Correlation between Blood Pressure and Parameters of Systemic Hemodynamics during Pressor and Depressor Effects of the Ventral Medulla Oblongata

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Correlation between blood pressure and parameters of systemic hemodynamics was studied on cats using electrical stimulation of the ventral medulla oblongata. Changes in blood pressure induced by pressor activation were related to venous hemodynamics in the anterior rather than posterior vena cava. Venous blood flow is assumed to be involved in hemodynamic changes caused by stimulation of pressor centers but not depressor centers in the ventral medulla oblongata.

Key Words: *ventral medulla oblongata; systemic hemodynamics*

According to current views on neuronal mechanisms of regulation of systemic blood flow, the ventral medulla oblongata (VMO) plays an important role in changes in systemic blood pressure (BP) [3]. Neuroanatomical and electrophysiological data on projections of VMO neurons descending to sympathetic preganglionic neurons in the spinal cord have been reported [1,2]. Adjacent pressor and depressor zones were identified depending on pressor and depressor changes in BP caused by VMO stimulation [4,5]. However, the interrelation between changes in BP and the major parameters of systemic hemodynamics caused by activation of pressor and depressor zones of VMO received little attention.

Here we studied the interrelation between changes in BP and parameters of systemic hemodynamics caused by pressor and depressor effects of VMO and evaluated the role of arterial and venous hemodynamics in BP shifts under these conditions.

MATERIALS AND METHODS

Experiments were performed on 13 cats weighing 2.5-3.8 kg. The animals were narcotized with urethane (1 g/kg intravenously) and subjected to thoracotomy under conditious of artificial ventilation. The azygous vein was ligated, and venous return (VR) was determined as the total blood flow in the venae cavae. Electrical stimulation was performed in rectangular area in VMO 4 mm rostrally, 3 mm caudally, and 3 mm laterally to the intersection of the basilar artery and nerve XII orifice from the medulla oblongata [4,5]. Pressor and depressor sites in VMO were stimulated with constant current unipolar rectangular pulses (50-200 μA, 0.5 msec, 50 Hz) for 30-100 sec using a glass-insulated nichrome electrode (diameter 100 μ). Electrical stimulation was continued to obtaining maximum BP changes.

BP in the left subclavian artery and central venous pressure (CVP) in the right auriculum were recorded with EMT-34 and EMT-33 electron manometers (Elema-Schonander). Cardiac output (CO) and blood flow in the anterior and posterior venae cavae (BFAVC and BFPVC, respectively) were measured with cuff transducers of MFV electromagnetic blood flow meters (Nihon Cohden). The total peripheral resistance (TPR) and total VR to the heart, heart rate (HR), maximum blood flow acceleration in the ascending aorta (dCB/dt_{max}), and stroke volume were calculated.

The amplitude (maximum deviation from baseline) and temporal (time to peak changes) characteristics of hemodynamic parameters were determined. The

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results were analyzed by the linear correlation analysis using the Fischer—Student test.

RESULTS

Baseline blood flow parameters were: BP=87±3 mm Hg; CO=215±10 ml/min; VR=218±15 ml/min; BFAVC=43±4 ml/min; and BFPVC=172±12 ml/min.

Table 1 shows the amplitude and temporal changes in blood flow parameters caused by activation of the pressor and depressor VMO zones. The amplitude of BP shifts correlated ($p<0.05$) with the amplitudes of changes in CO, VR, BFAVC, BFPVC, CVP, stroke volume, dCB/dt_{max}, HR, and TPR. The correlation coefficient for BFAVC was maximum ($r=0.83$) and 2-fold surpassed that of BFPVC ($r=0.39$, Fig. 1). The time to peak BP correlated ($p<0.05$) with temporal characteristics of CO, VR, BFAVC, TPR, CVP, and dCB/dt_{max} and the time to peak BFAVC ($r=0.71$). There was no correlation between temporal changes in BP and BFPVC ($r=-0.02$, $p<0.01$). Thus, BP changes caused by stimulation of pressor VMO zones correlated with arterial (CO, stroke volume, and TPR) and venous (BFAVC, BFPVC, VR, and CVP) hemodynamics and changes in dCB/dt_{max} and HR. The stronger correlation between BP and BFAVC (compared with that between BP and BFPVC) indicates that BP changes are more closely related to venous hemodynamics in the rostral part than in the caudal part of the venous bed.

