ROLE OF REGIONS OF THE HYPOTHALAMUS IN THE DEVELOPMENT AND COURSE OF REFLEXOGENIC HYPERTENSION

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The intensity of the pressor effect during reflexogenic hypertension in dogs produced against the background of preliminary coagulation of the hypothalamus depends on the level of injury: the higher the lesion,the less marked its effect on the hypertensive response. Introduction of electrodes without coagulation of the brain substance has no effect on the development of hypertension.

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The hypothalamus is the relay station for afferent connections of the cardio-vascular system on their way to the vasomotor region of the cortex [10, 13, 14] and also an autonomic collector of vascular reactions [4, 9]. It has now been shown that selective changes in the functional state of different parts of the hypothalamus plays an important role in elevation of the arterial pressure [5, 7, 8, 11, 12].

The object of the present investigation was to study the role of various parts of the hypothalamus in the mechanism of reflexogenic hypertension.

EXPERIMENTAL METHOD

Reflexogenic hypertension was produced by application of procaine to the principal reflexogenic zones by the method described by Gordienko and co-workers [1]. Experiments were carried out on 33 male dogs, of which 14 were controls (electrodes inserted into various parts of the hypothalamus but without subsequent coagulation). Different parts (anterior, middle, and posterior) of the hypothalamus were destroyed by the use of specially designed electrodes introduced by means of a stereotactic apparatus. Bipolar coagulation of the hypothalamus was produced by a high-frequency current applied to the electrode from an ÉN-57 apparatus. The arterial pressure in the femoral artery was recorded on a kymograph. The area of brain damage was localized macroscopically and histologically.

EXPERIMENTAL RESULTS

The results are given in Table 1. Anesthesia of the reflexogenic zones in the animals of the control group was accompanied by a sharp increase in arterial pressure for 10-15 min, returning to normal after 25 min. However, between the 45th and 105th minutes the pressure fell, returning to its initial level after 165 min.

Anesthesia of the reflexogenic zones in animals with preliminary coagulation, just as in the control experiments, was accompanied by the development of reflexogenic hypertension, but the degree of elevation of the arterial pressure was directly dependent on the level of coagulation. After destruction of the middle part, it was less marked than after destruction of the anterior hypothalamus, and less marked after destruction of the posterior than of the middle part. The arterial pressure of these animals returned to its initial level after 15-25 min. No further decrease of pressure was found after coagulation of the anterior or middle hypothalamus, as in the control series, but after destruction of the posterior hypothalamus the pressure fell in the period from the 45th to 75th minutes after anesthesia. By the end of the experiments the normal arterial pressure was restored in all investigations.

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TABLE 1. Effect of Coagulation of Different Parts of Hypothalamus on Arterial Pressure (in mm Hg) in Reflexogenic Hypertension (M ± m)

Time of investigation (in min)		Anterior hypothalamus		Middle hypothalamus		Posterior hypothalamus	
		insertion of electrodes	coagulation	insertion of electrodes	coagulation	insertion of electrodes	coagulation
Initial background		143±2,6	150±2,5	143±3,8	151±3,2	149±3,8	143±3,0
Anesthesia of reflexogenic zones after	1	286±8,5 0,001	287±2,3 0,001	272±13,0 0,001	240±6,1 0,001	284±4,8 0,001	226±5,3 0,001
	5	240±8,8 0,001	233±9,2 0,001	222±13,0 0,001	207±5,9 0,001	214±6,6 0,001	208±6,7 0,001
	10	180±4,0 0,001	184±4,8 0,001	173±1,0 0,001	161±2,4 0,05	178±5,8 0,01	191±3,9 0,001
	15	156±3,0 0,02	162±3,1 0,02	154±3,9 0,06	155±2,2 0,3	159±3,3 0,13	173±3,6 0,001
	25	145±1,3 0,5	151±2,8 0,7	140±3,2 0,4	146±2,3 0,2	145±2,8 0,4	139±4,0 0,18
	45	119±2,8 0,001	144±2,8 0,1	124±3,3 0,007	144±3,1 0,18	123±3,9 0,005	114±2,6 0,001
	75	118±2,5 0,001	142±2,9 0,07	118±1,1 0,001	144±2,0 0,1	113±5,13 0,003	111±1,1 0,001
	105	123±2,0 0,002	143±3,0 0,09	122±1,8 0,003	144±2,1 0,1	115±4,3 0,004	107±3,1 0,001
	165	143±4,5 0,6	148±2,9 0,6	143±4,5 1	151±2,2 0,1	140±1,1 0,3	138±3,9 0,1

The results thus indicate that different parts of the hypothalamus have roles of different importance in the mechanism of the hypertensive response, injury at a higher level being accompanied by a less marked pressor response to blocking of the principal reflexogenic zones. The hypertensive reaction after destruction of the anterior hypothalamus may perhaps be associated with mobilization of biologically active vasopressor substances from its neurosecretory nuclei [2], and after destruction of the posterior hypothalamus, with activation of adrenergic structures located in this region [3].

Introduction of electrodes without coagulation of the brain substance had no effect on the development of the hypertensive response, which was identical in intensity with that in intact animals after application of procaine to the reflexogenic zones [6].

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