PATHOGENESIS OF HYPERTENSION

PART II. EFFECT OF INHIBITION OF THE CENTRAL NERVOUS SYSTEM ON THE DEVELOPMENT AND COURSE OF EXPERIMENTAL REFLEXOGENIC HYPERTENSION

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Having established the way in which hypertension develops in normal animals after anesthetization of the carotid zone and the aortal arch [1], we proceeded to the study of its development in animals in a state of inhibition of the central nervous system due to the action of narcotic substances, and to the action of bromides.

Ether narcosis, which acts chiefly on the cerebral cortex, has a considerable effect on the development of hypertension. Anesthetization with 2% procaine of the carotid sinuses and vagus nerves leads in most cases to a rise in blood pressure of the ether anesthetized animals of 30.9% (mean value); this rise is about 3 times smaller than in conscious animals (Fig. 1), and the pressure falls to the initial value within 3-7 minutes. In some cases the blood pressure falls by 20-30 mm during anesthetization of the carotid sinuses and depressor nerves, and subsequently not only does not rise, but falls even further. In a few cases the fall in blood pressure takes place during the first 2-5 minutes after beginning anesthetization, and respiration ceases when the pressure falls to 50-60 mm, after which it falls abruptly, and the animal dies.

Such fatal outcomes, which are due to primary arrest of respiration, were also encountered in those cases in which there was an initial rise in blood pressure (Fig. 2). In such cases blood pressure fell to zero, usually after 6-10 minutes, which is evidence of impairment of adaptive functions of the organism, performed by the cerebral cortex.

Impairment of adaptability of the organism, and the dependence of the changes in blood pressure on the functional state of the cerebral cortex, were demonstrated by experiments in which slight deepening of the level of ether anesthesia caused lowering of blood pressure with, in some cases, death of the animal from primary respiratory arrest. Blood pressure rose with lightening of ether anesthesia in animals with procaine anesthetized carotid sinuses and aortic arch.

It may be concluded that the onset of a sleepy state of inhibition of the cerebral cortex promotes development of reflex hypertension, whereas deeper narcosis suppresses its development.

In the next series of experiments (17 dogs), we studied the effect of sleep inhibition, induced by Hexenal, which acts chiefly on the subcortical formations. Narcosis was induced by intravenous injection of 10% Hexenal, and hypertension by the procedures described above.

In some cases (Experiments Nos. 3, 5, 7, 9, 11, 12, 13), procainization of the depressor nerves and sinuses caused a rise in blood pressure, by from 21.9 to 34.1% in Experiments 7, 9, 11, and 12, and by 4.4 to 16.7% in Experiments 3, 5, and 13; the raised level lasted for from 5 to 30 minutes. In other cases (Experiments Nos. 1, 8, 14, 15, 16, and 17), the blood pressure rose very slightly for a short time, without regaining the initial level before injection of Hexenal. In a number of experiments the blood pressure thereafter fell below the initial level (Fig. 3).

Finally, in some animals, anesthetization of the depressor and sinus nerves (Experiments Nos. 2, 4, 6, and 10), not only did not cause development of hypertension but, on the contrary, led to a steep fall in blood pressure, respiratory arrest, and death.

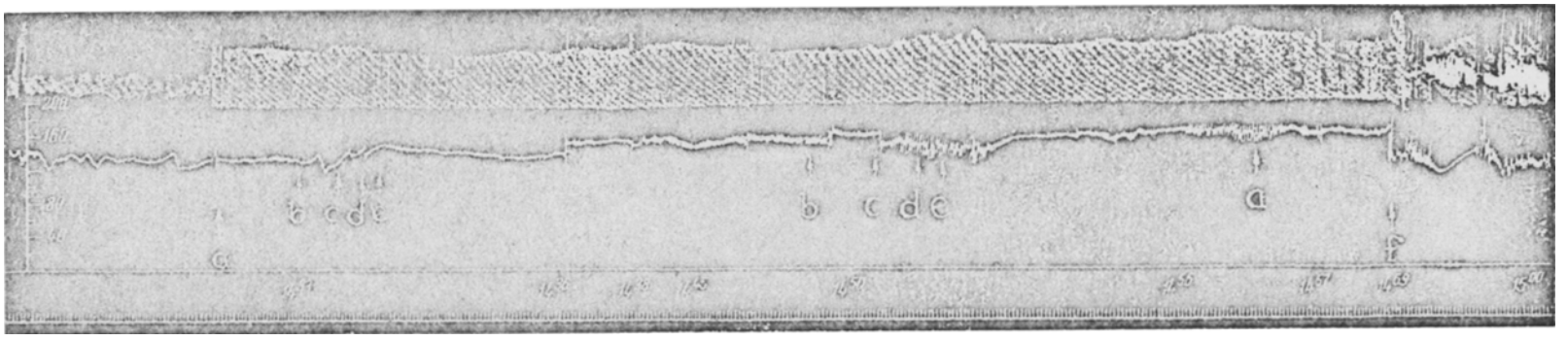

Fig. 1. Development of course of reflexogenic hypertension in dogs under ether anesthesia. Explanation of tracings, for all the figures (from above down): respiration, blood pressure, zero line, time marker (2 seconds). a) Ether. Procaine: b) right c) left sinuses; d) right e) left vagus nerves. f) Dog regains consciousness.

