PRINCIPLES OF FUNCTION OF ARTERIOLO-VENULAR ANASTOMOSES IN PHARMACOLOGIC ARTERIAL HYPEREMIA

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KEY WORDS: spasmolytics; microcirculation; arteriolo-venular anastomoses.

The study of the role of arteriolo-venular anastomoses (AVA) in the terminal circulation has received increasing attention in recent years. Available information on the pathways shunting the blood flow is based mainly on morphological studies, the results of which have been used to draw up a detailed classification of AVA [5]. According to this classification, different types of communications between arterioles and venules, differing both in the complexity of their structure and the mechanisms of their regulation, are placed in this group of microvessels. Besides anatomically established AVA, in various hemodynamic situations dynamic shunts of the blood flow may be formed from capillaries. The existence of such widely different types of AVA suggests that they serve different functions [12]. However, this is a problem which has not yet been adequately studied.

The aim of the present investigation was a biomicroscopic and morphometric study of adaptive reactions of AVA in pharmacologic arterial hyperemia.

EXPERIMENTAL METHOD

Forty male Wistar albino rats were used. The test object was the mesentery of the small intestine of rats anesthetized with pentobarbital (5 mg/kg, intramuscularly), into which euphylline (aminophylline) (12 mg/kg; series I) or the indigenous preparation no-shpa (2 mg/kg; series II) was injected intravenously. Visual observation of the microcirculation, morphom-

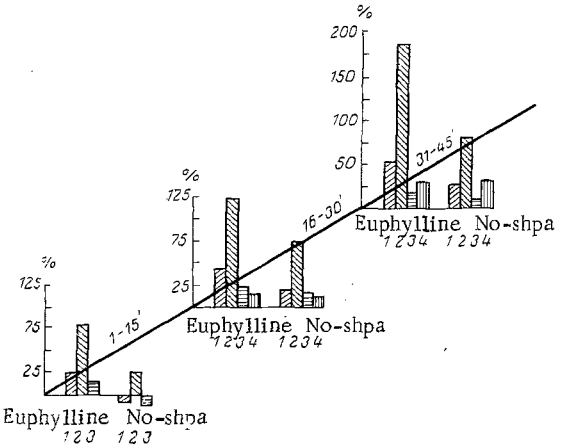

Fig. 1. Morphometric parameters of mesenteric microcirculation of rats receiving injections of euphylline (a) and no-shpa (b). Deviation of parameters from control (in %): 1) arterioles, 2) AVA, 3) muscular venules, 4) nonmuscular venules.

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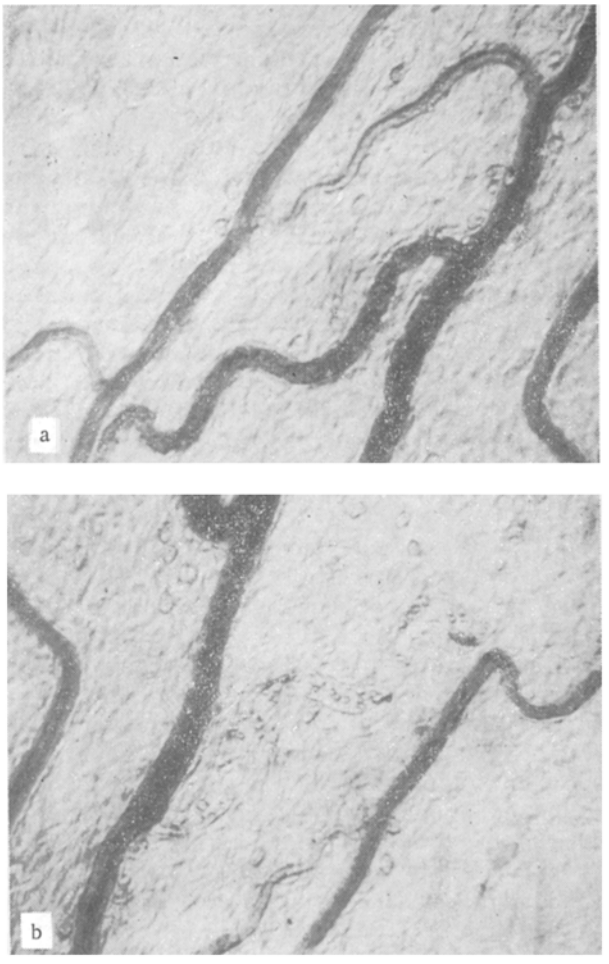

Fig. 2

Fig. 3

Fig. 2. Area of vascular network of rat mesentery 20 min after injection of euphylline. a) Functioning of proximal and distal AVA; b) inactivation of proximal AVA. 220 ×.

Fig. 3. Region of mesenteric vascular network of rat after injection of spasmolytics. Branching pathways of shunted blood flow formed by structures of the capillary network. 220 ×.

etry, and photographic recording were carried out before infusion of the drugs and at intervals during 1 h of their action. The method of investigation and the apparatus used were described by the writers previously [6].

