Adaptive Structures of Arterial Bed in Norm and in Modeled Congenital Heart Failure

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Material from 85 dogs with modeled congenital heart failure and from 20 control dogs was studied. Adaptive structures were revealed in the pulmonary and systemic circulation. These structures are formed by smooth muscle cells, are located at arterial bifurcations, compensate for increased hemodynamic load, and regulate blood flow in the vascular bed of a given internal organ.

Key Words: *adaptive structures; artery; hemodynamic regulation*

Regulation of blood flow in vascular beds of internal organs is a major problem of angiology [4,11]. It involves both extravascular factors [6] and vascular tone. Various adaptive formations affecting blood circulation have been described in the literature [8, 10,13]. However, in the majority of the investigations the descriptions do not provide sufficient detail.

In the present study we attempted to identify adaptive structures in the arteries of pulmonary and systemic circulation in health and in modeled congenital heart failure and to describe their structure, localization, to elucidate the mechanisms of their formation and influence on regional hemodynamics.

MATERIALS AND METHODS

Cardiac, cerebral, pulmonary, and renal vascular beds were studied in 20 healthy dogs and in 85 pups with hemodynamic models of ductus arteriosus, coarctation of the aorta, and stenosis of the pulmonary trunk existing for about a year. The animals were euthanized by bloodletting under anesthesia. Pieces of the studied organs were fixed in 10% neutral formalin or Carnoy's fluid and embedded paraffin. Sections were stained with hematoxylin and eosin, by the methods of Van Gieson, Masson, and Hart. Silver impregnation was performed by the method of Homori. Glycogen and glucosaminoglycans were visualized by the McManus and Hale methods. Succinate dehydrogenase and cytochrome oxidase activities in smooth muscle cells (SMC) were identified as described [2]. Acid phosphatase and nonspecific esterase were revealed by nitrogen coupling. Adaptive structures in the vascular bed were counted, and the transverse section area of their muscle bundles was calculated.

RESULTS

Qualitatively different circulatory disturbances in the studied arterial vascular beds were observed in dogs with modeled congenial heart failure. However, with some exclusions, these structures could be classified as polyp-like cushions (PLC), muscular-elastic sphincters (MES), oblique-longitudinal muscles in the intima (OLMI) and in the adventitia (OLMA).

The occurrence of PLC was low. They were observed at bifurcations of large and medium arteries supplying the heart and kidneys. These structures were not found in pulmonary arteries, which is probably associated with their specific (elastic) structure, and in cerebral arteries. PLC are ovoid in shape, protrude into the lumen (Fig. 1, *a, b*), and are attached to the vascular wall by a stem. The presence

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of the stem allowed us to classify them as polyp-like [10]. The stem consist of SMC bundles oriented in different directions and surrounded by reticular and elastic fibers. PLC are covered by elastic membrane and endothelium.

Muscular-elastic sphincters were observed in all arteries, their frequency being higher in medium and small vessels. They were located at arterial bifurcations and were formed by SMC bundles oriented in transverse or oblique direction relative to arterial axis (Fig. 1, *c*). These bundles were surrounded by a structure similar to the inner elastic membrane and fibers outgrowing from it. They are lined with endothelium.

Oblique longitudinal muscles of the intima were identified at arterial bifurcations, predominantly in small and large arteries. They consist of SMC bundles that differ in orientation: in some arteries they protrude into the lumen (Ebner cushions) [12] and encircle it in others, forming a layer (Fig. 1, *d*, *e*). OLMI are characterized by a well-developed elastic frame.

Oblique longitudinal muscles of the adventitia were located at bifurcations of large arteries. In cerebral and renal arteries they were represented by small accumulations of SMC. OLMA were well developed in pulmonary and particularly in cardiac arteries (Fig. 1, *f*). The stroma of OLMI is formed by thick collagen fibers (Fig. 1, *f*) and has a high content of glucosaminoglycans and a poorly developed elastic frame. OLMA were observed in healt-

