MECHANISMS OF DISTURBANCES OF RESPIRATION AND THE CIRCULATION DURING HIGH SPINAL ANESTHESIA*

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Among the various types of anesthesia, spinal anesthesia occupies an important place. The physiological mechanisms of the respiratory and circulatory disturbances which are the principal danger of high spinal anesthesia have not been adequately studied; views regarding their pathogenesis are contradictory, and no agreement has been reached on the question of methods of their prevention and management.

The authors of the majority of modern textbooks and monographs claim that respiratory arrest and a fall in the blood pressure are the results of spread of the anesthetic solution into the fourth ventricle and of paralysis of the bulbar centers [2, 16, 21, 28, 31]. Other researchers [1, 9, 10, 22] strongly consider that the anesthetic has a paralyzing action on the peripheral motor and vasomotor conductors. V. A. Petrov [10], Babcock [22],MacIntosh [29], and others attach the main importance to the respiratory disturbances. A. F. Popova [12], S. A. Rusanov [15], and Thorek [32] consider that the fall in blood pressure is primary, and regard the respiratory arrest as the result of inadequacy of the circulation in the centers of the medulla.

In B. E. Frankenberg's opinion [20], a group of connected and mutually aggravating factors is concerned in the pathogenesis of the respiratory and circulatory disturbances during high spinal anesthesia. In anesthesia at levels up to the first thoracic segment, all the roots of the intercostal nerves are blocked, and the vasoconstrictor sympathetic innervation is interrupted. The intercostal muscles are excluded from the act of breathing, which is maintained by the diaphragm alone. The fall in the blood pressure resulting from the preganglionic block of the vasoconstrictors is aggravated by the decrease in the pumping power of the chest, by the reduction in the venous return flow to the heart , and by the fall in the systolic volume. During spread of the anesthetic to the region of the superior cervical segments, the roots of the phrenic nerves are blocked , and total respiratory arrest develops. The anoxemia which ensues under these circumstances leads to anoxia of the bulbar centers, with a subsequent secondary, catastrophic fall of the blood pressure.

Clinical observations on the course of high spinal anesthesia by B. E. Frankenberg's method [20] show that preliminary subcutaneous injection of caffeine with ephedrine, and subarachnoid injection of ephedrine, prevent or considerably minimize the primary fall in the blood pressure associated with blocking of the vasoconstrictors, but do not prevent the disturbances of respiration. In 2000 cases of spinal anesthesia performed during the last ten years, we observed respiratory arrest in 18 patients in conjunction with a stable, or even increased (above the initial level), blood pressure. The period of apnea lasted from 15 to 45 minutes. In all cases artificial respiration was applied by means of the breathing bag of the anesthetic machine. In 16 patients the blood pressure remained stable throughout the period of apnea. In two patients a fall in blood pressure was observed on account of the delayed application of artificial respiration; when this was applied, the blood pressure was restored.

The possibility of the development of apnea without a fall in the blood pressure suggests that circulatory failure in the medullary centers cannot be regarded as the principal cause of the respiratory disturbances during high spinal anesthesia. The ensuring of pulmonary ventilation by means of artificial respiration prevents a secondary fall in the blood pressure and ultimately has a decisive influence on the outcome of the spinal anesthesia.

These clinical observations led us to make an experimental study of the pathogenesis of the respiratory and circulatory disturbances during high spinal anesthesia, and of the efficacy of artificial respiration as a method of dealing with these disturbances.

EXPERIMENTAL METHOD

Experiments were carried out on dogs. The results of 95 experiments, grouped in five series, are considered.

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For the subarachnoid injection of the anesthetic, we used the two following methods: 1) the dura mater was exposed in the lumbar region by means of laminectomy, and punctured under direct vision; 2) the atlantooccipital membrane was exposed and dissected, and through an opening made in the membrane, a No. 5 ureteric catheter was passed in the caudal direction as far as the lumbosacral division, through which the solution was injected. Post-mortem examination of the skull and spine in experiments in which a colored solution was injected (methylene blue), and roentgenological examination of the spread of a contrast solution (sergosine) showed that, by the first method, the anesthetic floods the subarachnoid space of the spinal cord and fills the ventricular system; in the second case, the anesthetic escapes through the opening in the membrane and does not reach the fourth ventricle. The blood pressure was recorded from the carotid artery and respiration from the trachea (through a tracheotomy tube); the laminectomy, introduction of the catheter, tracheotomy, and manipulation of the carotid artery were carried out under morphine-ether anesthesia. The spinal anesthetic was injected after the interval which was required to bring the animal round from the general anesthesia.

EXPERIMENTAL RESULTS

In the first series of experiments (20 dogs) 4 ml of 2% procaine was injected into the spinal subarachnoid space. Irrespective of the method of injection, considerable respiratory disturbances developed, followed by a catastrophic fall of the blood pressure. Respiratory arrest in all cases preceded the cessation of the heart beat. Attempts to restore the breathing by the suboccipital injection of procaine, which, according to D. Ya. Daron [6], Flagg [24], and others, is an effective method of stimulation of the respiratory center, were unsuccessful in our experiments. Nineteen of the 20 dogs (95%) died.