EXPERIMENTAL RESULTS

The effect of euphylline and no-shpa on the microcirculation was similar in its main features. Differences were observed only in the first 1-3 min after injection of the drugs and consisted of initial constriction of the main microvessels and acceleration of the blood flow in them, followed by dilatation under the influence of no-shpa, whereas euphylline induced dilatation immediately, and it increased steadily. In the doses tested the myotrophic effect of euphylline was stronger than that of no-shpa. The rates and intensity of increase of dilatation of the resistive and capacitive terminal vessels differed, so that 20 min after injection of the spasmolytics into the blood stream the mean volume of the arteriolar system was increased by 44% by the action of euphylline and 24% by that of no-shpa; the mean volume of the venular components of the system was increased by only 14 and 12.5% respectively (Fig. 1). The unequal degree of dilatation of the arteriolar and venular microvessels by these drugs with myotrophic action can evidently be explained by differences in the ability of smooth muscles of these segments of the terminal circulation to preserve the mechanisms of their electromechanical coupling, which is greater in the case of the muscular venules [7, 8, 10, 11].

As a gradient of volume increase developed between the arterioles and venules a progressive increase was observed in the number of functioning AVA, the structure of which gradually became more complex. First to be activated were AVA consisting of short bridges with an intermittent blood flow. In the case of an arteriole and venule connected in parallel by several short shunts all functioned simultaneously for some time, but later those situated more proximally (and consequently, larger; Fig. 2) were inactivated and the AVA occupying a distal position along the course of the arteriole continued to function. However, not all large AVA closed, but evidently only those whose functioning overloaded the venular system in that particular region. This reaction, evidently reflex in its mechanism, is adaptive in character under conditions of developing relative insufficiency of the venular system. AVA are known to have a well-developed nervous apparatus. The extent of its development decreases with a decrease in diameter of the vessels between which the anastomosis is formed [5, 9]. Consequently, proximal AVA are the components of the by-pass circulation that respond most delicately to changes in hemodynamic characteristics in the opposing flows, and they are activated to avoid functional overloading of the venular system. At the capillary bridge level there is virtually no nervous apparatus, and their incorporation into the microcirculation is effected by the blood flow itself [1].

With an increase in the discrepancy between inflow and outflow the center of gravity of the by-pass circulation was shifted more and more from the proximal into the distal segments, until the anatomoses were formed by structures of the capillary network, especially the main channels (what Kupriyanov called half-shunts). The latter were greatly dilated under these circumstances, especially their venular portion. Capillaries arising from them also began to function and formed numerous curved bridges both outside and inside the looped main channels, with the result that the dynamic shunts thus formed acquired a lace-like appearance (Fig. 3). This increase in the complexity and length of the pathways of the by-pass circulation evidently helps to smooth out differences in velocities and pressures in the arteriolar and venular blood flows, i.e., it serves to damp the shunted blood flow. A similar picture of activation of complex, branched AVA has also been observed in the skin during reactive hyperemia by other workers [2-4]. Analysis of the morphometric data showed that the greater the gradient of volume increase of the arterioles and venules, the greater the increase in the capacity of the activated AVA (euphylline 196%, no-shpa 76%; Fig. 1).

However, the response of AVA described above is adaptive in character only up to a certain limit. If, because of lengthening and complexity of the blood drainage channels the fall in the velocity of the blood flow and the intravascular pressure is insufficient, damage to the vessel walls of this dynamic anastomosis will result, with the formation of microhematomas.

In pharmacologic arterial hyperemia an adaptive readjustment of the shunt circulation is observed, with inactivation of the largest, proximal AVA and formation of longer dynamic shunt pathways with a more complex organization in the distal portions of the terminal vascular system composed of structures of the capillary network. These changes reflect the damping function of the shunt circulation in a given hemodynamic situation.

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ELECTRON-MICROSCOPIC STUDY OF DISTURBANCES OF CARDIOMYOCYTE MEMBRANE PERMEABILITY IN IMMUNE CARDIOPATHY

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KEY WORDS: immune cardiopathy; permeability of the sarcolemma; phencarol; cardiomyocyte.

Previous investigations [1, 3, 4] showed that focal immune injury to the heart, produced by intracoronary injection of anticardial cytotoxic serum (ACS) is accompanied by considerable changes in the contractile apparatus of cardiomyocytes: contracture, fragmentation, and rupture of the myofibrils. These changes were found in the early period of development of immune cardiopathy, namely during the first 5-7 min.

In the modern view [5-7] disturbances of the myocardial contractile system of contracture type are based on an excessive inflow of calcium into the sarcoplasm of the cardiomyocytes as a result of disturbance of ionic permeability of the sarcolemma.

Since anticardial antibodies can be fixed on the sarcolemma of cardiomyocytes in the subsarcolemmal position also [10], it might be supposed that damage to cardiomyocyte mem-

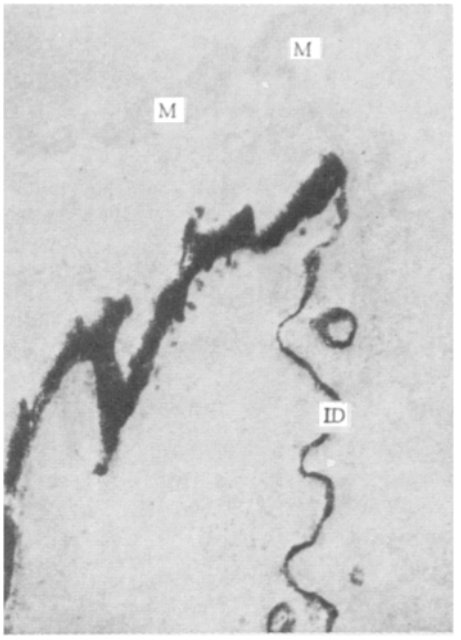

Fig. 1. Ultrastructure of intact area of myocardium. Colloidal lanthanum outlines intercalated disk (ID) and intercellular space. M) Mitochondria. 18,000 ×.

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