HEMODYNAMIC CHANGES IN RESPONSE TO AN INCREASED FUNCTIONAL LOAD ON THE CARDIOVASCULAR SYSTEM

A. A. Podkolzin, S. O. Trenin, A. P. Legoshin, and A. V. Khokhlov

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The development of surgery of the heart and blood vessels makes the study of changes in the central hemodynamics during operations on the heart and great vessels particularly important. During such operations a temporary disturbance of the cerebral circulation [7] or constriction of the great vessels [4, 9] frequently arises, and leads to ischemia of vital organs. Under these circumstances there is a marked increase in contractile activity of the heart [3, 10]. However, the dynamics of these changes and the compensatory powers of the heart have not been adequately investigated.

Our aim was to study changes in the central hemodynamics during constriction of the ascending aorta, asphyxia, and cerebral ischemia.

EXPERIMENTAL METHOD

Series of experiments were carried out on 26 dogs weighing 8-10 kg, anesthetized with hexobarbital (50 mg/kg) and undergoing thoracotomy with artificial ventilation. In series I, coarctation of the ascending aorta was produced by a tourniquet for 5 min, with reduction of its lumen by 3 or 5 times. In series II the brachiocephalic trunk was constricted for 3 min. In series III asphyxia was induced for 3 min by stopping the artificial ventilation, and with both pleural cavities open.

During the experiment and in the recovery period, the electroencephalogram (EEG), ECG, central venous pressure (VP), and the blood pressure in the femoral and subclavian arteries were recorded by means of an MKh-01 monitor. The Wetzler–Boher formula was used to calculate the systolic volume of the heart (SV), the cardiac output (CO), the specific peripheral vascular resistance (SPVR), and the work of the left ventricle (A) by methods described in [1, 6]. The results were subjected to statistical analysis by Student's test.

EXPERIMENTAL RESULTS

In the initial state the heart rate (HR) was 121 ± 6.4 beats/min, the systolic blood pressure (BP_s) 122 ± 4.8 mm Hg, diastolic (BP_d) 90 ± 3.4 mm Hg, VP 15.9 ± 1.3 mm Hg, SV 11.4 ± 1.4 ml, CO 1.4 ± 0.1 liter/min, SPVR 28 ± 2.3 conventional units, and A 1519 ± 101 g/cm.

After constriction of the ascending aorta by two-thirds, changes in the EEG and ECG and the parameters of the central hemodynamics were not statistically significant. The exception was A, which decreased after the first minute by 18%, but then gradually rose to reach its initial level by the 5th minute, and remained unchanged after restoration of the lumen of the aorta. Constriction of the ascending aorta by four-fifths led to a reduction of 25% in SV, synchronization of rhythms was observed on the EEG, and the amplitude of the waves was increased and their frequency reduced. Changes in other

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TABLE 1. Changes in Hemodynamics during Coarctation of Aorta

Procedure	Parameter	Background	Constriction of aorta for 5 min	Recovery period	
				1 min	7 min
Threefold constriction of aorta	HR, beats/min	156±12,4	164±12,3	158±11,4	165±12,2
	BP_s	137±10,2	135±10,1	131±11,0	127±10,1
	BP_d mm Hg	98±6,8	97±6,8	90±5,9	92±6,7
	VP	18,1±2,3	18,2±2,3	18,2±2,4	17,8±2,4
	SV, ml	23,8±3,2	23±3,1	26±3,6	20±2,9
	CO, liters/min	3,7±0,8	3,7±0,4	4,2±0,8	3,9±2,5
	CI	4,8±0,4	5,0±0,3	6,5±0,4	4,7±0,5
	SPVR, conventional units	23±6,4	22±6,1	16±2,8	28±6,6
	A, g/cm	3592±124	3409±216	3680±108	3423±216
Fivefold constriction of aorta	HR, beats/min	136±6,4	146±8,2	164±7,2	138±6,8
	BP_s	146±5,6	130±4,4	141±4,1	138±4,1
	BP_d , mm Hg	100±3,4	103±4,6	98±2,4	95±2,7
	VP	15,4±1,0	16,1±1,8	16,0±1,4	15,5±1,4
	SV, ml	19±0,8	14,2±0,8	23,4±1,1	20±1,1
	CO, liters/min	2,6±0,2	2,1±0,4	3,8±0,4	2,8±0,3
	CI	5,4±0,4	4,4±0,2	7,9±0,5	5,8±0,4
	SPVR, conventional units	21±1,4	25±1,6	13±0,9	18±1,3
	A, g/cm	2868±102	2163±94	3310±121	2802±104

Legend. Here and in Table 2 SI denotes systolic index.

