MORPHOLOGY AND PATHOMORPHOLOGY

Regulatory Structures of Cerebral and Renal Arteries in Experimental Hyper- and Hypotension

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 133, No. 2, pp. 219-221, February, 2002
Original article submitted October 11, 2001

Brain and kidneys from 15 control and 70 experimental puppies with hemodynamic model of coarctation of the aorta were examined. The number of regulatory muscle formations was increased in the brain and decreased in the kidneys during modeling of aorta coarctation associated with hyper- and hypotension in the corresponding basins. The location of these structures in cerebral and renal distribution and resistance vessels is analyzed.

Key Words: *regulatory structures; arteries; hemodynamics*

Regulation of organ circulation under conditions of low and high blood pressure is a key problems of angiology [3,7,8]. Various types of regulatory formations in vessels of vitally important organs in health and hemodynamic disorders are described [2, 5,9]. However the location of regulatory structures in cerebral and renal arteries was not studied and the formation of these structures under different hemodynamic conditions was not compared.

We investigated the regulatory structures in the cerebral and renal arteries in health and experimental hyper- and hypotension, examined their morphology, location, and effects on the organ hemodynamics.

MATERIALS AND METHODS

Cerebral and renal vessels were examined in 15 control puppies and 70 puppies with a hemodynamic model of coarctation of the aorta; the animals were observed for up to 18 months. The animals were sacrificed by bleeding under narcosis. Brain and kidney specimens were fixed in 10% neutral formalin or Carnoy fluid and embedded in paraffin. The sections were stained with hematoxylin and eosin and by Masson and Hart method. Succinate dehydrogenase (SDH) and cytochromoxidase activities in arterial leiomyocytes was detected by Burston's method [1] and those of acid phosphatase and nonspecific esterase by the azocoupling method. The number of regulatory structures in the cerebral and renal vessels was estimated.

RESULTS

Experimental coarctation of the aorta led to hypertension in the cerebral vessels and hypotension in renal vessels, which resulted in the formation of a complex of similar structures in arteries of these organs. These changes manifested in increased number and size of polyp-like pads (PLP), myoelastic sphincters (MES), and bundles of oblique longitudinal muscles of the intima (OLMI). All these formations were located at sites of arterial branching (Table 1).

We distinguished two types of PLP. Type I pads consisted of interwined of nonstriated muscle bundles directed to different sides. The peripheral part of type II PLP consisted on smooth-muscle

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0007-4888/02/1332-0185$27.00

TABLE 1. Location and Degree of Development of Regulatory Structures in the Cerebral and Renal Arteries in Coarctation of the Aorta

Regulatory structures	Distribution vessels	Resistance vessels	
		small arteries	arterioles
PLP	Detected	Not detected	Not detected
MES	Poorly expressed	Well expressed	Poorly expressed
Intimal muscles	Poorly expressed	Well expressed	Poorly expressed

Note. Distribution vessels are large and medium-sized arteries of the cerebral pia mater, renal interlobar and arcuate arteries; small arteries are cerebral intraorgan and renal interlobular arteries; arterioles are cerebral intraorgan and renal glomerular arterioles

and the central from connective tissue formed by fibroblast-like cells and fibrous structures, primarily fine collagen fibers (Fig. 1, *a*).

Depending on the spatial organization, we distinguished two types of MES (Fig. 2). The transverse section of the lateral branch of type I sphincter looked like a ring and the longitudinal section looked like two valves located at an angle, with the apexes directed against the blood stream. Type II sphincter also looked like a shaft, but it just partially encircled the mouth of the lateral vascular branch and formed only one leaflet protruding into the main artery, seen on the longitudinal sections. Sphincters of both types were formed by smooth muscle cell bundles embedded in the duplicature of the internal elastic membrane and densely braided with elastic fibers. These structures were lined covered with endothelium.

OLMI bundles were located in the arterial intima and often looked like shafts modifying lumen configuration (Fig. 1, *b*). They consisted of leiomyocytes braided with a dense network of elastic fibers and encircled each smooth-muscle cell.

Some of these structures were occasionally seen in cerebral and renal arteries of control dogs. The size of these structures and their number notably increased after induction of coarctation of the aorta [7]. All this attested to a direct relationship between the development of these structures and severity of circulatory disorders; presumably, these structures corrected regional hemodynamics. PLP, MES, and OLMI, appearing under conditions of

