

## THE PHYLOGENETIC PLANES IN ANESTHESIA\*

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THE depression of the central nervous system as seen during the progress of general anesthesia takes place in an orderly fashion. The recognition of the clinical symptoms and their organization into stages and planes were great steps forward toward understanding this complicated process (1). Further progress in this field demands a knowledge of the brain stem and the pharmacologic effect of the anesthetic agent on neurophysiology.

Jackson (2) described the phylogenetic layers of the nervous system. This concept divides the central nervous system into groups of nuclei and nerve centers which are anatomically closely situated and whose functions are on the same level of neurologic integration. When translated into present day knowledge of the neuraxis the nerve centers which control the vegetative processes such as digestion, respiration and circulation are found in the medulla oblongata and constitute the most primitive level. Next is the midbrain with the control centers of more elective processes such as chewing, swallowing, coughing and ocular motion. Above this level is the diencephalon with its sensory centers and its master nuclei for the control of the autonomic system (3). The highest level is the cerebrum which is the level of sensory perception integration and voluntary motor control. Only those centers which are of primary importance to the anesthesiologist will be discussed in this paper.

With the advent of insulin shock it became possible to study the slow breakdown of neurophysiology (4). Himwich (5) has observed that this breakdown follows very closely the phylogenetic layers; however, the progress is in the reverse order of the origin of the layers.

In his book *Brain Metabolism and Cerebral Disorders* Himwich (6) described the process in great detail. However, his study is based on physiologic breakdown induced by hypoglycemia which is equivalent to an anesthetic induction of four hours' duration. This slow deterioration is never seen in anesthesia but much can be learned from his work.

It is evident to any anesthesiologist that the depression of the neuraxis does not take place equally in all parts at the same time. As

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the process of anesthesia is reviewed definite principles become apparent.

1. An anesthetic agent does not attack the nervous system as a whole; it inactivates the phylogenetic layers of the nervous system one at a time but in the reverse order of their origin. The more primitive layers may remain functionally intact while the higher layers are more or less completely depressed.

2. A nucleus usually shows signs of stimulation before depression sets in and the probability of its stimulation depends upon the rate of onset of anesthesia. Ether stimulates most nuclei and sodium pentothal very few.

3. The reflex activity of a structure remains active after complete depression of its usual activity, and may be produced by proper stimulus while the anesthesia is in the next lower level.

4. Recovery from an anesthetic agent takes place by phylogenetic layers in the order of their origin.

With the onset of anesthesia, consciousness is first impaired as the agent attacks the cerebrum. In accord with the second principle any center of the cerebrum may show stimulation just before it is inactivated. The patient may experience visual sensations of seeing bright lights or hearing sounds. The vestibular centers many times give the sensation of floating away. Finally, the motor centers are stimulated which give expression to movements of the muscle groups as expressed in the so-called excitement stage.

With the depression of the motor cells, the entire cerebrum is inactivated to its usual physiologic state and the anesthesia moves into the second layer, that of the diencephalon. Many nuclei are found in this layer. However, the only ones of major importance to anesthesia are those of the thalamic group and the master control nuclei of the autonomic system (7). The thalamus is first activated to such an extent that skin stimulation or pain stimuli of any kind will evoke motor expression on the reflex level long after the motor cortex has lost its usual control. This may be expressed by limb movement, laryngospasm or occasional vomiting. At the rostral end of the hypothalamus is a master control center for the parasympathetic system and at the caudal end is a similar center for the sympathetic outflow (3). Of the two systems, the sympathetic is the predominant one in emergency and a general stimulation of the diencephalic layer can be expected to result in sympathetic mastery which quickens the pulse, raises blood pressure and gives wide dilation of the pupils. After the sympathetic nucleus becomes inactivated the parasympathetic center is stimulated and its effect persists through the next lower level.

