Alterations in Systemic Hemodynamics Caused by Electrostimulation of the Ventral Medulla Oblongata

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Alterations in arterial blood pressure induced in cats by electrostimulation of pressor or depressor points in the ventral part of the medulla oblongata did not, as a rule, differ in direction from those in other hemodynamic parameters, including cardiac output, total peripheral resistance, central venous pressure, maximal acceleration of aortic blood flow, heart rate, venous return, blood flow in the superior and inferior venae cavae, and arterial inflow to and venous outflow from the posterior part of the body. It is postulated that the ventral medulla exerts direct influences on both the venous and arterial hemodynamics.

Key Words: *ventral medulla oblongata; systemic hemodynamics*

Current concepts concerning the cardiovascular center include the experimentally validated notion that an important role in the regulation of systemic arterial pressure (AP) is played by the ventral part of the medulla oblongata (VMO) [3]. As shown by neuroanatomical and electrophysiological studies, neuronal structures of the VMO have projections to sympathetic preganglionic neurons of the spinal cord [1,2]. However, what we know so far about how ventral bulbar structures influence systemic hemodynamics is largely based on experimentally recorded changes in only one hemodynamic parameter of the systemic circulation, namely AP [4,5]. The pattern of variation in these parameters upon stimulation of the VMO has not been described.

The present study was designed to examine the nature and magnitude of variation in major hemodynamic parameters upon electrical activation of pressor and depressor zones in the VMO and to identify the hemodynamic pattern of associated systemic responses.

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MATERIALS AND METHODS

The study was conducted on 11 Urethan-anesthetized and artificially ventilated cats (body weight 2.5-3.8 kg) with open chest. The azygos vein was ligated and so the venous return was equal to the total blood flow in the venae cavae. Electrostimulation was carried out in a rectangular area of the VMO with the boundaries located 3 mm rostral, 2 mm caudal, and 3 mm lateral to the site of the basilar artery's intersection with the 12th cranial nerve at the level of its exit from the medulla oblongata [4,5]. Pressor and depressor (in respect of AP) points of the VMO were stimulated, via a glass-insulated Nichrome electrode of 100 μ diameter, with current-stabilized square pulses (50-200 μA, 0.5 msec, 50 Hz) for periods of 30 to 100 sec. Electrostimulation was discontinued as soon as the maximal change in AP was recorded.

AP and central venous pressure were recorded with EMT-34 and EMT-33 electronic manometers (Elema) in the left subclavian artery and the right auricle of the heart, respectively. Cardiac output and blood flows in the superior and inferior venae cavae and at the distal bifurcations of the abdominal aorta (arterial inflow to and venous outflow from the pos-

