FUNCTIONAL STATE OF THE PRECORTICAL ARTERIES IN EXPERIMENTAL HYPO- AND HYPERTENSION

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Microscopic investigations of intravitally fixed intermediate vascular segments between the the pial arteries and the radial arteries of the cerebral cortex (the precortical arteries) in arterial hypertension produced by slow intravenous injection of noradrenalin into rabbits revealed marked constriction of these arteries and corresponding changes in the nuclei of the smooth-muscle cells in their walls. In arterial hypotension resulting from bleeding from the major arteries, no significant changes were found in the diameter of the lumen of the precortical arteries.

New methods of investigation developed in the last decade have enabled differences in the functional behavior (under different conditions) of the major arteries and the pial and cortical arteries to be detected [3]. The possible existence of other vascular mechanisms controlling the cerebral blood supply has been demonstrated morphologically. In cadaver material, for example, thickening of the muscular layer and changes in the innervation have been found in the region where the pial arteries are continuous with the radial arteries. These transitional regions are called the precortical arteries [2, 8, 9]. However, because of the absence of suitable techniques, the functional behavior of these vessels under different conditions has not yet been studied.

The object of the investigation described below was to determine, by experiments on animals, the functional state of the precortical arteries during arterial hypertension produced by slow intravenous infusion of noradrenalin, and hypotension caused by bleeding.

EXPERIMENTAL METHOD

In experiments on 19 rabbits the cerebral vessels were studied microscopically after intravital fixation at the required moment. For this purpose the vascular system of the brain was perfused under standard conditions through the internal carotid artery with fixing fluid of the following composition: 6% formalin solution made up in a mixture of equal parts of 0.85% NaCl solution and 96° ethyl alcohol.

Tracheotomy was performed under procaine anesthesia and the common carotid arteries mobilized bilaterally. On the right, after ligation of all branches (except the internal carotid), a catheter was inserted in the cranial direction for injecting the fixing fluid, and another catheter in the thoracic direction for recording the arterial pressure and for removing blood at the end of the experiment. A thick silk thread was introduced beneath the left common carotid artery to occlude the artery during perfusion of the brain. A catheter was introduced into the external jugular vein for the injection of drugs. The skull was trephined in the parietal region for direct observation on the pial arteries under a binocular microscope during the experiment. Heparin (2000 units/kg body weight) was injected intravenously after the operation.

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TABLE 1. Changes in Diameter (in μ) of Precortical Arteries of a Rabbit during Hypertension and Hypotension (M ± m)

State	Caliber of precortical arteries							
	more than 25 μ				under 25 μ			
	external diameter		internal diameter		external diameter		internal diameter	
	initial portion	active portion	initial portion	active portion	initial portion	active portion	initial portion	active portion
Control	35± 2,4	31± 2,4	28± 2,2	24± 2,4	22± 1,7	18± 1,9	14± 1,3	10± 1,5
Hypertension	34± 2,3	20± 2,9[1]	26± 2,1	11± 2,1*	24± 1,4	17± 1,4	15± 1,3	5± 1,9[1]
Hypotension	38± 2,8	30± 2,6	29± 3,0	21± 2,8	22± 1,3	20± 1,5	14± 1,5	11± 1,5

*P < 0.001.

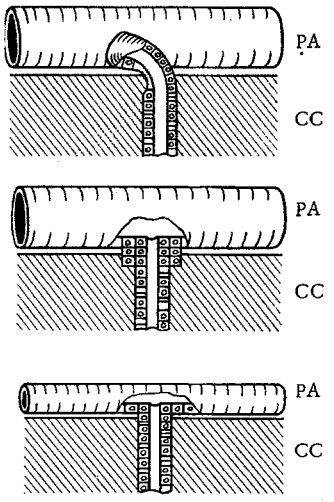

Fig. 1. Types of precortical arteries, i.e., regions of transition of pial arteries (PA) into radial, entering the cerebral cortex (CC), and character of arrangement of smooth-muscle cells in their walls.

