Dependence of Blood Pressure Changes in Cats during Acute Hypoxic Hypoxia on the Type of Carotid Sinus Reflex

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The type and degree of blood pressure changes in cats were studied in acute experiments under conditions of acute hypoxic hypoxia (40% decrease in oxygen partial pressure). During hypoxia, blood pressure increased in cats with pressor type of the carotid sinus reflex and decreased in animals with depressor type of this reflex. Our results indicate that the direction and degree of hypoxic changes in blood pressure in animals coincide with variations in this parameter in response to the carotid sinus reflex.

Key Words: *blood pressure; cardiac output; carotid sinus reflex; acute hypoxia*

The cardiovascular response to functional load (orthostatic hypoxic exposure, Valsalva maneuver, *etc*.) can depend on the basal blood pressure (BP) [1-5]. BP is determined by sensitivity of baro- and chemoreceptors in the aorta and carotid sinus modulating pulse activity in afferent and efferent fibers of autonomic cardiac and vascular nerves [6-8]. Acute hypoxic hypoxia is used in circulatory physiology as a common functional load on the cardiovascular system [1,6-8]. Previous studies showed that hydraulic resistance of the vascular bed in humans and vertebrate animals decreases during hypoxia [5-8]. Hypoxia decreases the sensitivity of arterial chemoreceptors in systemic circulation to neurogenic and humoral adrenergic influences [6-8]. However, the dependence of these influences on the type of hypoxia and individual features of the cardiovascular system was not studied.

Here we studied the direction and degree of BP changes under conditions of acute hypoxic hypoxia as a function of basal BP and type of reflex responses to short-term compression of the carotid arteries (carotid sinus reflex).

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MATERIALS AND METHODS

Experiments were performed on 10 cats weighing 3.5-5.0 kg. The animals were anesthetized with nembutal (35-40 mg/kg intramuscularly) under conditions of artificial ventilation and thoracotomy. BP in the left femoral artery was measured with a Statham P23XL sensor. Cardiac output in the ascending aorta was estimated using a cuff transducer of a MVF-2100 electromagnetic flowmeter (Nihon Kohden). Total peripheral vascular resistance (TPVR) was calculated as the ratio of mean BP to cardiac output using an analog divider. Heart rate (HR) was measured with a tachometer on the basis of ECG recorded in standard lead II. Arterial blood gas composition was determined using an ABL-3 gas analyzer (Radiometer). Heparin in a dose of 1000 U/kg was injected intravenously to prevent thrombus formation. Acute hypoxic hypoxia was induced by 5-min inhalation of a gas mixture with 8% oxygen and 92% nitrogen via an artificial ventilation apparatus. This exposure led to a 40% decrease in oxygen partial pressure of the arterial blood and insignificant (under conditions of open thorax) changes in CO_2 pressure, which is consistent with published data [1].

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BP, cardiac output, and estimated TPVR were recorded using an N 338-8P ink recorder. The results were analyzed by paired Student's t test at $p<0.05$ (Axum software, MathSoft Inc.).

RESULTS

Basal BP underwent various changes in response to short-term compression of the carotid arteries (10 sec). Carotid artery compression in group 1 cats (n=5) increased systemic BP by 25±5% from the basal level (100±5 mm Hg). These animals exhibited a typical pressure type of the carotid sinus reflex. BP in group 2 cats (n=5) decreased by 15±4% after carotid artery compression. It should be emphasized that basal BP in group 2 cats (128±6 mm Hg) was higher than in group 1 animals. Hence, group 2 cats were characterized by an atypical depressor type of the carotid sinus reflex. No intergroup differences were found in basal characteristics of systemic hemodynamics. The exception was the estimated value of TPVR. TPVR in group 2 cats was higher than in group 1 animals (Table 1).

The type and degree of BP changes in animals of various groups were compared after acute hypoxic hypoxia for 40 sec and 5 min (Figs. 1 and 2). BP in group 1 cats increased 40 sec and 5 min after the start of hypoxia (by 28±4 and 22±5%, respectively, Fig. 1 and 2). BP in group 2 animals decreased 40 sec and 5 min after the start of hypoxia (by 6±3 and 23±6% of the basal level, respectively). Therefore, BP in cats of these groups underwent opposite changes under conditions of acute hypoxic hypoxia. The direction and degree of these changes coincided with BP variations in response to short-term compression of the carotid arteries.