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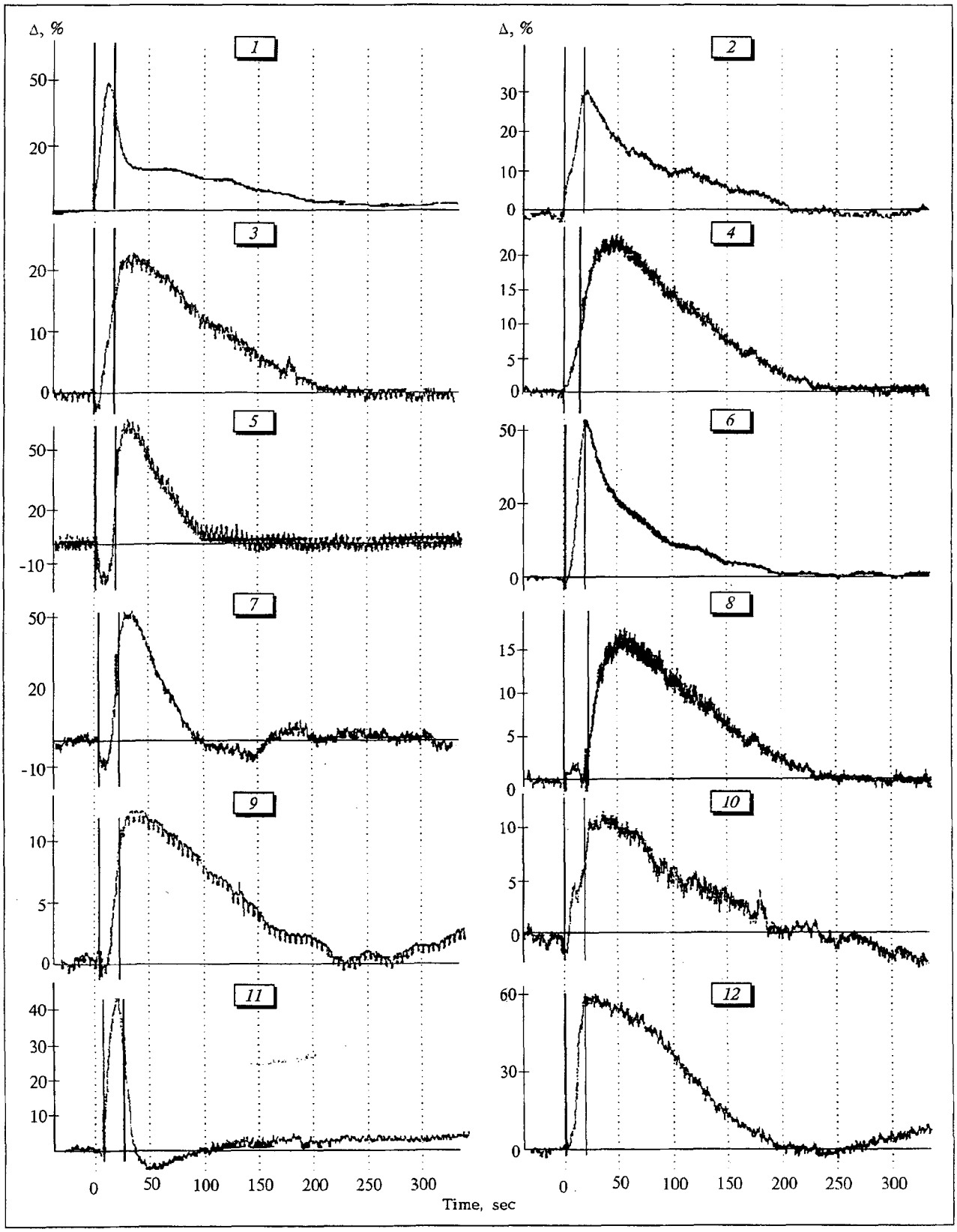

Fig. 1. Variations in major parameters of systemic circulation caused by electrostimulation of pressor points in the ventral medulla oblongata in a cat. Here and in Fig. 2: *1*) AP; *2*) central venous pressure; *3*) cardiac output; *4*) total venous return to the heart; *5*) arterial inflow to the posterior part of the body; *6*) blood flow in the superior vena cava; *7*) venous outflow from the posterior part of the body; *8*) blood flow in the inferior vena cava; *9*) heart rate; *10*) stroke volume; *11*) total peripheral resistance; *12*) maximal acceleration of aortic blood flow. Vertical lines mark the start and end of electrostimulation.

TABLE 1. Effects from Electrostimulation of Pressor and Depressor Points in the Ventral Medulla Oblongata on Parameters of Systemic Circulation in Cats (*M*±*m*)

Parameter	Baseline	Pressor zone (*n*=30)		Depressor zone (*n*=18)	
		Δ, %	time, sec	Δ, %	time, sec
Arterial pressure, mm Hg	84.61±2.31	21.1±2.4	18±10	-12.9±2.0	56±10
Total peripheral resistance, arb. units	0.38±0.02	18.9±2.9	14±1	-7.6±1.6	47±11
Cardiac output, ml/min	210.20±9.09	11.3±1.2	35±3	-10.5±0.8	65±14
Venous return, ml/min	213.43±14.76	9.1±1.2	36±3	-11.8±2.5	64±14
Blood flow in vena cava, ml/min:					
superior	42.55±3.01	17.1±2.3	32±2	-9.5±1.0	44±6
inferior	170.52±10.30	8.1±1.2	40±5	-11.4±2.9	66±14
Posterior part of body					
arterial inflow, ml/min	31.36±1.28	23.2±2.6	43±6	-16.3±2.0	44±4
venous outflow, ml/min	40.43±2.09	20.2±2.9	40±2	-14.1±2.9	43±6
Heart rate, beats/min	172.73±3.01	4.4±0.8	41±6	-2.5±0.4	46±8
Stroke volume, ml	1.22±0.05	8.6±1.0	36±4	-10.0±0.9	72±14
Central venous pressure, mm H_2O	29.03±2.36	7.2±1.6	26±4	-4.7±0.7	74±12
Maximal acceleration of aortic blood flow, arb. units	317.17±72.59	12.9±5.1	26±6	-6.5±2.1	41±11

terior part of the body) were measured with cuffed sensors of electromagnetic blood flowmeters (Nihon Kohden). All experimental data were recorded in real time on a computer.