The animal died immediately after the injection of fixing fluid. The brain was immersed for 24 h in the same fluid without alcohol. Next, under the binocular microscope, the pia mater containing all the branches of the pial arteries, their regions of transition to the radial arteries, and the initial portions of the radial arteries measuring 100–150 μ, was carefully removed. These total preparations were examined under the microscope either unstained or after staining with hematoxylin and eosin by Van Gieson's method.

The following parameters of the precortical arteries were determined with an ocular micrometer: a) the total length of the vessels from the point of their branching from the pial arteries to their entrance into the brain (where there is a characteristic thickening of the adventitia at the place where it merges with the pia mater); b) the caliber of the vessels, i.e., the external diameter of their initial portion [4]; c) the external diameter and thickness of the wall and the diameter of the lumen of different parts of the vessel; d) the length and diameter of the nuclei of the smooth-muscle cells in the vessel walls.

The investigations were carried out under the following conditions: 1) control: the brain vessels were fixed after a standard operation (either intravitally or post mortem), but without any additional form of treatment; 2) hypertension: during elevation of the arterial pressure on the average by 50–70 mm for 7–10 min following continuous intravenous infusion of noradrenalin (1×10^{-5}) solution; 3) hypotension: lowering of the systematic arterial pressure on the average to 35 mm Hg for 15–20 min following the removal of blood into the reservoir of the compensator [3].

EXPERIMENTAL RESULTS

Three types of regions of transition of the pial arteries into radial, i.e., of precortical arteries were distinguished (Fig. 1). Among the 189 vessels investigated, type I was found in 89%, type II in 7%, and type III in 4% of cases. The precortical arteries branched from pial arteries, whose external diameter in the case of type I was 75± 6 μ, type II 78 ± 6.7 μ, and type III 29 ± 5.4 μ (arithmetic mean values and confidence limits given here and subsequently). The caliber of the precortical arteries varied from 16 to 70 μ, with a mean value of 33 ± 1.6 μ. The greater the caliber of the precortical arteries, the longer the nuclei of their muscle fibers: in vessels less than 25 μ in caliber the length of the nuclei was 14 ± 1 μ, and their width 3 ± 0.08 μ; in vessels less than 25 μ in caliber the length of the nuclei was 20 ± 1 μ ($P < 0.001$), and their width 3 ± 0.006 μ. The length of the type I precortical arteries was 124 ± 15 μ, while the arteries of types II and III entered the cerebral cortex immediately after branching. Muscle cells in the wall of the precortical arteries of the various types were arranged as follows: in the case of a curvature of a vessel (type I)

their nuclei lay mainly on its outer aspect (Fig. 1); in the case of branching from a wide pial artery (type II), thickening of the media like a sphincter (Fig. 1) was present at the junction; in the case of arteries of type III, branching from interarterial anastomoses, no special features distinguished the muscular layer.

The results given in Table 1 show that along the course of the precortical arteries there is an active segment whose lumen is sharply constricted during infusion of noradrenalin; in hypotension no such constriction was found. Precortical arteries greater than 25 μ in diameter were more active than the narrow arteries. At the sites of constriction the smooth-muscle cells were evidently contracted, because the muscle nuclei were $12 \pm 0.8\ \mu$ long and $4.5 \pm 0.4\ \mu$ wide, whereas in the control, as mentioned above, they were $20 \pm 1.0\ \mu$ long and $3 \pm 0.006\ \mu$ wide [1]. When the precortical arteries contracted, their lumen was on the average 35% smaller than the lumen of the corresponding radial artery (32% greater in the control). The absence of significant changes in the width of the precortical arteries in hypotension indicates that under these conditions they are not concerned with regulation of an adequate blood supply to the cerebral cortex, unlike the pial arteries which are dilated [5, 6, 7].

The present investigation, like others published previously [3], thus shed light on the function of yet another active vascular mechanism, namely the precortical arteries, which are characterized by relatively independent responses and which may evidently play an important role in regulating the blood supply to the cerebral cortex.

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