Cardiac output in group 1 cats increased 40 sec after the start of acute hypoxic hypoxia (by 4±2%) and significantly exceeded the basal level by the 5th minute (by 11±2, $p<0.05$). Cardiac output in group 2 animals decreased 40 sec and 5 min after the start of hypoxia (by 7±4 and 14±5%, respectively, Figs. 1 and 2). We conclude that exposure to hypoxia induces opposite changes in cardiac output, which coincides with BP variations in animals of these groups.

TABLE 1. Basal Characteristics of Systemic Hemodynamics in Cats with the Pressor and Depressor Types of Carotid Sinus Reflex ($M±m$)

Parameter	Group 1 (pressor type)	Group 2 (depressor type)
BP, mm Hg	100±5	128±6
Cardiac output, ml/min	320±10	340±15
TPVR, arb. units	0.31±0.01	0.38±0.02
HR, bpm	195±3	198±5

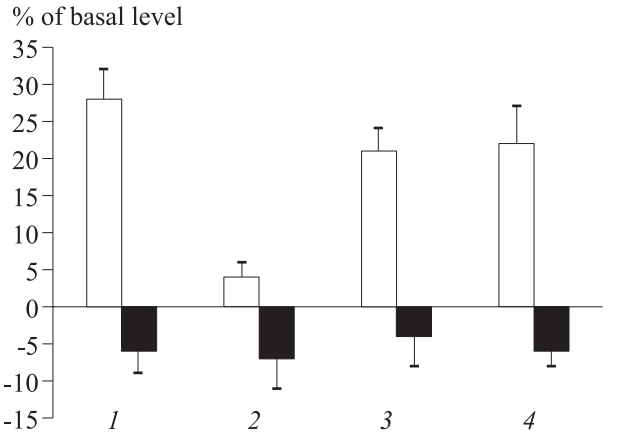

Fig. 1. Type and degree of changes in systemic hemodynamics in cats 40 sec after the start of acute hypoxic hypoxia. Here and in Fig. 2: BP (*1*), cardiac output (*2*), TPVR (*3*), and HR (*4*). Light bars, animals with the pressor type of the carotid sinus; dark bars, animals with the depressor type of the carotid sinus.

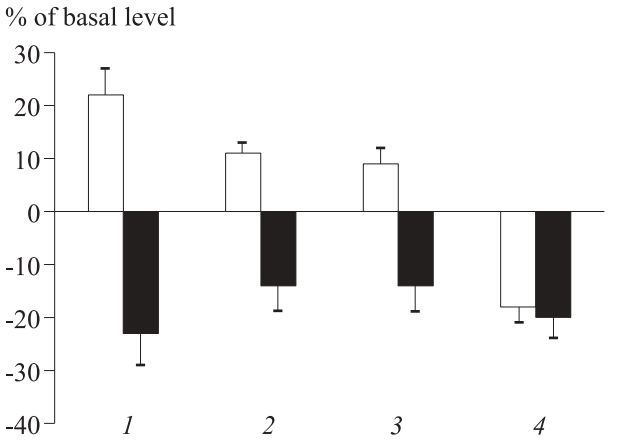

Fig. 2. Type and degree of changes in systemic hemodynamics in cats 5 min after the start of acute hypoxic hypoxia.

The estimated value of TPVR in group 1 cats increased after 40-sec acute hypoxic hypoxia (by 21±3%), but decreased in the follow-up period. Five minutes after the start of hypoxia, TPVR in these animals exceeded the basal level by 9±3% (Fig. 2). The estimated value of TPVR in group 2 cats decreased after hypoxia for 40 sec and 5 min (by 4±4 and 14±5%, respectively, Fig. 2). TPVR in group 1 cats with the pressor type of the carotid sinus reflex underwent biphasic changes during acute hypoxic hypoxia (increase and decrease), but remained above the basal level. TPRVR in group 2 animals with the depressor type of the carotid sinus reflex decreased under conditions of hypoxia. Hence, these animals were characterized by opposite changes in the estimated value of TPVR.