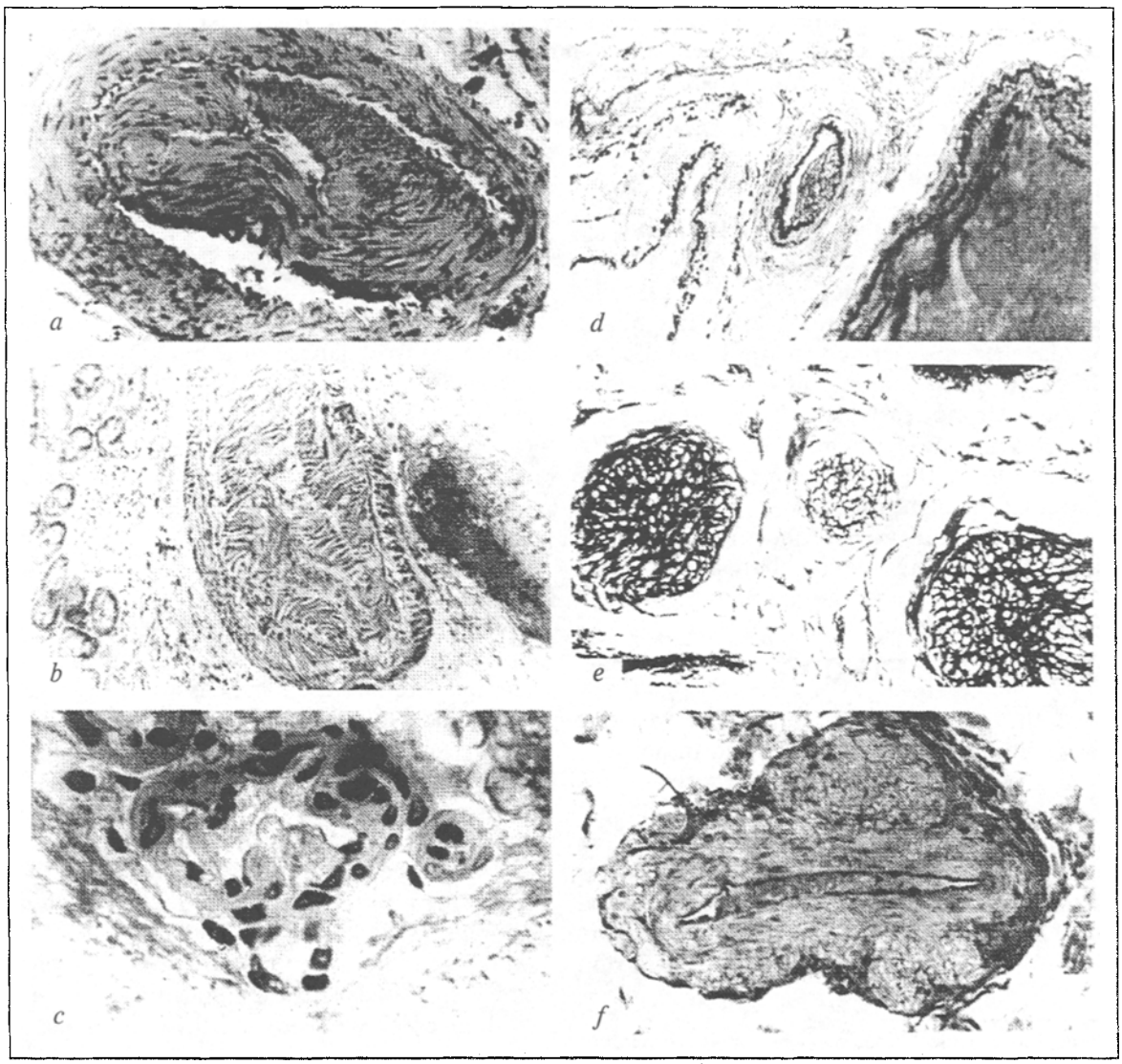

Fig. 1. Adaptive structures of arteries in models of congenital heart failure. *a*) PLC in the renal artery arch; coarctation of the aorta, ×240; *b*) high activity of acid phosphatase in a PLC in the renal artery arch; coarctation of the aorta, ×160; *c*) muscular-elastic sphincter in orifice of a small cerebral artery; coarctation of the aorta, ×400; *d*) OLMI in bronchial artery; open arterial duct, ×160; *e*) OLMI in bronchial artery; stenosis of the pulmonary trunk and open arterial duct, ×160; *f*) OLMA in a large coronary artery, coarctation of the aorta, ×240. Staining: *a*, *c*) hematoxylin and eosin; *b*) nitrogen coupling; *d*) Hart method; *e*) silver impregnation; *f*) Van Gieson method.