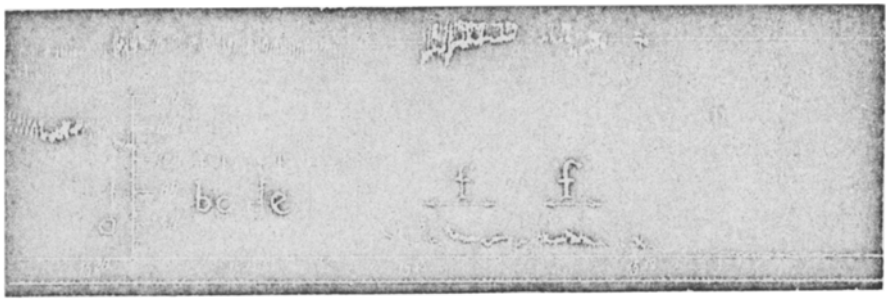

Fig. 2. Changes in respiration and blood pressure following procaine anesthetization of the carotid sinuses and depressors of a dog under anesthesia, in an experiment terminated by the death of the animal. a) Ether. Procaine: b) right c) left sinuses; d) right e) left vagus nerves. f) Artificial respiration.

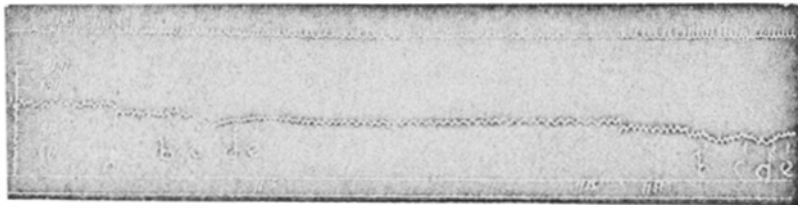

Fig. 3. Effect of Hexenal narcosis on development and course of reflexogenic hypertension. a) Narcosis. Procaine: b) right c) left sinuses; d) right e) left vagus nerves.

Effect of Previous Administration of Bromide on the Changes in Blood Pressure Caused by Application of Procaine to the Reflexogenic Zones

No. of experiment	Blood pressure (mm)				
	before administration of bromide	after the last injection of bromide	time after application of procaine		
			2 minutes	5 minutes	10 minutes
1	180	156	162	160	152
2	176	160	150	146	148
3	162	160	156	162	158
4	146	120	122	130	134
5	170	148	160	162	154
6	180	164	170	180	150
7	160	126	138	104	100
8	186	170	164	160	162
9	134	130	126	136	118
10	180	200	226	220	190
11	160	138	142	134	126

Repeated procainization, performed in Experiments Nos. 3, 5, 7, 8, 12, 13, 14, and 15, raised the blood pressure of those animals only in which the first application had this effect.

In the last series of experiments we studied the effect on development of hypertension of bromide, which raises the tonus of inhibitory processes in the cerebral cortex.

In one group of dogs of this series (12 experiments) we gave 5 or 6 intravenous injections of 10 or 20% sodium bromide, 1-1 $^{1}/_{2}$ hours before procainization of the reflexogenic zones; the blood pressure fell by 10-15 mm.

The results of these experiments are presented in the table; they show that development of hypertension in animals under the influence of bromide differs from that found in animals not receiving bromide [1].

Similar results were obtained in experiments in which the sodium bromide solution was administered subcutaneously for 3 days, with a final injection on the 4th day, 30-40 minutes before the operation.

Procainization of the carotid zones and aortal depressors of animals under the influence of bromide causes a slight, transient rise in blood pressure, followed by pressures lower than the initial ones.

The results of our experiments leave no doubt that ether and Hexenal narcosis, as well as administration of bromide, can modify the development of acute reflex hypertension. It may be concluded that the development of this form of hypertension depends on the state of inhibitory processes in the central nervous system; enhancement of this state by various means protects the animals from development of acute hypertension.

LITERATURE CITED

[1] A. N. Gordienko, V. I. Kiseleva, R. B. Tsynkalovsky and B. A. Saakov, Byull. exptl. Biol. i Med. 11, No. 2, pp. 27-30 (1956).(T.p. 125). *

* T.p. = C. B. Translation pagination.