LIGHT-OPTICAL AND ULTRASTRUCTURAL CHANGES IN THE MICROCIRCULATION OF THE KIDNEY DURING FUNCTIONAL VASOCONSTRICTION

E. P. Mel'man and B. V. Shutka

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Experiments on rats showed that after vasoconstriction of the kidney caused by adrenalin solution, in the early stages of the experiment (5 and 15 min) marked constriction of the lumen of the intramural arteries and, in particular, of the afferent arterioles develops. Ultrastructural changes in the capillary components of the renal glomeruli observed under these circumstances are a morphological expression of the effect of angiospasm and of circulatory hypoxia. In the later stages (3 and 7 days) the residual phenomena of constriction of the microcirculation in the kidney still persist. These changes characterize the state of unique mobility and of the contractile properties of the endothelial cells s caused by spasmogenic disturbance of the microcirculation.

KEY WORDS: angiospasm; blood capillaries of the renal glomeruli; circulatory hypoxia.

The morphological equivalent of transient spasm of the arterial components of the microcirculation which perform the function of "values" of the vascular system has been inadequately studied. According to the available data [2, 4, 6, 7, 12], functional angioneurospastic disorders in various organs leads to the development of foci of necrosis and hemorrhages in them. The kidney is more sensitive than other organs to circulatory hypoxia caused by angiospasm [11].

In the present investigation the effects of experimental angiospasm on the various components of the renal circulation were studied at light-optical and electron-microscopic levels.

EXPERIMENTAL METHOD

Angiospasm was induced in 30 adult albino rats by injection of 0.1 ml of 0.1% adrenalin solution into the abdominal aorta (above the origin of the left renal artery). Pieces of kidney were taken immediately after injection of adrenalin and 5 and 15 min and 1, 3, and 7 days later. The material was fixed in 1% OsO_4 solution, dehydrated, and embedded in a mixture of Epon and Araldite. Sections cut from blocks were examined under the UÉMV-100V electron microscope with an accelerating voltage of 75 kV. For the histopathological investigation of the kidney sections 3-5 μ thick were stained with hematoxylin–eosin and with fuchselin by Weigert's method, and counterstained by Van Gieson's method. Changes in the caliber of the vessels were determined morphometrically [1] and numerical results were subjected to statistical analysis. Control animals were subjected to the analogous operation but the same volume of physiological saline was injected into the abdominal aorta. All animals were kept on an ordinary diet.

EXPERIMENTAL RESULTS

Immediately and 5 and 15 min after injection of adrenalin a sharp increase was observed in the tone of the renal blood vessels. After 5 min the lumen of the interlobar, arcuate, and interlobular arteries was reduced and the area of the media was increased (Table 1). The inner elastic membrane formed tight and deep twists and turns. The nuclei of the endothelial cells were displaced to the apices of the folds of the membrane and were swollen and near together. In places where the endotheliocytes separated from the membrane, subendothelial spaces could be seen. Disorientation of the smooth-muscle fibers was found in the media. Their sarcoplasm contained numerous vacuoles and their nuclei were shortened. The outer elastic membrane was slightly twisted, with shallow invaginations and evaginations.

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TABLE 1. Indices (in μ) Characterizing Dynamics of Spasms of Renal Arteries Following Injection of 0.1% Adrenalin Solution

Time after injection of 0.1% adrenalin solution	Statistical index	Internal diameter of arteries							
		less than 20 μ		20—40 μ		40—100 μ		100—300 μ	
		diameter of lumen	area of media	diameter of lumen	area of media	diameter of lumen	area of media	diameter of lumen	area of media
5 min	M	4,3	296,1	7,0	608,3	22,0	4221,1	68,4	23433,0
	$\pm m$	0,3	21,4	0,61	25,01	1,8	466,7	7,7	1931,0
	P	>0,001	>0,001	>0,02	>0,02	>0,001	>0,02	<0,01	<0,01
3 days	M	5,0	253,7	10,0	743,5	30,2	2120,0	100,0	14586,0
	$\pm m$	0,5	17,5	0,81	80,65	2,2	231,0	11,7	2611,0
	P	>0,001	>0,2	<0,2	<0,2	<0,5	<0,5	<0,5	<0,5
7 days	M	5,8	160,8	8,4	663,8	30,4	3360,7	133,0	9321,98
	$\pm m$	0,8	14,0	1,1	101,0	3,8	808,0	9,7	818,0
	P	>0,001	<0,5	<0,1	<0,5	<0,5	<0,5	<0,5	<0,1
Normal renal arteries (control)	M	7,6	168,3	12,8	677,6	34,6	2594,8	118,8	15369,6
	$\pm m$	0,26	17,5	2,0	17,9	0,6	465,0	16,0	2744,0

