Effect of the Carotid Body Excision on the Development of Corazol-Induced Seizures

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Bilateral glomectomy in albino rats produced protective effect against the development of corazol seizures, which was manifested in prolonged latency of seizures and a 2-fold shortening of their total duration. Acute hypoxia led to shortening of the latency both in glomectomized and in sham-operated rats and significantly decreased the number of seizures in glomectomized rats, although a similar decrease in sham-operated rats was insignificant. Possible participation of sinocarotid reflexogenic zones in hypoxic protection against experimental seizures is discussed.

Key Words: *corazol seizures; hypoxia; carotid glomectomy*

Pathogenic therapy of epilepsy is aimed at annihilating a pathological system by activation of natural resources and reflex regulatory mechanisms. Considerable attention has been focused on the possibility of inhibiting the activity of diverse functional systems inducing various disturbances in the central nervous system (CNS) by activation of the "anti-systems" [9]. This approach is based on natural functional antagonism between specific structures in the CNS. Some cerebral structures whose stimulation inhibits convulsive activity have been identified. There is evidence that "anti-systems" can be activated not only by electrical stimulation or pharmacological treatment, but also by hypoxia. Adaptation to hypoxia enhances the resistance to seizures [3,10] and decreases the number of fatal outcomes during the fits [8]. Both chronic and acute hypoxia produce protective effect against corazol seizures [4,6]. Our aim was to study a possible participation of sinocarotid reflexogenic zones in the realization of the protective effect of acute hypoxia during the development of experimental seizures.

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MATERIALS AND METHODS

The study was carried out on 46 male albino rats weighing 220-240 g. In the first series of experiments, which was carried out on day 14 after bilateral carotid body excision (glomectomy) [7] or sham operation, the following parameters of convulsive activity induced by a single subcutaneous injection of corazol (60 mg/kg) were measured in 11 glomectomized (GE) and 15 sham-operated (SO) rats: latent period (LP) of the first seizure, duration of a single seizure, number of seizures, and total duration of all seizures. In the second series of experiments, these parameters were measured on the same postoperation day for convulsive activity provoked by analogous injection of corazol in 10 GE and 10 SO rats kept under acute hypoxia in an altitude chamber with Po_2=110 mm Hg (3200 m above the sea level).

RESULTS

Injection of corazol under normoxia revealed a clear tendency to prolongation of LP and significant shortening of the total duration of seizures in GE rats as compared to SO rats: while the number of seizures was the same in both groups, the duration

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TABLE 1. Indices of Convulsive Activity after Corazol Injection in GE and SO rats under Normoxic and Hypoxic Conditions (*M±m*)

Experimental conditions		LP, sec	Single seizure duration, sec	Number of seizures	Total duration of seizures, sec
Normoxia	GE rats	794±205	69±19*	1.82±0.18	126±38*
	SO rats	630±167	138±39	1.87±0.21	260±72
Hypoxia	GE rats	640±216	31±8*	1.25±0.16*	39±10
	SO rats	474±325	74±21	1.56±0.24	116±43

Note. $p<0.05$: *compared with normoxia; +compared with SO rats.

of a single seizure and the total duration of convulsions were halved after glomectomy (Table 1). Both in GE and SO rats, injection of corazol under hypoxia produced convulsions with a similar LP which were shortened, respectively, by 154 and 156 sec. In comparison with normoxic rats, injection of corazol in the hypoxic rats caused the same decrease (by 2 times) in duration of a single seizure in both groups, a significant decrease in the number of seizures in GE rats, and a tendency to a decrease in the number of seizures in SO rats. This difference resulted in a more pronounced decrease in the total duration of convulsions in GE rats (by 3.2 times) than in SO rats (by 2.2 times). Therefore, a distinctive feature of corazol seizures in glomectomized rats is a significant shortening of a single seizure both in normoxic or hypoxic conditions.

The data on the effects of various pathological states of the cardiovascular system on induction and development of epilepsy raised the problem of elucidating the role of vascular reflex factors in the genesis of this disease [12]. Multiple attempts to reveal the physiological role of carotid receptors in the development of seizures preceded the comprehensive investigation of this reflexogenic zone. The sinocarotid reflexes were found both to inhibit and potentiate the epileptic activity, depending on the parameters of electrical stimulation of the sinocarotid nerve [11]. Taking into consideration the potentiation of epileptic attack by the carotid receptor hypersensitivity, some authors successfully used chemical and surgery denervation of these receptors in various forms of epilepsy [5]. Since the maintenance in the altitude chamber raises seizure threshold and LP, and moreover, shortens the seizures and moderates their severity [3], our data on prolongation of LP of corazol seizures and on shortening of seizures in rats 2 weeks after carotid glomectomy may be explained by the effect of hypoxemia appearing after the surgery [1,2,13]. In other words, during the postoperative period GE rats are subjected to hypoxic "training" before corazol injection. The fact that acute hypoxia inhibits seizures both in GE and SO rats as in intact animals [4] indicates that it is realized not through the sinocarotid reflexes, but through other structures. The anticonvulsive effect of acute hypoxia may be related to the inhibition of cerebral electrical activity or to the activation of anti-epileptic cerebral structures. It would be instructive to compare the data on the duration of corazol-induced seizures in normoxic GE rats and in hypoxic SO rats. Further study is necessary to elucidate the mechanisms underlying this similarity.

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