Morphofunctional Changes in Renal Vessels Caused by Experimental Coarctation of Aorta

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In coarctation of the aorta tangential tension of the walls of renal arteries and arterioles decreases, which leads to a drop in the glycogen content and activity of respiratory enzymes in medial leiomyocytes and accumulation of glycosaminoglycans in the media. This is accompanied by atrophy and sclerosis of the vessels. The observed changes disappear after surgical correction.

Key Words: *coarctation of aorta; renal arteries; tangential tension; reversibility*

Structural changes occurring in the blood vessels of vital organs associated with circulation disorders caused by congenital cardiac disorders have been poorly investigated. The information on the reversibility of these changes is scarce. However, this issue is of theoretical and practical importance, since the condition of a patient and results of surgery depend on the state of his/her vascular bed.

Our goal was to examine structural changes of renal arteries and arterioles in experimental coarctation of the aorta and after its correction, and to find out how these changes are related to tangential tension of the vascular wall.

MATERIALS AND METHODS

Coarctation of the aorta was modeled in 20 puppies as described previously [4]. The animals were observed for 6-12 months, after which 10 dogs were removed from the experiment, and 10 dogs were subjected to repeated surgery with dissection of the narrowed site and implantation of a fluorolan-lavsan prosthesis. These dogs were observed for 6-12 months. Control group consisted of 10 dogs of the same age.

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The dogs were euthanized by bleeding under Promedol-ether anesthesia. Samples were taken from various sites of the kidneys, fixed in 10% neutral formalin and Carnoy's fluid, and embedded in paraffin. Sections were stained with hematoxylin and eosin and by the method of Masson and Hart. Interlobar, arcuate, and interlobular arteries and afferent and efferent glomerular arterioles were measured with the ocular-micrometer. The thickness of arterial media and the cross-section area were calculated from conventional formulas [1]. Smooth muscle cells were counted in the media of interlobular arteries. The size of these cells was assessed by the size of their nuclei. Glycogen in the media of renal vessels was revealed by PAS-reaction; glycosaminoglycans (GAG) were identified by the method of Hale. Activities of succinate dehydrogenase (SDG) and cytochrome oxidase (CCO) were measured on cryostat sections using nitro blue tetrazolium. The enzyme activities and glycogen and GAG contents in the kidneys were calculated as described [7]. Blood pressure in renal arteries was measured with a mercury manometer before sacrifice. Tangential tension of the renal artery wall was calculated from the following formula: $T = P \times R/d \times 133.3$, where P is the pressure (mm Hg), R is the radius of blood vessel (μ), d is the thickness of arterial wall (μ), and 133.3 is the correction factor for SI units. The results were processed by variational statistics methods.

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RESULTS

Experimental coarctation of the aorta was accompanied by a decrease in the cross-section area of the media and thinning of the walls of renal arteries. Cross-section area of the media in interlobar arteries and efferent arterioles decreased 1.4-fold ($p<0.001$), in arcuate arteries 1.3-fold ($p<0.001$), and in interlobular arteries and afferent arterioles 1.5-fold ($p<0.001$). The length and diameter of medial smooth muscle cells in renal arteries decreased as well as their cross-section area and volume: by 1.7- and 2.4-fold (Table 1), respectively; the number of smooth muscle cells dropped 1.2-fold (Table 1). These changes were accompanied by a 1.9-fold decrease in tangential tension of arterial wall (Fig. 1). Histochemical examination showed that glycogen content in leiomyocytes decreased 1.2-fold compared with the control (Fig. 1). Activities of SDG and CCO dropped 1.5- ($p<0.001$) and 1.4-fold ($p<0.001$), respectively (Fig. 1, Fig. 2, *a*, *b*). At the same time, the GAG concentration in the media increased 1.6-fold (Fig. 1), which coincided with the appearance of histological signs of angiosclerosis.

Correction of aortic coarctation reversed these changes. The cross-section area in interlobar and afferent arteries increased 1.4-fold ($p<0.001$), in arcuate and interlobular arteries, respectively, 1.2- and 1.6-fold ($p<0.001$), and in efferent arterioles 1.1-fold ($p<0.001$). The area and volume of smooth muscle cell nuclei increased, respectively, 2- and 3.8-fold as well as the number of smooth muscle cells (1.6-fold, Table 1). Tangential tension of renal arteries increased 1.1-fold (Fig. 1). Glycogen content in smooth muscles increased 1.3-fold as well as activities of SDG and CCO: 1.3- and 1.4-fold, respectively (Fig. 1). The concentration of GAG in the media of renal arteries decreased 1.3-fold (Figs. 1 and 2, *c*, *d*). Angiosclerosis was less pronounced.

Thus, prolonged experimental coarctation of the aorta induces morphological changes in renal arteries, which may be associated with a decrease in tangential tension of the vascular wall. Lower functional load leads to reduction in the glycogen content