With a reduction in the caliber of the arteries the narrowing of their lumen became more marked. The inner elastic membrane of the afferent arterioles assumed the form of a highly twisted spiral. The nuclei of the endothelial cells were arranged closer together in the form of a pallisade, sharply constricting the lumen of the vessel. The media, with an area of 296.1 ± 21.4 μ^2, contained a few round nuclei with irregular outlines (Fig. 1).

Electron-microscopic investigation immediately and also 5 and 15 min after injection of adrenalin revealed collapse of the glomerular capillaries. Their lumens were sharply constricted. The cytoplasm of the endotheliocytes was swollen. The nuclei were oval in shape and their nucleoplasm of average electron density. The mitochondria, with a translucent matrix, lay in the perinuclear zone. The basement membrane of the capillaries was homogeneous and irregularly thickened. The podocytes in contact with it were elongated and partly deformed. The nuclei of the epitheliocytes contained pale nucleoplasm. The cytoplasmic reticulum consisted of narrow tubules and distended sacs, the membranes of which contained ribosomes. The mitochondria were elongated and their matrix moderately osmiophilic (Fig. 2).

After 3 and 7 days the inner elastic membrane of the interlobar and arcuate arteries still remained irregularly twisted. Its fibers were more loosely arranged and in some cases were thickned or broken. In these areas the endothelium was absent and elements of the media of the vessels evaginated into their lumen. The nuclei of the smooth myocytes were deformed. The afferent arterioles had a coarser elastic stroma. The nuclei of the endotheliocytes in some regions formed groups.

In the later stages (3 and 7 days) the after-effects of experimental angiospasm were reflected ultrastructurally as persistent swelling of the cytoplasm of the endotheliocytes of the glomerular capillaries. The erythrocytes moving along the constricted capillaries were deformed and elongated in shape (Fig. 3). The nuclei of the endotheliocytes preserved their oval outlines. The mitochondria had a translucent matrix. The elements of the lamellar complex were widened. In some renal glomeruli the blood capillaries were sharply dilated and congested. In the flattened part of the endotheliocytes lining them the diameter of the fenestrations and pores was increased. Outgrowths of the mesangial cells at points of contact with the endotheliocytes projected into the lumen of the capillaries and contained many micropinocytotic vesicles. The basement membrane of the blood capillaries was thinned in some places. The cytopodia of the podocytes were low in electron density. Many vesicles were present in the cytoplasm of the epitheliocytes. The cytoplasmic reticulum had a well marked pattern of smooth and rough tubules and cisterns.

The results are evidence that during experimental vasoconstriction of the rat kidney ultrastructural changes in the components of the capillaries of the renal glomeruli are observed in the early stages (5 and 15 min) against the background of a sharp narrowing of the lumen of the arterial system within the kidney. In the later stages (3 and 7 days) residual phenomena of vasoconstriction still persisted.

The intensity of the circulation along the path to the blood capillaries of the renal glomeruli is determined by the spontaneous vasomotor activity of the interlobular arteries and afferent arterioles, which are under the influence of sympathetic vasoconstrictor fibers and of vasoactive substances [8, 13].