TABLE 2. Changes in Hemodynamics during Asphyxia and Constriction of Brachiocephalic Trunk

Procedure	Parameter	Background	Constriction of aorta for 5 min	Recovery period	
				1 min	5 min
Occlusion of brachiocephalic trunk	HR, beats/min	146±11,9	151±11,4	140±11,2	129±9,4
	BP_s	168±5,6	153±8,6	132±4,2	132±4,1
	BP_d mm Hg	118±3,3	110±3,8	99±3,4	101±3,4
	VP	13,2±0,7	15,9±0,8	17,9±0,4	15,7±0,8
	SV, ml	20,2±0,8	26±1,1	18,7±0,9	21±0,8
	CO, liters/min	3,3±0,09	3,7±0,08	2,6±0,04	2,9±0,08
	CI	5,3±0,3	5,7±0,3	3,7±0,8	3,9±0,9
	SPVR, conventional	25,6±0,9	22±0,8	29,5±0,6	28,5±1,9
	A, g/cm	3763±12,4	4455±12,8	2772±108	3261±104
Asphyxia	HR, beats/min	121±6,4	68±5,1	130±6,5	126±7,2
	BP_s	122±4,8	268±12,1	232±10,4	127±4,7
	BP_d mm Hg	90±3,4	169±11,8	159±10,4	100±5,0
	VP	15,9±1,3	21,1±1,3	16,2±1,4	15,8±1,6
	SV, ml	11,4±1,4	29±2,1	26±2,1	15,5±1,7
	CO, liters/min	1,4±0,1	1,7±0,1	3,4±0,3	1,9±0,2
	CI	2,6±0,3	3,4±0,3	6,9±0,7	3,7±0,4
	SPVR, conventional	28±2,3	56±3,8	26±2,1	33±2,7
	A, g/cm	1519±101	7651±173	6294±152	1697±124

parameters were not statistically significant. After restoration of the lumen of the aorta, at the first minute SV was increased and exceeded its initial value by 23%. HR rose by 21%, A by 15%, and CO by 46%. During the next 5 min all these parameters gradually returned to normal (Table 1).

In cerebral ischemia caused by constriction of the brachocephalic trunk, BP_s increased by 20%, BP_d by 18%, A by 58%, SV by 29%, and CO by 32%. The EEG and ECG were virtually unchanged. In the postischemic period activity of the cardiovascular system returned to normal in the course of 1 min (Table 2).

Asphyxia led to inhibition of the EEG, which by the 3rd minute consisted either of separate low-amplitude Δ-waves or remained on the isoelectric line (Fig. 1). At the first minute of asphyxia HR rose to 145 ± 5.2 beats/min (by 20%, $p < 0.05$), but later it fell, and by the 3rd minute it was only 56% of its initial value. VP rose by the 3rd minute by 33% (to 21.1 ± 1.3 mm Hg, $p < 0.05$). BP_s rose by 120%, BP_d by 88%, SV by 154%, A by 404%. and SPVR by 100%. CO did not differ significantly from its initial level (1.7 ± 0.1 liter/min, $p > 0.05$). After restoration of respiration VP and HR at the first minute regained their initial level (Fig. 2). In 66% of cases in this period arrhythmia developed. At the first minute the EEG showed recovery: a Δ-rhythm of increased amplitude appeared, with short periods of α-rhythm against its background.

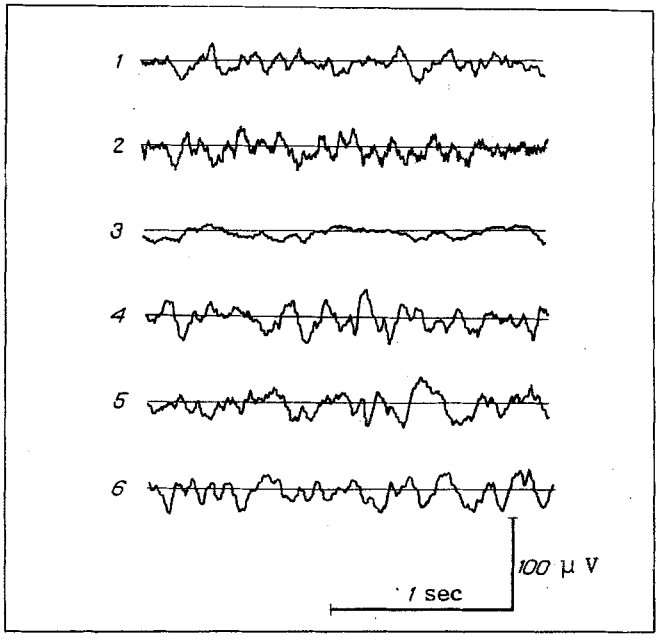

Fig. 1. EEG changes during asphyxia. 1) Background, 2 and 3) 1 and 3 min of axphyxia respectively; 4, 5, 6) 1, 3, and 5 min after asphyxia.

