Neurogenic Vasoconstriction of Pial Arterial Vessels of Various Branching Orders in Normotensive and Spontaneously Hypertensive Rats

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Intravital television microscopy was employed to study the reaction of pial arteries in normotensive and spontaneously hypertensive rats to stimulation of the superior cervical ganglion. The amplitude of constrictor response was similar in WKY and SHR animals. Under conditions of normal arterial pressure and arterial hypertension, the maximum constrictor effect was attained due to equal involvement of the arteries from all generations in the normotensive rats and predominant constriction of the precortical arterioles in SHR animals.

Key Words: *pial arterial vessels; neurogenic vasoconstriction; spontaneously hypertensive rats*

The data obtained in the study of the responses of various parts of the pial vasculature to identical stimuli [1] showed that changes in vessel diameter downstream their successive branching can be caused by the dominating influence of one or another mechanism, which control the vascular tone at the level of a single vascular generation. It can be hypothesized that the changes of vascular reactions observed under various pathological conditions in cardiovascular system are underlain by redistribution of the roles of these control mechanisms in various vascular generations.

Our aim was to examine the degree of neurogenic control of vascular tone in various segments of the artery-arteriole generations of pial vascular network under normal conditions and during arterial hypertension. We used Wistar-Kyoto (WKY) and SHR rats to compare the degree of neurogenic vasoconstriction in pial arteries with generation number varying from 1 to 5. This vasoconstriction was induced by stimulation of the superior cervical ganglion with electrical pulses.

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MATERIALS AND METHODS

Experiments were carried out on WKY rats (n=9) weighing 250-290 g and SHR (n=9) weighing 240-280 g. Blood pressure in the caudal artery measured in wakeful animals with the cuff method was 120-130 mm Hg in WKY rats and 175-180 mm Hg in SHR.

The rats were intraperitoneally narcotized with urethane (1.25 g/kg). Heparin (500 U/kg) was used as the anticoagulation. Intravital television microscopy was used to scan the pial vasculature in reflected light under a modified MBS-2 microscope via an orifice made in the parietal region of the skull [1]. Arterial reaction (changes in diameter) was assessed by the width of erythrocyte flow in the vessel. The diameter of each vessel was measured in a series of images made before and after ganglionic stimulation. To compare the data obtained on vessels with different diameters, the values were expressed in percentage of the initial diameter [1].

The superior cervical ganglion (ipsilateral to the examined cerebral hemisphere) was stimulated with a series of rectangular voltage pulses presented via the bipolar silver electrodes applied directly onto the ganglion for 20 sec at a repetition rate of 10 Hz. The sympathetic tract below the ganglion was not cut. The threshold values of stimulating pulses were chosen experimentally. In WKY rats, the amplitude and pulse duration were 6.1 V and 0.5 msec, respectively. In SHR, the corresponding values were 9.1 V and 1 msec. The stimulation threshold in SHR was higher than in normotensive rats.

The examined vessels had generation numbers of 1-5 (A1-A5) and belonged to the basin of the medial cerebral artery. The first segment with A1 generation number was the region of this artery located proximately to the lateral surface of the skull (it emerged from under the bone at the margin of operation field). The generations A2-A5 were located successively downstream A1 segment. The diameters of the examined vessels were 8-106 μ in WKY (n=452) and SHR (n=543).

The constrictor reactions were analyzed with respect to the generation number of the arteries. The number of constricted arteries was assessed every 10 sec over 1.5 min after the end of stimulation.

RESULTS

During hypertension the amplitude of the constrictor response of pial arteries to stimulation of the superior cervical ganglion did not increase, which contradict published data [6,8]. In both rat groups, the amplitude of the constrictor response was 10-20% of the initial diameter. While there were no differences in the amplitude of the constrictor response between normotensive and hypertensive rats, the number of constricted vessels increased with generation number in SHR from 31% in A1 segment to 55% in A5 segment (Fig. 1), while in WKY rats it was constant (no more than 5%) in all examined segments A1-A5 (Fig. 2). The peak constrictor time (counted from the end of stimulation train) was defined as the moment when the greatest number of vessels of any generation constricted in response to ganglionic stimulation. In SHR, the peak constrictor time decreased from 70 to 40 sec in A1 to A5 arteries, respectively. By contrast, in WKY rats it increased from 0 to 20 sec in the same arteries. Thus, in normotensive rats the most rapid response to ganglionic stimulation was observed in A1 arteries, while in SHR the most rapid response was demonstrated by A4-A5 arteries. Both groups of rats had similar maximum number of constricted vessels: in

