Structural and Functional Analysis of Cerebral Basin Arteries in Different Periods of Hypertension

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Structure of arteries of different diameter in early and late periods of hypertension caused by experimental coarctation of the aorta was studied by histological and morphometric methods. Blood pressure was measured directly via catheterization of the common carotid artery and used for evaluation of hemodynamic in the cerebral basin. It was shown that the late period of hypertension is characterized by dilatation of medium arteries and constriction and increase tone of small arteries and arterioles. Comparison of blood pressure parameters with the parameters of inner diameter and thickness of the tunica media attests to progressive decrease of permissible tensile strength and initiation of rapture of the wall of small arteries and arterioles during chronic hypertension.

Key Words: *hypertension, permissible tensile strength, cerebral arteries, morphometry*

Morphological study of cerebral arteries in different periods of arterial hypertension (AH) development is interesting from both theoretical and practical points of view, because of the risk of hemodynamic disturbances in the cerebral basin that leads to disability or death [4,10]. This can be a result of vascular wall rupture. The density of the vascular wall is determined by permissible tensile strength (PTS) [5,8]. Simulation of elevated blood pressure in the cerebral basin in experimental animals opens new perspectives in evaluation and understanding of the influence of different factors on vascular tissues under conditions of increased functional load.

Here we performed complex morphometric analysis of arterial walls and studied hemodynamic parameters in the cerebral basin during different periods of AH.

MATERIALS AND METHODS

Hemodynamic model of aortic arch coarctation was surgically reproduced in 20 puppies (age 3-6 months) [7]; the control group comprised 8 dogs. The animals were sacrificed by bloodletting under narcosis (10 animals per group) in 3-5 days (early period of hypertension development) or 12 months (late period of hypertension development). BP was measured directly with a mercury manometer by catheterization of the common carotid artery and hemodynamic parameters in the cerebral basin were evaluated from these values. The outer (D) and inner (d) diameters were measured using a MOB-1-15× screw ocular micrometer; thickness of the tunica media [1] in pia matter arteries (medium-caliber) and thickness of intraorganic arteries (small-caliber) and arterioles were calculated. The objective criteria of vascular wall strength, PTS (σ), is calculated by the formula: $\sigma=pd/2\delta$, where p is blood pressure (mm Hg) and δ is thickness of the vascular wall (μ). Sections (5 μ) were stained with hematoxylin and eosin, after Hart, and after Masson. The significance of differences was evaluated using Student's *t* test.

RESULTS

BP in the common carotid artery 3 days after aorta coarctation increased by 1.4 times (to 128 *vs*. 90 mm Hg in control group). Similar increase was noted for

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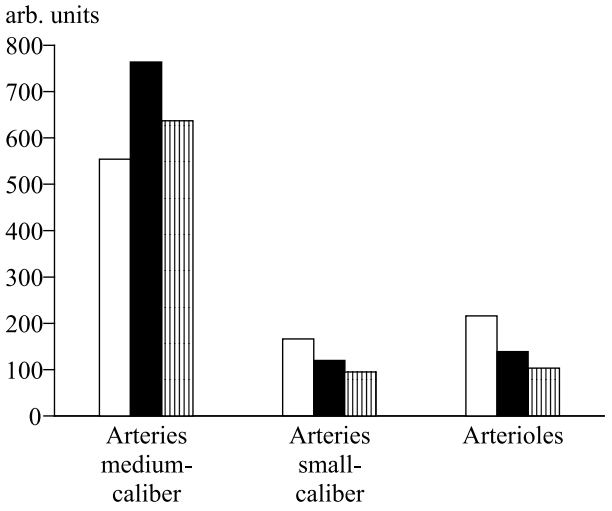

Fig. 1. PTS of the walls of cerebral arteries in dogs. Open bars: control; dark bars: AH, 3-5 days; hatched bars: AH, 12 months.

pressure of the blood entering the cerebral basin. The diameter of the medium-caliber arteries and thickness of the tunica media have not changed in the early period of AH development (Table 1, 2); PTS of the wall increased by 1.4 times (σ=764 units *vs.* 554 units in control group; Fig. 1). The inner diameter of small-caliber intraorganic arteries and arterioles decreased by 1.5 times (Table 1); the thickness of the tunica media of small arteries and arterioles increased by 1.3 and 1.4 times, respectively (Table 2), which attested to their increased tone. PTS of the walls of small arteries decreased by 1.4 times (σ=120 units *vs*. 166 units in the control group), PTS of the walls of arterioles decreased by 2.7 times (σ=80 units *vs*. 216 units in the control group).

In 12 months after aorta coarctation, BP in the common carotid artery attained 140 mm Hg, which led to further blood pressure rise in cerebral vessels. The lumen of the medium-caliber vessels increased by 1.2 times (Table 1) and thickness of their walls increased by 1.6 times (Table 2) in comparison with the control. PTS of the pia matter vessels decreased in comparison with the emergency stage of AH (σ=657 units), but remained above the initial level (by 1.2 times). At the same time, the inner diameter of small-caliber arteries and arterioles continued to decrease (by 1.7 and 1.6 times from the control, respectively; Table 1) and the thickness of their walls continued to increase (by 1.6 and 2 times in comparison with the initial values; Table 2). These facts suggest that intraorganic cerebral arteries in long-term AH are in the state of hypertonus, which may result in dissection of the tunica intima and sclerosis of the tunica media (Fig. 2). PTS of small arteries decreased by 1.8 times (σ=94.5 units *vs.* 166 units in the control), PTS of arterioles decreased by 2.1 times (σ=102 units vs. 216 units in the control; Fig. 1).