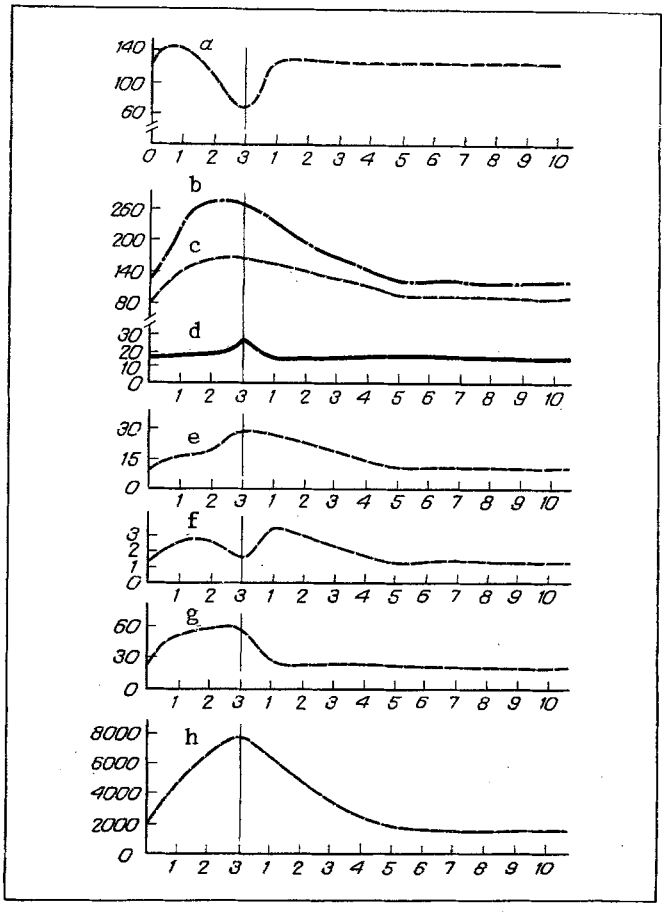

Fig. 2. Changes in hemodynamic parameters during asphyxia. On left of vertical axis — period of asphyxia, on right — period after asphyxia. Abscissa, time (in min); ordinate: a) HR, beats/min, b) BP_s (in mm Hg), c) BP_d (in mm Hg), d) VP (in mm Hg), e) SV (in ml), f) CO (in liters/min), g) SPVR (conventional units), h) A (in g/cm).

CO was increased by 143%. Later the EEG, ECG, and hemodynamic parameters returned to normal within the course of 5 min (Table 2).

The experiments showed that because of an increase in the contractile activity of the heart compensation of disturbances of the circulation in the organs took place in response to constriction of the ascending aorta by two-thirds, and also in response to compression of the brachiocephalic trunk. In the latter case blood reached the brain via collateral vessels, which are well developed in dogs [5]. That the blood supply to the organs was adequate was shown by the absence of changes in bioelectrical activity of the brain, which is extremely sensitive to oxygen deficiency [8].

Constriction of the ascending aorta by 80% led to hypoxia of the organs, as shown by synchronization of the EEG waves, characteristic of cerebral hypoxia [2]. However, this disturbance of the circulation, which lasted 5 min, did not lead to hypoxic damage of the organs and decompensation of the cardiovascular system.

During asphyxia, as a result of an increase in vascular resistance and myocardial hypoxia, heart failure developed, leading to an increase in VP. During this time HR fell on account of the lengthening of diastole, which enabled the heart to maintain an increase in BP and SV. However, as a result of bradycardia, CO did not increase under these circumstances. Despite considerable changes taking place in the body, asphyxia for 3 min did not lead to irreversible consequences, and after re-establishment of ventilation of the lungs, the activity of the cardiovascular system quickly returned to normal.

Thus the cardiovascular system possesses high powers of compensation and can completely neutralize disturbances of the circulation during occlusion of the brachiocephalic trunk for 3 min and constriction of the aorta by two-thirds for 5 min. Constriction of the ascending aorta by 80%, however, leads to hypoxia of the organs. However, this state, which lasts 5 min, does not lead to decompensation of cardiac activity, and after restoration of the lumen of the aorta the parameters of the central hemodynamics return to normal in the course of the first 5 min. During asphyxia heart failure develops and the heart fate falls. Meanwhile asphyxia for 3 min is not critical for dogs. During this period the heart continues to maintain high values of BP and CO, and after restoration of respiration, activity of the cardiovascular system returns to normal within the first 5 min.

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