In addition to the hemodynamic parameters measured directly, the following parameters were determined by calculation: total peripheral resistance, total venous return to the heart, heart rate, maximal blood flow acceleration in the ascending aorta, and stroke volume.

RESULTS

The activation of pressor or depressor points in the VMO caused shifts in the AP that did not, in most cases, differ in direction from those in the other hemodynamic parameters (Figs. 1 and 2). However, whereas the parameters characterizing cardiac activity and the arterial compartment of the circulatory system always varied in the same direction as did the AP, alterations in the venous compartment were opposite in direction in a number of instances. Thus, stimulation of pressor VMO points led to increased blood flows in the superior and inferior venae cavae in 7 cats (30 tests) (Fig. 1) and to an increased blood flow in the superior vena cava but a decreased flow in the inferior vena cava in 2 cats (5 tests), whereas one animal was observed to show increased as well as decreased blood flows in both venae cavae. Similar results were obtained with stimulation of depressor VMO points: the fall in AP was accompanied by decreases in blood flows via both the superior and inferior venae cavae (indicating diminished venous return) in 9 cats (18 tests) (Fig. 2), but by an increase in blood flow via the superior vena cava and its decrease via the inferior vena cava in one cat.

Alterations in the hemodynamic parameters induced by stimulation of the pressor and depressor zones of the VMO are summarized in Table 1. As is evident from this table, the times at which the differences from baseline values reached their maxima were for most parameters substantially shorter with stimulation of the pressor points than of the depressor points. Thus, the greatest alterations were recorded at 14-43 sec after stimulation of the pressor zone was started, but at 41-74 sec after the start of depressor zone stimulation. It was only for the heart rate and arterial and venous blood flows in the posterior part of the body that no significant temporal differences were detected. It should be noted that the times during which parameters remained altered (i.e., the times taken by the parameters to regain their baseline values) also differed, being shorter with stimulation of the pressor VMO zone (132-142 sec *vs.* 196-207 sec with depressor zone stimulation).

In addition to shedding light on variations in blood flows in the venous and arterial parts of the circulatory system in response to stimulation of the VMO, this study also enabled us to gain important information on the contribution of cardiac and vascular components to shifts in AP, on the role which the stroke volume and heart rate play in altering cardiac output, and on how the blood flow via each