In the second series of experiments (19 dogs) we studied the reaction of the respiration and circulation to direct contact of the anesthetic with the medulla. The solution was injected by suboccipital puncture. The dog lay in the prone position, with its head hanging over the operating table, forming almost a right angle, open forward, in the cervical region of the spine. Post-mortem examination showed that, when injected in this manner, the colored solution filled the system of the ventricles and did not descend into the subarachnoid space of the spinal cord.

Doses of procaine which were lethal when injected into the subarachnoid space of the spinal cord (2 ml of 4% procaine) did not lead to arrest of respiration. In contrast to the view most widely held in the literature, that the anesthetics have a paralyzing effect on the vasomotor center, a considerable rise of blood pressure was observed (Fig. 1). The subsequent suboccipital injection of caffeine brought about stimulation of respiration and a new pressor reaction. In our opinion, this result showed that the anesthetic does not block the bulbar centers and does not prevent them from reacting to the injection of stimulants. It must be emphasized that we deliberately used concentrations of procaine which were far higher than any which might reach the fourth ventricle in clinical conditions (about 0.1% according to Green [26], 0.021% according to Koster [27]).

In view of the possibility of diffusion of the anesthetic into the subarachnoid space of the skull [29], the rise of blood pressure after the suboccipital injection of the solution might be regarded as the consequence of a "chemical vagotomy," abolishing the inhibitory action of the vagus nerves on the cardiovascular system. The suboccipital injection of procaine in four experiments in association with bilateral vagotomy, however, led to a no less marked pressor reaction. We suggest that the rise in blood pressure in response to the suboccipital injection of procaine depends on the direct stimulation of the vasomotor center by the anesthetic. This agrees to some extent with the viewpoint of M. A. Bubnov [3], who produced pressor reactions by injection of procaine into the carotid artery.

The results of the first two series of experiments thus suggest that respiratory arrest and the fall in blood pressure during high spinal anesthesia are the result of the action of the anesthetic on the peripheral conductors and not on the bulbar centers.

In the third series of experiments (17 dogs) we injected 4-5 ml of 2% procaine into the subarachnoid space of the spinal cord (a dose which was lethal to 95% of the dogs of the first series), and on the appearance of respiratory disturbances, the animal was changed over to artificial respiration by means of the usual apparatus used in the physiological laboratory. Sixteen of the 17 dogs in this series survived. The application of artificial respiration resulted in stabilization of the blood pressure and prevented death of the experimental animal.

In the fourth series of experiments (20 dogs) the same usually lethal dose was injected along the catheter into the subarachnoid space. On the first signs of impending respiratory arrest, electrophrenic respiration was applied [17, 23, 30]. In 17 experiments we used an apparatus for electrical stimulation of the phrenic nerves constructed in the workshops of the Leningrad State Postgraduate Medical Institute (GIDUV), and, in three experiments, an improved apparatus supplied to us for testing by the Moscow experimental factory of the All-Union Research Institute of Medical Instruments and Apparatus of the Ministry of Health of the USSR [19]. Rhythmic stimulation of the motor points of the phrenic nerve in the skin of the neck in all cases led to rhythmic contraction of the diaphragm—contraction of a given depth and frequency. We used rhythmic interrupted impulses of current with a frequency of 50 to 80

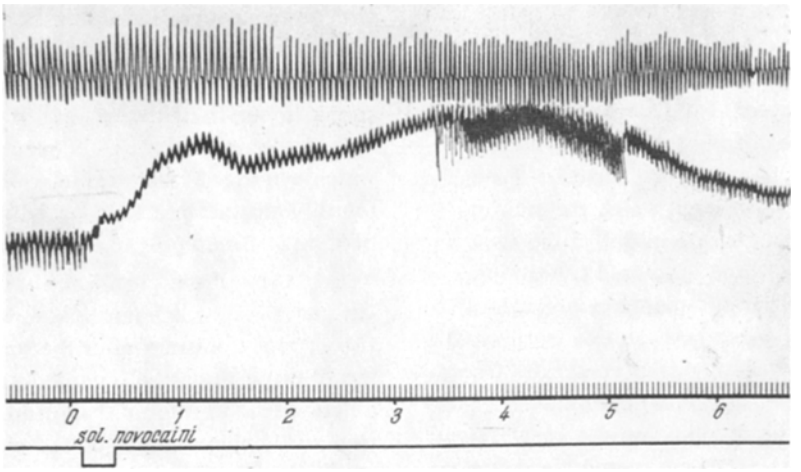

Fig. 1. Recording of the respiration and blood pressure after the suboccipital injection of 2 ml of a 4% procaine solution. Time marker – 3 seconds.