During the stimulation of VMO depressor sites, we revealed no significant correlation between the amplitude of BP changes and BFAVC ($r=0.44$) or BFPVC ($r=-0.02$). The coefficient of temporal correlation between BP and BFAVC ($r=0.96$, $p<0.01$) was the

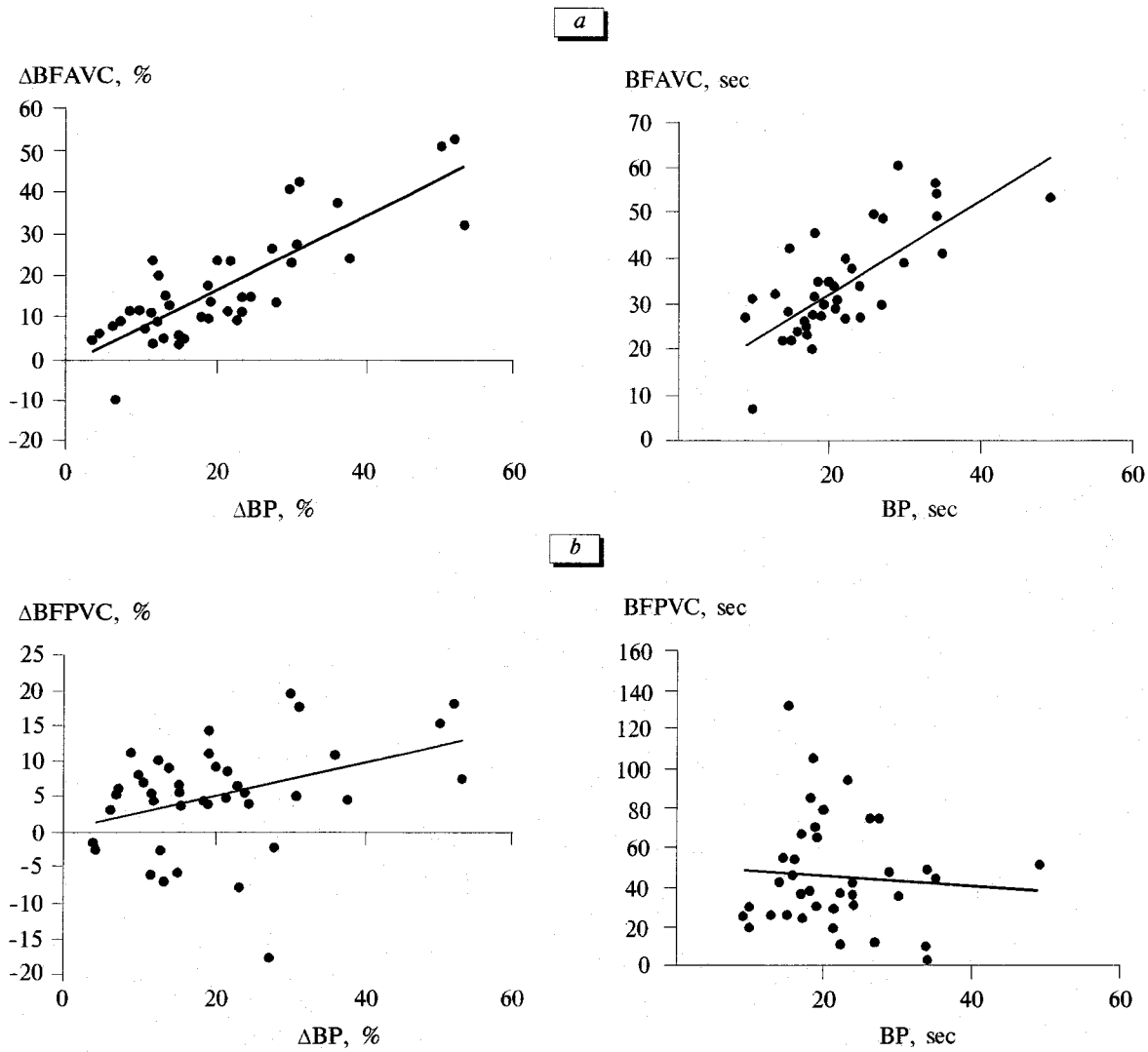

Fig. 1. Amplitude (left) and temporal (right) correlations between blood pressure and blood flow in the anterior (*a*) and posterior (*b*) venae cavae during electrical stimulation of pressor zone in the ventral medulla oblongata. Here and in Fig. 2: BP, blood pressure; BFAVC and BFPVC, blood flow in the anterior and posterior venae cavae, respectively.