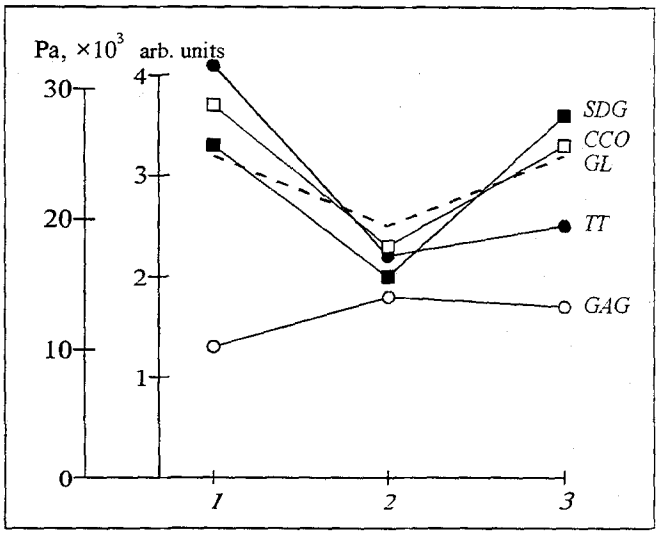

Fig. 1. Activity of CCO and SDG, contents of glycogen and GAG, and tangential tension in renal arteries. *1*) control; *2*) coarctation of the aorta; *3*) correction of coarctation. *SDG*) succinate dehydrogenase; *CCO*) cytochrome oxidase; *GL*) glycogen; *GAG*) glycosaminoglycans; *TT*) tangential tension.

and activity of respiratory enzymes in smooth muscle cells. A decrease in the activity of respiratory enzymes [3,6] leads to a slowdown of the oxidation-reduction processes and a decrease in the phosphorylation potential of leiomyocytes, their size, and protein synthesis rate in their cytoplasm. This results in atrophy of smooth muscles and thinning of the vascular wall. The concentration of GAG in the vascular wall increases, which promotes the development of angiosclerosis. After surgical correction of coarctation, blood flow through the renal bed increases, which elevates tangential pressure. This stimulates reversion of these changes. The glycogen content and the activities of CCO and SDG increase, which intensifies oxidation-reduction processes in arterial smooth muscle cells and increases the pool of marcoergic phosphates involved in muscle contraction [2,5]. An increase in protein synthesis is accompanied by cell hypertrophy and vascular wall thickening. The GAG concentration in the media drops. Thus, correction of coarctation of the aorta reverses morphological changes in renal arteries.

TABLE 1. Medial Smooth Muscle Cells of Small Renal Arteries in Coarctation of Aorta and After Its Correction ($M \pm m$)

Group	Nucleus length, μ	Nucleus diameter, μ	Nucleus area, μ^2	Nucleus volume, μ^3	Number of cells
Control	11.3±0.2	3.4±0.1	29.0±0.8	75.0±3.5	12.0±0.5
Coarctation of the aorta	8.7±0.2	2.6±0.06	17.0±0.4	31.0±1.2	10.0±0.6
Correction of coarctation	9.1±0.2*	4.7±0.1	34.0±0.9	109.0±0.5	14.0±0.5

Note. *$p>0.05$; other values are significantly different from the control at $p<0.001$.

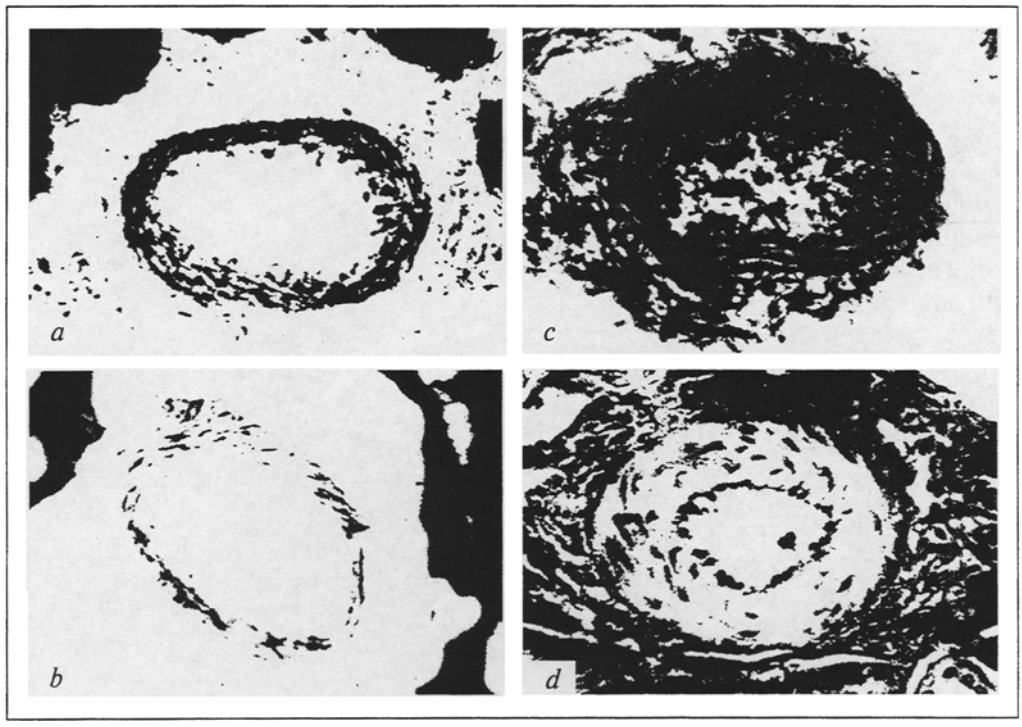

Fig. 2. Activity of succinate dehydrogenase (SDG) and glycosaminoglycans (GAG) content in renal arteries. ×280. *a*) high SDG activity in smooth muscles of interlobular artery of a control animal; *b*) low SDG activity in smooth muscles of interlobular artery in coarctation of the aorta; *c*) high GAG content in the wall of interlobular artery in coarctation of aorta; *d*) low GAG content in the wall of interlobular artery after correction of coarctation. *a*, *b*) nitro blue tetrazolium; *c*, *d*) method of Hale.

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