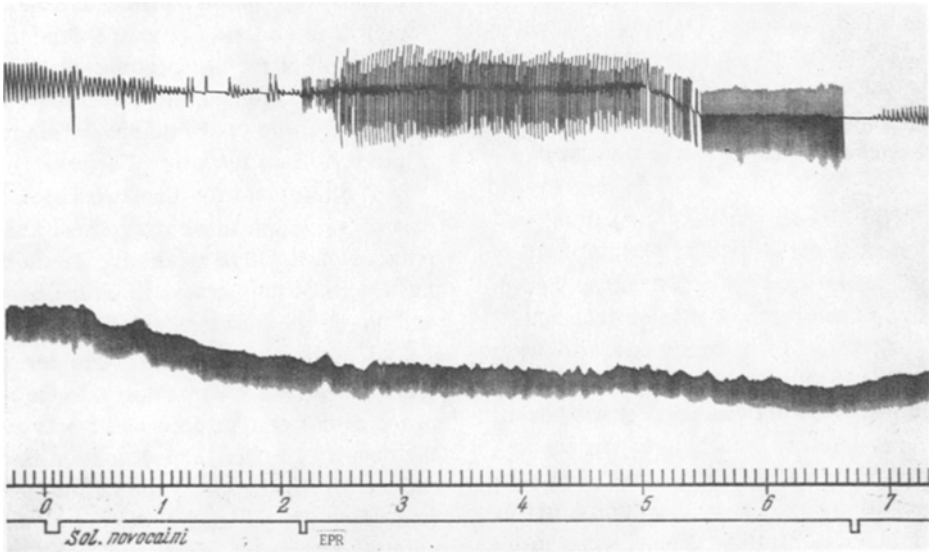

Fig. 2. Recording of respiration and blood pressure after subarachnoid injection of 4 ml of a 2% procaine solution into the lumbar division, followed by the use of electrophrenic respiration (at the third minute). At the eighth minute spontaneous respiration was restored. Time marker – 5 seconds.

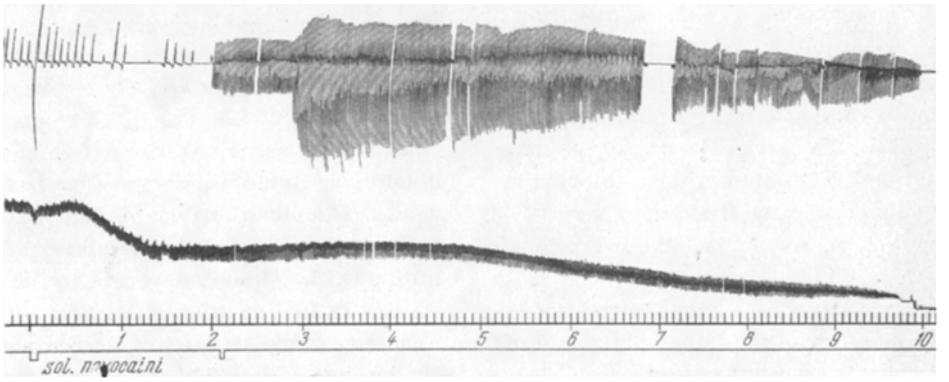

Fig. 3. Recording of the respiration and blood pressure after the subarachnoid injection of 4 ml of a 2% procaine solution into the lumbar division after bilateral vagotomy. At the third minute the animal was changed over to electrophrenic respiration, but spontaneous breathing was not restored. Time marker – 5 seconds. The second stimulation marker corresponds to the application of EPR.

cps and a voltage of 2.5 to 7.0 v at the poles of the electrodes.

Electrophrenic respiration in all the experiments led to stabilization of the blood pressure and to the restoration of spontaneous breathing (Fig. 2). All the dogs of this series survived.

In order to define more precisely the mechanisms involved in the restoration of the vital functions under the influence of artificial respiration, at Prof. R. O. Faitel'berg's suggestion, we carried out a series of experiments (19 dogs) in which the vagus nerves were divided. These nerves are known not to be blocked by even the highest spinal anesthesia [7, 8, 13]. After unilateral vagotomy, artificial respiration led to the restoration of spontaneous breathing in nine of the ten experimental animals. With bilateral vagotomy, artificial respiration was ineffective (Fig. 3). All nine dogs died.

Afferent impulses from the receptors of the respiratory system play a considerable part in the regulation of respiration [4, 11, 14, 18]. These impulses are conducted along the vagus nerves. The combination of high spinal anesthesia with bilateral vagotomy essentially amounts to complete isolation of the respiratory center from afferent impulses, which leads to the development of a state of atony of the center. Artificial respiration after bilateral vagotomy does not restore the flow of afferent impulses from the receptors of the respiratory system and does not overcome the atonic state of the center. The mechanically induced ventilation and gas exchange is insufficient for restoration of the vital functions. We attribute the death of the vagotomized animals to the exclusion of the reflex stimulating role of the artificial respiration.

SUMMARY

Respiratory and circulatory disturbances during high spinal anesthesia were made the subject of an experimental study. The dominating role of respiratory disturbances results from the action of the anesthetic upon the peripheral conductors. Direct contact of the anesthetic with the medulla oblongata not only fails to induce bulbar paralysis, but acts as an excitant on the vasomotor center. The maintenance of respiratory functions by artificial respiration is of decisive significance for the outcome of the spinal anesthesia.

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