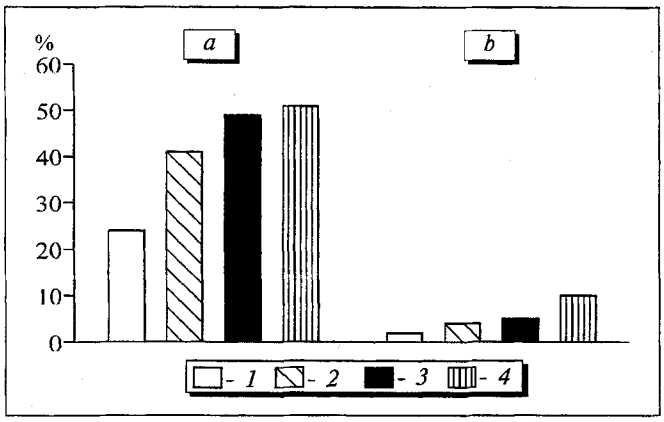

Fig. 2. Number of large arteries with OLMA (*a*) and small arteries with OLMI (*b*) in the coronary bed. *1*) control, *2*) arterial ductus arteriosus; *3*) stenosis of pulmonary trunk; *4*) coarctation of the aorta.

hy dogs. In dogs with modeled congenital heart failure their occurrence was much higher, as exemplified by OLMI and OLMA (Fig. 2). Consequently, there is a direct relationship between the hemodynamic factor and the occurrence of adaptive structures in the arterial bed. Their location at arterial bifurcations is due to considerable hemodynamic load on the vascular wall in this area. The greater the angle of bifurcation, the greater the load. This is confirmed by the following considerations. Hemodynamics load (P) can be calculated from the following formula [3]: $P=p\pi d^2/2\times sin\alpha/2$, where p is blood pressure; $\pi=3.14$; d is the diameter of an artery, α is the angle of bifurcation. Assuming $\alpha_1=5°$ and $\alpha_2=50°$, we obtain $P_1=0.04d^2p$, $P_2=0.4d^2p$, i.e., the load increases 10-fold when the bifurcation angle is increased by 45°. In arterial bifurcations this force causes migration of SMC from the media into the intima [9], formation of PLC, OLMI, and MES, transformation of adventitial pericytes into SMC [5], and formation of OLMA.

Polyp-like cushions are located in distributive arteries. In strain they protrude into the lumen and block blood flow in a considerable part of vascular bed. Muscular-elastic sphincters and OLMI are located in resistive arteries. When these arteries are contracted, blood flow in the microcirculatory bed is decreased. Periodic modulation of arterial tone by these structures is involved into regulation of blood filling of various part of vascular bed in accordance with functional activity of a given internal organ. Thus, these adaptive structures regulate hemodynamics, confirming the law of alternate activity of biological structures [7].

OLMA are not associated with arterial lumen. They are best developed in the bifurcations of cardiac and pulmonary arteries, the volume and bifurcation angles of which constantly change. This allows us to assume that OLMA do not regulate blood flow but maintains the longitudinal tone of the arteries at the sites where hemodynamic load is the greatest, i.e., serve as an adaptive structure compensating for this load.

All adaptive structures described in this study are active components of the vascular bed, judging from their high content of glycogen and high activities of succinate dehydrogenase, cytochrome oxidase, acid phosphatase, and nonspecific esterase (Fig. 1, *b*), indicators of high energy potential of these structures and intense metabolism in SMC.

Thus, various adaptive structures have been revealed in pulmonary and systemic circulations at arterialbifurcations. These structures are involved in the regulation of blood flow (PLC, OLMI, and MES) or compensate for hemodynamic load (OLMA). The initial stage of their morphogenesis is migration of SMC from the media into the intima or transformation of pericytes into SMC. This reaction is stimulated by circulatory disturbances associated with modeled congenital heart failure. It is genetically determined and functionally reasonable, providing formation of adaptive structures at all levels of the cardiovascular system.

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