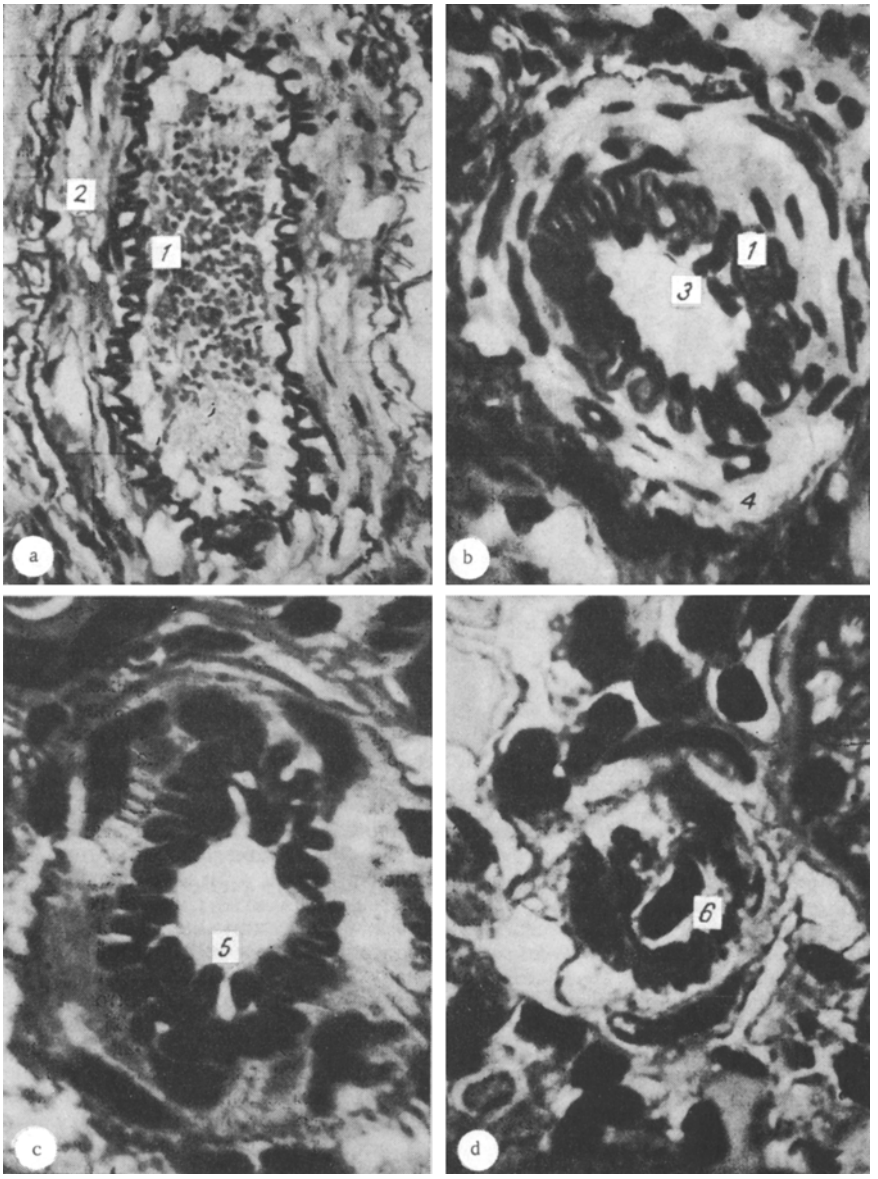
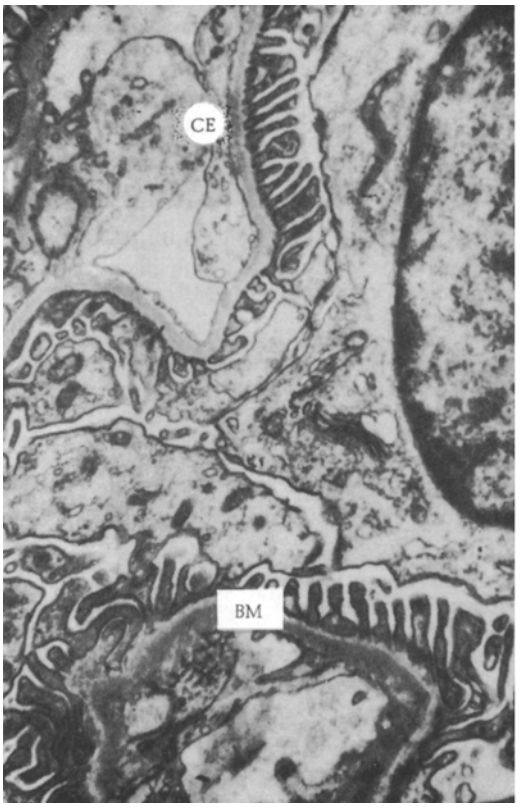


