRODUCTION

The function of the brain is at the center of the anaesthesiologists professional life, yet it is remarkable how little attention has been given to it in anaesthetic research until recently.

The main reason has not been lack of interest, but the scarcity and complexity of methods for monitoring cerebral function. The EEG is commonly used as a monitor of cerebral function.

Narcotics play an important role in clinical anaesthesia. They have long been used to supplement general anaesthesia and to provide preoperative and postoperative analgesia. In more recent years they have been utilized as the primary anaesthetic agent in major surgical procedures.

It can be difficult clinically to estimate the depth of anaesthesia during high - dose narcotic - oxygen anaesthesia.

More scientific use of these narcotics along with avoidance of underdosing or overdosing could be attained if the anaesthesiologists had a sensitive, continuous, noninvasive measure of narcotic effect. Perhaps the electroencephalogram (EEG) could provide an indicator of anaesthetic depth. Compared with inhalation agents, however, there are few data available about EEG effects of high - dose narcotic anaesthesia.

Physiology of Sleep

The Reticular Activating System (RAS)

The reticular activating system controls the overall degree of the central nervous system activity, including control of wakefulness and sleep, and control of at least part of our ability to direct attention toward specific areas of our conscious mind.

It begins in the lower brain stem and extends upwards through the mesencephalon and thalamus to be distributed throughout the cerebral cortex (Ganong, 1983).

Function of the reticular activating system in wakefulness:

Diffuse electrical stimulation in the mesencephalic, pontile, and upper medullary portions of the reticular formation causes immediate and marked activation of the cerebral cortex and even causes sleeping animal to awaken instantaneously. Furthermore, when this mesencephalic portion of the reticular formation is damaged severely, the person passes into coma and is completely nonsusceptible to normal awakening stimuli (Andrew, 1974).

Function of the mesencephalic portion of the reticular formation:

Electrical stimuli applied to different portions of RAS have shown that the mesencephalic portion functions quite

differently from the thalamic portion. Electrical stimulation of the mesencephalic portion causes generalized activation of the entire brain, including activation of the cerebral cortex, thalamic nuclei, basal ganglia,hypothalamus, other portions of the brain stem, and even the spinal cord. Therefore, it is believed that the mesencephalic portion of RAS is basically responsible for wakefulness of the brain.

Norepinephrine plays a role in the wakefulness process. It has been suggested that both dopamine and epinephrine, both of which are very similar to norepinephrine, might also contribute to wakefulness because neurons in closely allied regions of the brain stem secrete these transmitter substances and seem to be activated in many instances, along with the norepinephrine system (Gillin, 1978).

Functions of the thalamic portion of RAS:

The thalamic portion of the activating system has two specific functions: First, it relays most of the diffuse facilitatory signals from the mesencephalon to all parts of the cerebral cortex to cause generalized activation of the cerebrum; Second, stimulation of selected points in the thalamic activating system causes specific activation of certain areas of the cerebral cortex in distinction to the other areas.

This selective activation of specific cortical areas probably plays an important role in our ability to direct our attention to certain parts of our mental activity (Ganong, 1983).

The arousal reaction- Activation of RAS by sensory signals

When an animal is asleep, the level of activity of the reticular activating system is greatly decreased. Yet almost any type of sensory signals, for instance, proprioceptive signals from the joints and muscles, pain impulse from the skin, visual signals from the eyes, auditory signals from the ears, or even visceral sensation, can activate RAS and therefore arouse the animal. This is called arousal reaction. Some types of sensory stimuli are more potent than others in eliciting the arousal reaction, the most potent are pain and proprioceptive somatic impulses.

Stimulation of RAS by the cerebral cortex.

In addition to activating the reticular formation by sensory signals, the cerebral cortex can also stimulate this system. Direct fiber pathways pass into the reticular formation from almost all parts of cerebrum but particularly from the sensorimotor cortex, the cingulate gyrus, the hippocampus, the hypothalamus and the basal ganglia.