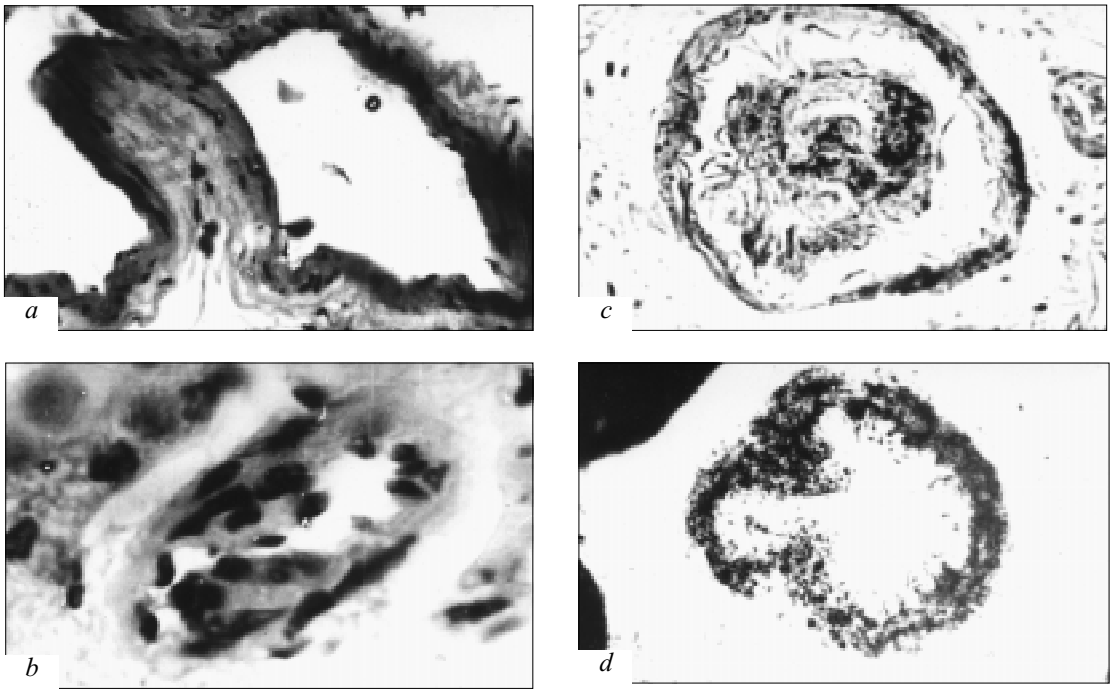

Fig. 1. Regulatory structures in the cerebral and renal arteries in experimental coarctation of the aorta. Hematoxylin and eosin staining (*a*, *b*), azo coupling (*c*, *d*). *a*) polyp-like pad in the large artery in the pia mater under conditions of hypertension, ×100; *b*) two bundles of oblique longitudinal muscles of the intima in the interlobular artery of the kidney under conditions of hypotension, ×400; *c*) high activity of nonspecific esterase in the polyp-like pad of the renal arcuate artery under conditions of hypotension, ×160; *d*) high activity of acid phosphatase in the myoelastic sphincter of the renal interlobular artery under conditions of hypotension, ×400.

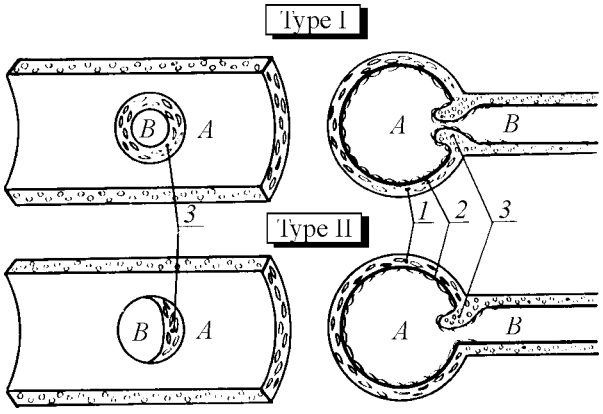

Fig. 2. Scheme of spatial organization of myoelastic sphincters in the cerebral and renal arteries. *A*) main vessel; *B*) lateral branch; *1*) tunica media; *2*) tunica intima; *3*) sphincter.

altered circulation in the brain and kidneys, start regulating the circulation impaired in these organs.

These structures located at sites of arterial branching can modify the volume of local blood flow by increasing or decreasing their tone. Contraction of PLP decreases blood flow in the main vessel and in its lateral branches, which alters hemodynamics in a large part of the brain or kidneys. Increase of MES and OLMI bundle tone leads to a decrease of the blood flow mainly in the lateral branches, which is associated with ischemia of a smaller volume of the cerebral and renal tissue. Hence, the detected formations are hemodynamic regulators, ensuring the transport confirmation of the known law of intermittent activity of biological structures [4].

The possibility of active regulatory functioning of the studied structures was confirmed by histoenzymatic investigations. Activities of respiratory and hydrolytic enzymes in muscle cells of these formations is notably higher than in myocytes of the tunica media of the corresponding arteries (Fig. 1, *c*, *d*). The presence of these enzymes indirectly reflects the concentration of phosphate compounds in myocytes, these compounds being essential for muscle contractions, and the intensity of oxidation processes [6].

Hence, similar regulatory structures (PLP, MES, and OLMI) form after coarctation of the aorta under different hemocirculation conditions in the cerebral and renal vessels. These formations are located at different levels of the vessels at sites of arterial branching and actively modulate the organ hemodynamics.

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