Fig. 1. Light-optical changes in wall of renal arteries of rat 5 min after injection of 0.1% adrenalin solution: a) interlobar artery (300×); 1) irregular folding of inner elastic membrane, 2) vacuolation of media; b) arcuate artery (400×); 3) swollen nuclei of endothelial cells, 4) subendothelial spaces; c) interlobular artery (1350×): 5) swollen endothelial cells forming palisade facing lumen of vessel; d) afferent arteriole (1350×): 6) of nuclei in lumen of vessel. Stained with fuchselin–picrofuchsin.

In the present experiments the action of adrenalin on the arterial system of the kidney was manifested as sharply defined spasm of all its components, as far as the arteriolar branches. The ultrastructural changes described above in the glomerular blood capillaries, which have no smooth muscle cells and are unable to contract actively, must be regarded as the result of the temporary disturbance of the circulation and hypoxia arising under those conditions. These changes (a decrease in turgor, swelling, increased translucency of the cytoplasmic matrix, narrowing of the lumen of the capillaries, etc.) are the reactive reflection of the unique mobility and contractile properties of their endothelial cells during spasmogenic disturbance of the microcirculation and in a state of hypoxia. It should be remembered that the specific granular epithelial cells in the media of the afferent arteriole of the glomerulus, belonging to the juxtaglomerular system of the kidney, are particularly sensitive to a disturbance of the transglomerular hemodynamics. Restriction of the flow of



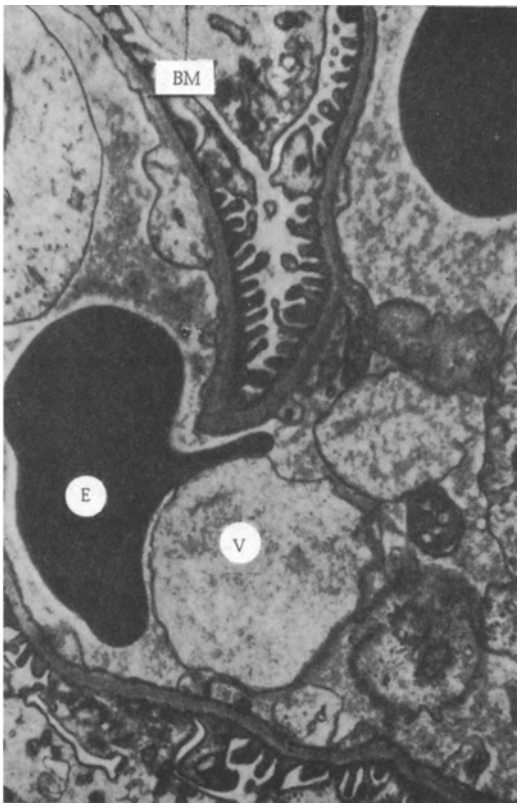


Fig. 2 Fig. 3

Fig. 2. State of individual components of glomerular blood capillaries in kidney 5 min after induction of experimental angiospasm: cytoplasm of endotheliocytes (CE) swollen; basement membrane (BM) homogeneous, and widened in some areas (13,500×).

Fig. 3. Ultrastructure of glomerular blood capillaries in kidney 3 days after injection of 0.1% adrenalin solution: large vacuoles (V) and deformed erythrocytes (E) in lumen of capillaries; thinning of basement membrane (BM) observed (10,000×).

blood toward the capillaries of the glomerulus during ischemia causes hyperplasia of these cells, and stimulates the synthesis and liberation of renin [3, 5]. Comparison of the present results with those of the writers' previous investigations indicates the high sensitivity of the kidney to circulatory ischemia, whether caused by temporary exclusion of the organ from the blood flow [9, 10], or by functional angiospasm.

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