Because of an exceedingly large number of nerve fibers that pass from the motor regions of the cerebral cortex to the reticular formation, motor activity in particular is associated with a high degree of wakefulness, which potentially explains the importance of movement to a person awake (Webb, 1983).

Sleep and Wakefulness

Sleep is defined as a state of unconsciousness from which a person can be aroused by appropriate sensory or other stimuli.

Therefore, the unconsciousness caused by deep anaesthesia, by total inactivity of RAS in diseased states (coma), and by excessive activity of RAS as occurs in grand mal epilepsy would not be considered to be sleep.

However, anaesthesia, sometimes described as controlled or reversible production of unconsciousness, do have many characteristics similar to those of deep sleep (Freeman, 1973).

There are two different types of sleep:

1- Slow wave sleep. (dreamless sleep)

It results from decreased activity in the reticular activating system. It is called so, because the brain waves are very slow.

Most of the sleep during each night is of the slow wave variety, this is the deep restful type of sleep that the person experiences after having been kept awake for the previous 24 to 48 hours.

It is associated with a decrease in peripheral vascular resistance and 10-30% decrease in blood pressure, respiratory rate and basal metabolic rate (Ganong, 1983).

2- Paradoxical sleep. (REM sleep)

It results from abnormal channeling of signals in the brain even though brain activity may not be significantly depressed. Therefore, it is assumed that it results from a curious mixture of activation of some brain regions while other regions are still depressed.

In a normal sleep of night, bouts of paradoxical sleep lasting 5 to 20 minutes usually appear on the average every 90 minutes, the first such period occuring 80-100 minutes after the person falls asleep. When the person is extremely tired, the duration of each bout of paradoxical sleep is very short, and it may even be absent. On the other hand as the person becomes more rested through the night, the duration of paradoxical bouts greatly increases (Winfree, 1982).

Characteristics of paradoxical sleep:

1- It is usually associated with active dreaming.
2- The person is more difficult to arouse than druing deep slow wave sleep.
3- The muscle tone throughout the body is exceedingly depressed, indicating strong inhibition of the spinal projections from RAS.

4- The heart rate and respiration usually become irregular, which is characteristic of dream state.

5- Despite the extreme inhibition of the peripheral muscles, a few irregular muscle movements occur. These include, in particular, rapid movements of the eyes, consequently, it has often been called rapid eye movement "REM" sleep.

In summary, paradoxical sleep is a type of sleep in which the brain is quite active. However the brain activity is not channeled in the proper direction for the person to be aware of his surrounding and therefore to be awake (Stern and Morgane, 1974).

Neuronal centers, transmitters, and mechanisms that can cause sleep:

Stimulation of several specific areas of the brain can produce sleep .

1- The most conspicious stimulation area for causing natural sleep is the raphe nuclei in the pons and medulla. Nerve fibers from these nuclei spread widely in the reticular formation and also upwards into the thalamus, hypothalamus, and most areas of the limbic system. In addition, they extend downwards into the spinal cord, terminating in the posterior horns where they can inhibit incoming pain signals. it is also known that endings of fibers from these raphe neurons secrete serotonin.

Therefore, it is assumed that srotonin is the major transmitter substance associated with production of sleep (Gillin , 1978).

2- The rostral part of the hypothalamus, mainly in the suprachiasmal area.

3- An occasional area in the diffuse nuclei of the thalamus.

Effect of lesions in the sleep-promoting centers:

Discrete lesions in the raphe nuclei or hypothalamus cause intense wakefulness that the animal actually dies of exhaustion (Ganong, 1983).

The cylce between sleep and wakefulness:

Neuronal center, transmitters and mechanisms are related either to wakefulness or to sleep.

However, the cyclic, reciprocal operation of the sleep-wakefulness cycle has not been explained.

It is quite possible that this is caused by a free running intrinsic oscillator within the brain stem that cycles back and forth between the sleep and wakefulness centers.

Feedback signals from the cerebral cortex and also from the peripheral nerve receptors might also play a very important role in causing the sleep-wakefulness rhythm.