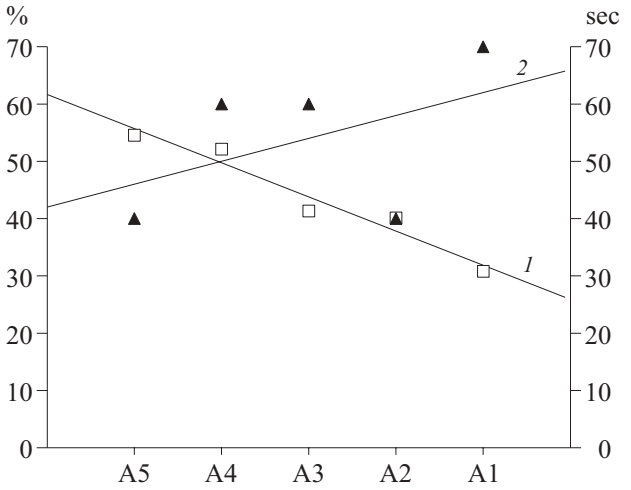

Fig. 1. Maximum number of constricted pial arteries in each generation (*1*) and peak constrictor time (*2*) in SHR. Here and in Fig. 2: abscissa, generation number of pial arteries; left ordinate, maximum number of constricted vessels relatively to the total number of arteries (%); right ordinate, peak constrictor time counted after the end of ganglionic stimulation, which corresponds to the maximum number of constricted vessels in each generation.

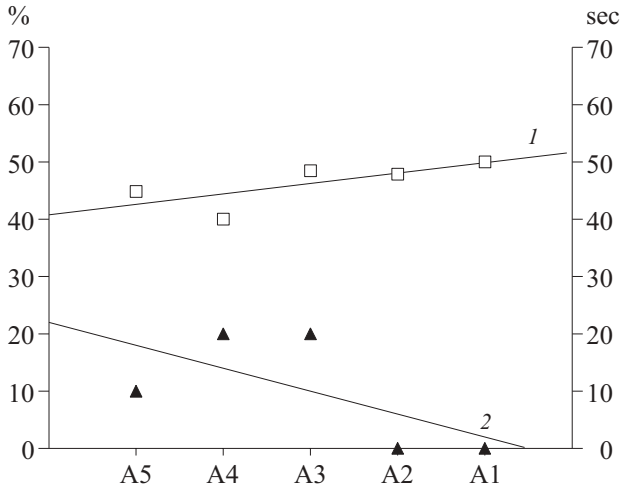

Fig. 2. Maximum number of constricted pial arteries in each generation (*1*) and peak constrictor time (*2*) in normotensive WKY rats.

WKY rats, 50% constricted vessels belonged to A1 generation, while in SHR 52-55% constricted vessels were the arteries of A4-A5 generations.

Theoretically, the described peculiarities of cerebral vasculature can be explained by the difference in diameters of the artery of the same generation in both rat groups. However, our data showed that the arteries of different diameters are available in all vessel generations of both rat groups (Table 1), although the number of the largest arteries (66-106 μ) with rich innervation and thick muscle layer [3] decreased (sometimes to zero) in the vasculature with high generation order. This can explain the

TABLE 1. Diameter Distribution (%) of A1-A5 Pial Arteries in WKY and SHR rats

Generation number (branching order)	Diameter, μ									
	WKY					SHR				
	8-25	26-45	46-65	66-85	86-106	8-25	26-45	46-65	66-85	86-106
1	0	41	37	9	13	1	44	32	18	5
2	11	39	30	13	7	35	34	26	5	0
3	35	54	11	0	0	49	35	15	1	0
4	51	41	8	0	0	65	35	0	0	0
5	78	22	0	0	0	100	0	0	0	0

decrease of stimulation-evoked constrictor responses in A3-A5 arteries. However, the total number of large arteries did not surpass 23% in each examined generation, where the arteries with diameter of 8-45 μ prevailed. Thus, the neurogenic control of the tone in pial arteries depends on their generation number.

Potentiation of neurogenic control of vascular tone in A5 arteries of hypertensive rats, together with previously established [1] moderation of myogenic tone can be considered as adaptation to elevated systemic arterial pressure. The more pronounced sympathetic vasoconstriction of the terminal segments of pial vasculature is probably aimed to defend the brain against hemodynamic stroke during drastic elevation of systemic arterial pressure, observed during depletion of pial vasculature [2,4, 5,7].

Thus, the constrictor response of pial arteries evoked by stimulation of the superior cervical ganglion was approximately equal in normotensive and spontaneously hypertensive rats. However, the maximum constrictor effect was realized by different ways under the normal and hypertensive conditions. In normotensive rats, it was attained due to rapid and uniform involvement of arteries of all generations, while in SHR it resulted from predominant constriction of precortical arterioles.

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