Coarctation of the aortic arch in experiment led to a significant BP elevation in the common carotid artery and cerebral basin. Morphometric analysis revealed different responses to hypertension in cerebral vessels of different calibers with increasing of AH duration: dilatations of medium-caliber arteries and constriction of small arteries and arterioles with increase in their tone. This response known as Bayliss–Ostroumov reflex is triggered by increased blood inflow to the cerebral basin [2]. The process plays an important compensatory and adaptive role aimed at the maintenance of stable capillary hemodynamics. Original mathematic analysis of blood pressure parameters, inner diameter, and thickness of the tunica media of cerebral vessels allows calculation and objective evaluation of PST of cerebral arteries of different calibers in the dynamics of AH development. We have also found that progressive increase of BP and duration of cerebral hypertension is accompanied by different changes in PTS of the walls of medium- and small-caliber arteries. Thus, PTS in the medium-caliber arteries slightly increased and in small intraorganic arteries and arterioles sharply decreased with increasing AH duration. It can result in progressive decrease of vascular wall strength and create prerequisites for destruction, initiate hemorrhages,

TABLE 1. Inner Diameter (μ) of Cerebral Arteries of Different Branching Orders (*M±m)*

Group	Arteries		Arterioles
	medium-caliber	small-caliber	
Control	394.0±5.5	27.7±0.3	14.4±0.3
AH, 3-5 days	406.0±6.5**	18.4±0.5*	9.4±0.4*
AH, 12 months	469.0±8.0*	16.2±0.4*	8.9±1.9*

Note. Here and in Table 2: *p<0.01, **p<0.05 in comparison with the control.

TABLE 2. Thickness of the Tunica Media (in μ) of Cerebral Arteries of Different Branching Orders in the Control and Experimental Groups (*M±m*)

Group	Arteries		Arterioles
	medium-caliber	small-caliber	
Control	32.0±1.5	7.5±0.2	3.0±0.1
AH, 3-5 days	34.0±2.0**	9.8±0.2*	4.3±0.1*
AH, 12 months	50.0±3.1*	12.0±0.2*	6.1±0.1*

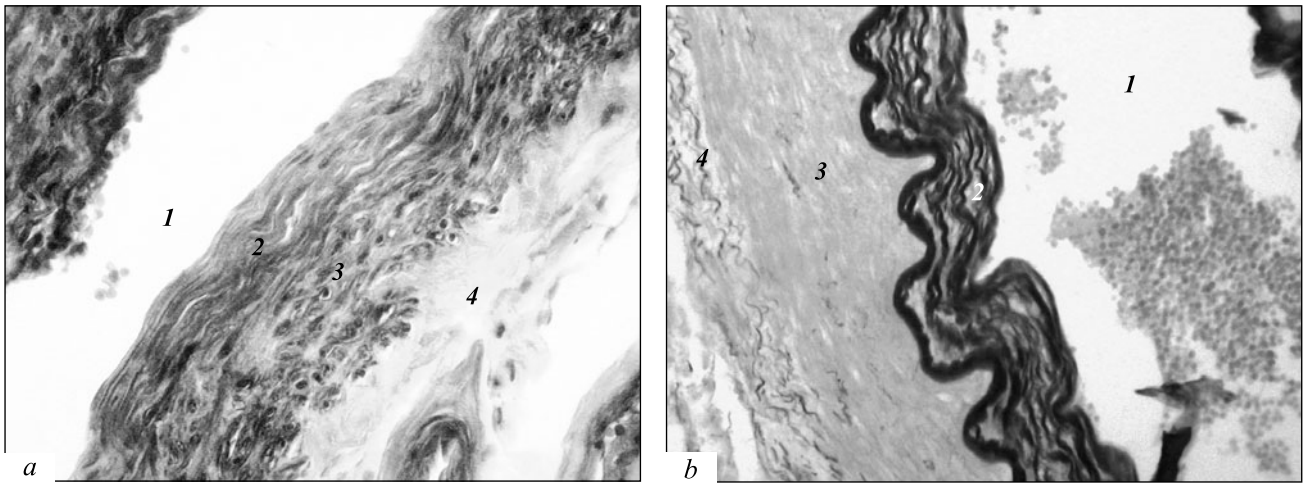

Fig. 2. Small-caliber cerebral artery of the muscle-type in a dog with 12-month AH. *a*) dissection of tunica intima, *b*) sclerosis of tunica media. Figures show vessel lumen (***1***), intima (***2***), media (***3***), and adventitia (***4***). Stained with fuchsin after Hart (*a*) and Masson (*b*), ×400.

and provoke acute cerebral circulation disturbances, which is confirmed by clinical observations [3,10].

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