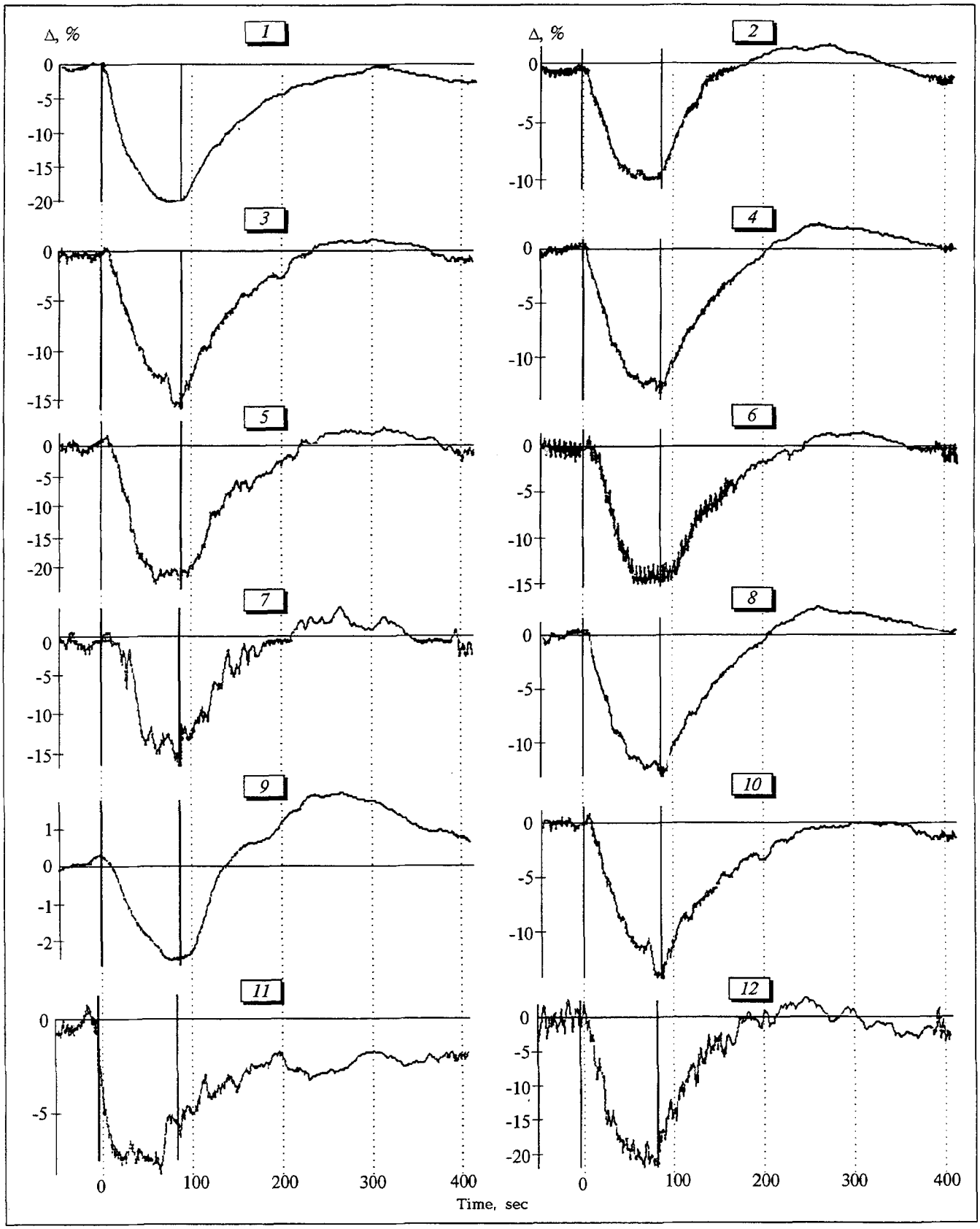

Fig. 2. Variations in major parameters of systemic circulation caused by electrostimulation of depressor points in the ventral medulla oblongata of a cat.

of the venae cavae influences the overall venous return to the heart.

In the hemodynamic mechanism of AP changes in response to stimulation of pressor and depressor points in the VMO, both the vascular (total peripheral resistance) and cardiac (cardiac output) components were found to be implicated (Figs. 1 and 2 and Table 1). However, when the time courses of the AP changes and AP-raising parameters (Fig. 1) are considered in relation to the magnitudes of shifts in these parameters (the 11.3% rise in cardiac output and the 18.9% increase in total peripheral resistance — Table 1), it becomes evident that the vascular component had a greater impact on pressor changes in AP than the cardiac component. Conversely, the cardiac component made a greater contribution to the depressor shifts in AP (Fig. 2) (the 10.5% fall in cardiac output — Table 1) than did the vascular (the 7.6% decrease in total peripheral resistance — Table 1).

Alterations in cardiac output were mainly due to changes in stroke volume (which, as shown in Table 1, increased by 8.6% and decreased by 10% with pressor and depressor zone stimulation, respectively) rather than to changes in heart rate (which increased 4.4% and decreased 2.5%, respectively). The changes in stroke volume observed with both types of stimulation can be accounted for by the impact of alterations in venous return on myocardial contractility, which was estimated by the maximal blood flow acceleration in the ascending aorta (the 12.9% increase and 6.5% decrease with pressor and depressor zone stimulation, respectively — Table 1).

Alterations in the arterial compartment of the circulatory system were accompanied, as can be seen in Table 1, by well-defined variations in the venous compartment. With both pressor and depressor zone stimulation, blood flow alterations in the inferior vena cava made a greater contribution to the changes in venous return than did those in the superior vena cava, as is evidenced by differential changes in blood flows expressed in absolute terms (ml/min): pressor zone stimulation increased (on average) blood flow by 13.8 ml/min in the inferior and only by 7.2 ml/min in the superior vena cava, while depressor zone stimulation decreased it by 19.4 ml/min in the inferior and only by 4.0 ml/min in the superior vena cava.

Comparison of blood flow variations in the arterial compartment with those in the venous compartment at the systemic and regional levels (i.e., in cardiac output and venous return, on the one hand, and in arterial inflow to and venous outflow from the posterior part of the body, on the other) does not show a substantial imbalance between the arterial and venous blood flows (Table 1), and it is therefore not possible to evaluate the degree to which the venous compartment was involved in the observed phenomena. Nonetheless, the results of this study, which point to the possibility of differential blood flow changes occurring in the superior and inferior venae cavae in response to electrostimulation of pressor or depressor points in the VMO, suggest that ventral bulbar neurostructures exert direct influences on both the venous and arterial hemodynamics.

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