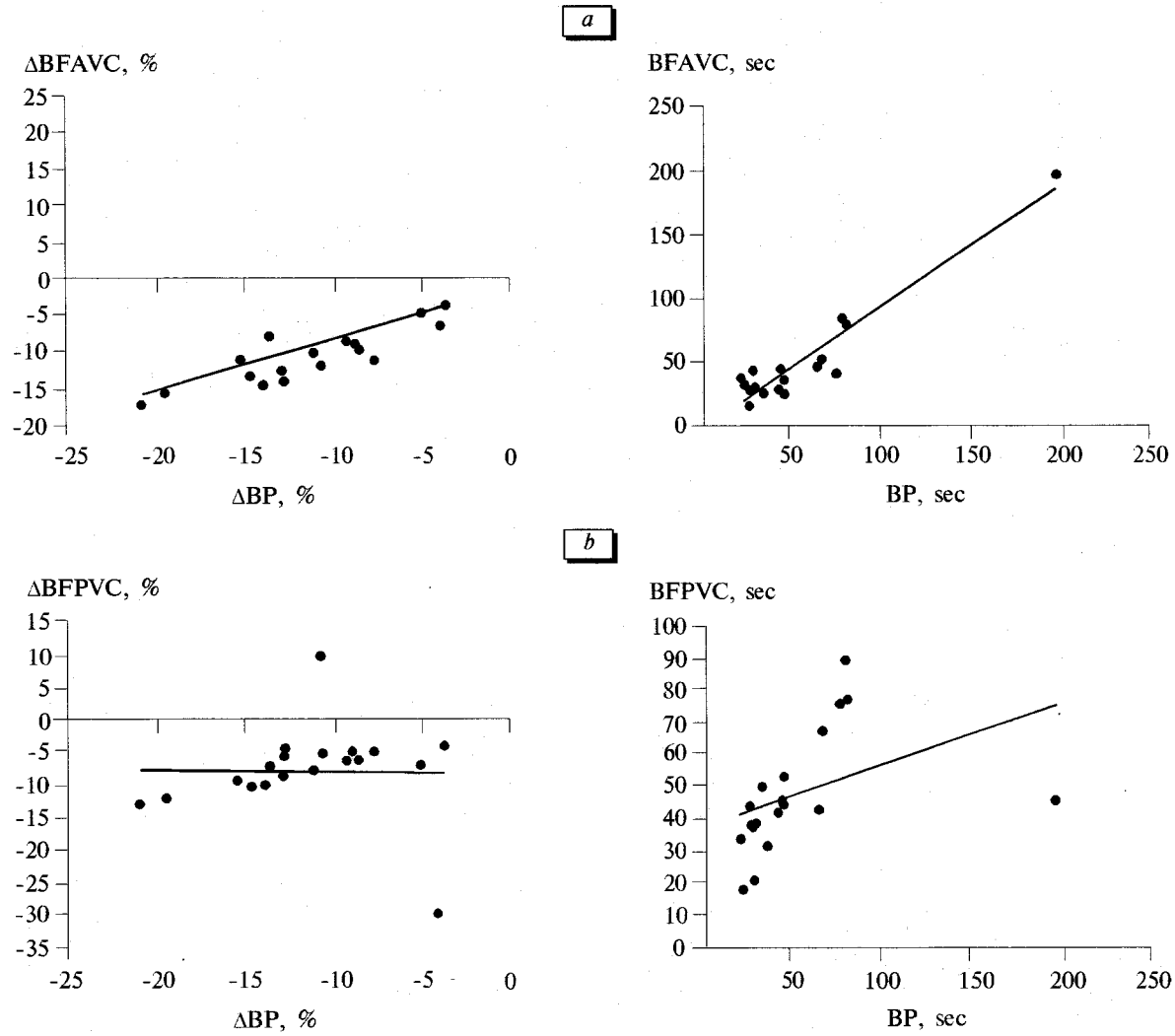

Fig. 2. Amplitude (left) and temporal (right) correlations between blood pressure and blood flow in the anterior (*a*) and posterior (*b*) venae cavae during electrical stimulation of depressor zone in the ventral medulla oblongata.

Table 1. Changes in Blood Flow Parameters Caused by Stimulation of the Ventral Medulla Oblongata (*M*±*m*)

Parameters	Stimulation			
	pressor zone		depressor zone	
	amplitude, %	time, sec	amplitude, %	time, sec
BP	+19±3	19±3	-13±3	56±10
CO	+11±2	35±3	-11±1	65±15
TPR	+20±3	15±1	-8±2	50±12
CVP	+7±2	26±4	-5±2	74±10

most important, while the interrelation between BP and BFPVC was insignificant (r=0.40, Fig. 2).

Significant correlation between the amplitude of BP and parameters of venous hemodynamics observed during activation of pressor but not depressor VMO zones indicates that veins are involved in the formation of BP changes caused by pressor rather than depressor effects of VMO.

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