HR in group 1 cats increased 40 sec after the start of hypoxia (by 22±5%), but decreased by the 5th minute (by 18±3%, Figs. 1 and 2). HR in group 2 animals decreased 40 sec and 5 min after the start of hypoxia (by 6±2 and 20±4%, respectively, Figs. 1 and 2). These data show that HR in group 1 cats increased at the beginning of hypoxia, but decreased in the follow-up period (biphasic changes). HR in group 2 animals decreased in response to acute hypoxic hypoxia. The decrease in HR in cats of both groups by the 5th minute of hypoxia was probably related to the increase in inhibitory influences of the vagus nerve on the heart [4,5].

According to published data, high initial level of mean systemic BP can determine less significant changes in the parameter during reflex pressor influences on the circulatory system [2,5]. Systemic BP in cats with cardiopulmonary bypass increases from 85 to 120 mm Hg after continuous infusion of norepinephrine. These changes abolish variations in BP under conditions of carotid artery compression. However, similar increase in BP induced by angiotensin II infusion does not modulate the systemic pressor reflex [2,3]. The decrease in sensitivity of the heart and vessels to reflex influences of the nervous system is probably associated with activation of inhibitory α_2-adrenoceptors on sympathetic nerve endings with circulating and neurogenic catecholamines [5]. Moreover, mechanical deformation of vascular endothelial cells due to the increase in perfusion pressure results in the release of nitric oxide. This compound has the vasodilatory effect, which decreases the vascular response to constrictor influences [2,9].

Our experiments showed that animals with relatively high and low levels (within the normal range) of mean systemic BP are characterized by opposite changes in this parameter after short-term compression of the carotid arteries and acute hypoxic hypoxia. All parameters of systemic hemodynamics in cats with a lower level of systemic BP and pressor type of the carotid sinus reflex increased over the first 40 sec of acute hypoxic hypoxia. HR decreased, while BP, cardiac output, and TPVR in these animals remained high after 5-min hypoxia. BP, cardiac output, TPVR, and HR in cats with higher level of basal systemic BP and depressor type of the carotid sinus reflex decreased after the start of acute hypoxic hypoxia and remained low by the 5th minute of treatment.

Our results indicate that the direction and magnitude of BP shifts in animals during acute hypoxic hypoxia coincide with changes in this parameter in response to the carotid sinus reflex. We conclude that BP changes under conditions of acute hypoxic hypoxia depend on the sensitivity of chemoreceptors in the carotid reflex zone. Reactivity of the cardiovascular system in animals with high basal level of BP and carotid sinus depressor reflex decreases during hypoxia. These changes are probably associated with the decrease in adrenergic influences on the heart and vessels due to the inhibition of catecholamine release from sympathetic nerve endings.

REFERENCES

1. A. A. Nurmatov, A. V. Samoilenko, and B. I. Tkachenko, *Ros. Fiziol. Zh.*, **74**, No. 1, 1515-1522 (1988).
2. L. I. Osadchii, *Izv. Akad. Nauk. Ser. Biol.*, No. 6, 694-704 (2002).
3. A. P. Pugovkin, V. N. Kalikhevich, Z. A. Ardemasova, and L. I. Osadchii, *Fiziol. Zh.*, **67**, No. 11, 1733-1736 (1981).
4. B. I. Tkachenko, V. I. Evlakhov, A. P. Pugovkin, and L. N. Shalkovskaya, *Uchenye Zapiski SPbGMY Im. Akad. I. P. Pavlova*, **6**, No. 1, 61-65 (1999).
5. O. Appenzeller and E. Martignoni, *J. Auton. Nerv. Syst.*, **57**, No. 1, 234 (1996).
6. V. E. Claydon, L. J. Norcliffe, J. P. Moore, *et al.*, *Exp. Physiol.*, **90**, No. 1, 103-110 (2005).
7. V. L. Cooper, S. B. Pearson, C. M. Bowker, *et al.*, *J. Physiol. (Lond.)*, **568**, Pt. 2, 667-677 (2005).
8. J. R. Halliwill and C. T. Minson, *J. Appl. Physiol.*, **98**, No. 3, 1050-1056 (2005).
9. L. I. Osadtchy, T. V. Balueva, and I. V. Sergeev, *Acta Physiol. Hung.*, **87**, No. 1, 77-86 (2000).