

CHANGES IN THE RHEOGRAM OF THE BRAIN AND SKELETAL MUSCLES DURING LETHAL BLOOD LOSS

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Lethal blood loss was produced in 19 acute experiments on cats under urethane anesthesia; during its course the arterial pressure, respiration, temperature, ECG, and rheogram of the cerebral cortex, diencephalon, and muscles were recorded. Disturbances of the hemodynamics in the muscle tissue were more marked than in the brain. Less severe changes in the circulation were observed in the diencephalon.

Convincing results have now been obtained [9-11, 19, 20] to show the varied character of circulatory disturbances in different parts of the vascular system in the various stages of shock and blood loss, whether possessing common or specific mechanisms [4]. Disturbances of the circulation of blood in the CNS play a particularly important role in the pathogenesis of these disorders [3, 8, 14]. According to some authorities [1, 2], the response of the cerebral vessels is similar in character to the disturbance of the peripheral circulation, while according to others [3, 17] it is opposite in direction and takes place in stages [9, 12]. Changes in the blood volume of the skeletal muscles in hemorrhagic collapse are also reflected by conflicting data [5, 15, 18].

The object of the present investigation was to study the rheographic indices reflecting changes in the circulation in different parts of the brain during lethal blood loss under experimental conditions. In view of reports [6, 11] that changes in the cerebral circulation are best compared with changes in the circulation at the periphery, the rheograms of skeletal muscles also were recorded.

EXPERIMENTAL METHOD

Nineteen experiments were carried out on cats of the same sex but of different litters, of approximately the same age and weight, anesthetized with urethane (1.2 g/kg, intraperitoneally). Lethal blood loss was induced by rapid bleeding from the femoral artery. To determine the functional state of the animals in the course of the experiment the pressure in the femoral artery, external respiration, and the rectal temperature (using an electric thermometer) were recorded. The rheogram (RG) of the parietal lobe of the cortex and of the thalamic region of the diencephalon was recorded in a screened chamber by means of the RG-1-01 rheograph (Hungary) using platinum electrodes with a working surface of 2 mm, inserted into the brain through burr-holes and secured with acrylic glue. The accuracy of insertion of the electrodes was verified after autopsy at the end of the experiment. The rheomyogram was recorded with needle electrodes with the same working surface. Recordings were made on a four-channel 4EEG-1 electroencephalograph, and the ECG was recorded simultaneously in one channel of the instrument in the standard lead.

For analysis of the RG the technique described by Yarullin [13] was used. The time t from the Q wave of the ECG to the beginning of the RG wave, the duration of the anacrotic (t_a) and catacrotic (t_x) phases, the ratio between the anacrotic phase and the duration of the complete wave (t_x), the angle of rise of the anacrotic wave, and the amplitude and area of the rheographic complex were determined.

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EXPERIMENTAL RESULTS

The initial arterial pressure in the cats was 103 ± 2 mm. The volume of the lethal blood loss under urethane anesthesia depended on the individual reactivity of the animals. In 11 cats it was 20% of the blood mass, and in 8 animals 40%. After bleeding the typical picture of severe hemorrhagic collapse developed: hypotension, hypothermia, disturbance of breathing. Depending on the volume of blood loss the animals survived for 20 ± 5 and 10 ± 4 min.

During the first minutes after 20% blood loss the cortical RG showed a decrease in the duration of the anacrotic and an increase in the duration of the catacrotic phases as well as a reduction of the inter-phase coefficient. The amplitude and area of the RG also were reduced. After 10 and 20 min, in the period of severe hypotension (35 and 20 mm, respectively) the temporal characteristics of the RG phases were restored but the complex itself was delayed and the angle of rise of the anacrotic wave reduced, while the amplitude and area of the RG remained low. The appearance of the RG signal in the diencephalon was accelerated, while the change in the temporal and amplitude parameters of the phases of the RG complex was much less marked than on the cortical RG. In particular, the decrease in area of the RG was reduced by half, and by the end of the experiment it was the same as on the cortical RG at the beginning of the experiment. During the development of blood loss, the appearance of the complex in the muscle tissue was delayed, and the angle of slope, the amplitude, and the area of the RG were progressively reduced. By the middle of the experiment the area of RG was reduced by the same extent as the area of the cortical RG by the end of the experiment, and the fall continued subsequently.

After 40% blood loss the acceleration of the anacrotic phase was absent on the cortical RG, the inter-phase coefficient was reduced by a greater degree and was not restored, while the decrease in area of RG was less marked. Acceleration of the RG complex in the diencephalon was more marked, and it was replaced by retardation only at the end of the experiment. Nearly all the temporal indices of the RG remained at their initial level and the decrease in amplitude of RG was less marked. In the muscle tissue, by contrast with the brain tissue, the decrease in amplitude and area of the RG reached their maximum during the first minutes of the experiment with severe blood loss.

These results are in agreement with data in the literature on the development of functional occlusion of the main vessels of the brain [7, 8], a decrease in the blood flow in the carotid artery [16], and a slower fall of pressure in the circle of Willis than of the systemic arterial pressure [5, 7, 8] during blood loss.

The results obtained by rheography thus show that during blood loss the brain and, in particular, the diencephalon is better supplied with blood than the skeletal muscles. The redistribution of blood is not only interorganic but also intraorganic in character, and it is aimed at ensuring a blood supply to the most important structures of the brain.

LITERATURE CITED

1. B. N. Klovskii, The Circulation of Blood in the Brain [in Russian], Moscow (1951).
2. A. A. Kedrov and A. I. Naumenko, Problems in the Physiology of the Intracranial Circulation and Their Clinical Interpretation [in Russian], Leningrad (1954).
3. V. K. Kulagin, Data on the Pathogenesis and Therapy of Traumatic Shock, Doctoral Dissertation, Leningrad (1961).
4. V. K. Kulagin, in: Shock and Collapse [in Russian], Kishinev (1970), p. 108.
5. V. B. Koziner, Pat. Fiziol., No. 1, 11 (1965).
6. Yu. M. Levin, The Regional Circulation during Lethal Blood Loss and Subsequent Resuscitation (under Experimental Conditions). Author's Abstract of Doctoral Dissertation, Frunze (1965).
7. G. I. Mchedlishvili, Fiziol. Zh. SSSR, 46, 1210 (1960).
8. G. I. Mchedlishvili, Function of the Vascular Mechanisms of the Brain [in Russian], Moscow (1968).
9. I. R. Petrov and G. Sh. Vasadze, Irreversible Changes during Shock and Blood Loss [in Russian], Leningrad (1966).
10. S. A. Seleznev, The Liver in Traumatic Shock. Doctoral Dissertation, Leningrad (1964).
11. S. A. Seleznev, in: Shock and Collapse [in Russian], Kishinev (1970), p. 171.
12. O. P. Khrabrova, Pat. Fiziol., No. 1, 58 (1969).
13. Kh. Kh. Yarullin, Clinical Rheoencephalography [in Russian], Leningrad (1967).
14. H. D. Green and C. E. Rapela, in: Microcirculation as Related to Shock, New York (1968).
15. D. F. J. Halmagyi et al., J. Appl. Physiol., 27, 508 (1969).

16. D. B. Hinshaw et al., *Am. J. Surg.*, 102, 224 (1961).
17. S. S. Kety and C. F. Schmidt, *J. Clin. Invest.*, 27, 476 (1948).
18. S. Mellander and H. David, *Circulat. Res.*, 13, 105 (1963).
19. C. I. Wiggers, *Physiology of Shock*, New York (1950).
20. B. Zweifach, *Functional Behavior of the Microcirculation*, Springfield (1961).