The layer of the diencephalon is quickly traversed even with the slow progress of ether and the patient is under the influence of the nuclei of the mesencephalon. The jaw is in spastic closure long after

skeletal muscles are relaxed. This attests to the stimulation of the nuclei of the midbrain which controls mastication. Swallowing is encountered in this stage and the cough reflex is very active. Airways and mouth gags should not be inserted until these nuclei are obtunded.

In this level the ocular motor nuclei are stimulated. When it is first entered, the eyes rotate in the vertical plane, either up or down. Separated from the ocular motor group, located near the junction of the mesencephalon and the medulla, is the nucleus of the abducens nerve. This produces lateral rotation of the eyeball long after the vertical motion has ceased. In the ocular motor group is found the Edinger-Westphal nucleus which produces contraction of the pupil. As is characteristic of other parasympathetic outflow, the stimulation from this center persists after the other nuclei of this layer are anesthetized.

As long as the anesthetic level is in the mesencephalon the patient is subject to reflex activity, which can be initiated by a strong stimulus reaching the thalamus. When the level passes over into the medulla, however, all reflexes of higher levels are obtunded and even abdominal relaxation is accomplished.

When the level of the medulla is entered, the anesthesiologist is chiefly concerned with the cardiac and respiratory centers. The latter controls respiration. A level of anesthesia capable of overcoming the respiratory center produces apnea. If respiration is maintained artificially until the level of anesthesia becomes a little lighter, the nucleus reactivates and respiration takes place by normal muscular activity. The circulatory system is under heavy influence of the cervical sympathetic chain. An anesthetic level capable of stopping respiration probably deeply affects the cardiac center which is close by, but sympathetic support outside the medulla permits the circulation to stand a much heavier insult than respiration. Another example of cervical sympathetic support is seen in activity of the pupil. After the Edinger-Westphal nucleus is anesthetized the pupil quickly dilates by sympathetic stimulation. This is not dilation by anoxemia as sometimes stated because it is easily controlled by changing the level of anesthesia while the respiratory wave is kept constant. Ocular movement does not occur just by chance but results from efferent outflow from some nucleus which is not completely inactivated. The abducens nucleus is widely separated from the rest of the ocular motor group and lateral drift may be noted any time the level of anesthesia is at or above the junction of the medulla and the midbrain and serves as a valuable guidepost to denote those levels.

Vomiting is a reflex mechanism of major importance to the anesthesiologist. It is produced by efferent innervation from the vagus complex and its general control center is in the medulla. However, the process can be initiated many ways. Stimulation of gastric mucosa or of the mucous membrane of the unanesthetized pharynx can produce the reflex. It can be produced by the effect of apomorphine on the vomit-

ing center in the medulla but it is often set off by stimulation from the cortex of the cerebrum. It is the latter method which concerns us in anesthesia. Any given layer may exhibit its reflex activity while anesthesia is in the next lower layer. Hence, any time the anesthesia is at the level of the diencephalon, the cortex can set off the vomiting reflex. This reflex can be avoided by having anesthesia pass rapidly through the diencephalon by means of quick induction and fast recovery.

Both laryngospasm and bronchospasm are the result of efferent stimuli along the vagus channel. They can be produced by the parasympathetic effect of cyclopropane or sodium pentothal any time the vagus nucleus is intact. When the anesthesia is in the level of the mesencephalon according to the third principle, sensory stimuli may reach the thalamus and produce reflex activity resulting in spasm.

It is realized that any rule in physiology may be broken in a given patient and the neuraxis does not always perform according to given laws. The nuclei do not always fall into definite groups as demonstrated by the fact that paired left and right nuclei are not always equally depressed. This is demonstrated by finding one pupil dilated more than the other. However, observation of many cases will show that the inactivation of the nervous system by anesthesia is an orderly process following the phylogenetic planes. An appreciation of this fact helps the anesthetist to attain a better understanding of the process and guard against its complications.

#### SUMMARY

The depression of the neuraxis by general anesthesia is an orderly process and follows the sequence of the phylogenetic layers. Four principles are pointed out which help the anesthesiologist to understand this process. The use of neurophysiology is suggested as a guide in conducting general anesthesia.

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