

RENAL MICROVASCULATURE IN ACUTE RENAL FAILURE

BY

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ABSTRACT

The renal microvasculature evidenced by intraarterial injection of silicone rubber performed in 10 cases of acute renal failure from various causes demonstrated definite reduction in filling with patchy deficit in subcapsular cortex, narrowing of preglomerular arterioles, especially at the branching from the interlobular arteries in the same area, relative increase in filling of vasa afferens and efferens of juxtamedullary cortex, and of vasa recta in medulla. The same injection figures are observed in acute renal failure in dogs experimentally induced by ligation and clamping of renal arteries, and intraarterial infusion of large quantities of angiotensin II (1000 ng/kg/min). Although the etiological factors of acute renal failure are manifold and the parenchymal damage is varied as well, the vasoconstriction of preglomerular arterioles should be considered as an initial common pathway which continues in subcapsular cortex. The result of present experiments is consistent with the hypothesis that renin and angiotensin participate in the pathogenesis of acute renal failure.

INTRODUCTION

Various studies have already been done on the lesions of acute renal failure. Lucké proposed the name "lower nephron nephrosis" for the epithelial lesion of lower segments of the nephrons. Although the cause of acute renal failure is multiple, the pathogenesis of renal parenchymal damage can be divided into two major groups; (a) ischemia and (b) nephrotoxins. Oliver *et al.*¹⁾ studied these lesions by microdissection of entire nephrons, and suggested that the lesion produced by nephrotoxins is a uniform necrosis of tubular epithelium, not involving the basement membrane, while that produced by ischemia alone is fragmentation and disruption of the basement membrane. On the other hand, many investigations^{3-7,10)} have contributed to renal hemodynamics in acute renal failure. Clas-

sically, Trueta *et al.*²⁾ demonstrated, using radiological methods, a marked reduction in cortical blood flow in shock, with maintenance of normal medullary circulation. Hollenberg *et al.*^{8,9)} pointed out cortical ischemia in acute renal failure induced by different etiological factors, and suggested that it might be due to preglomerular vasoconstriction. The exact mechanism in these hemodynamic alterations in acute renal failure has not been elucidated, and nervous and some pharmacological component could also be concerned. Among them angiotensin has been considered as the most potent renal vasoconstrictor. There are numerous evidences that renin and angiotensin might play an important role in the pathogenesis of acute renal failure in man and experimental animals.¹¹⁻¹³⁾ This paper describes the renal vascular architecture of human and experi-

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mental acute renal failure, and points out clearly the site of vasoconstriction.

MATERIALS AND METHODS

1. *Studies in man:*

The renal microvasculature produced by intraarterial injection of silicone rubber was studied in the left kidney obtained 1-5 hr postmortem from ten subjects with acute renal failure, aged 23 to 68 years. Duration of oliguria, defined less than 400 ml of urinary output per 24 hr, ranged from 2 to 13 days; 4 cases within 3 days, 5 cases within 10 days, and 1 case over 10 days. The etiological factors for acute renal failure are different. Eight of these patients had acute renal failure secondary to surgery, hemorrhage, and prolonged hypotension. Two patients developed acute renal failure after administration of Neocarzinostatin and Mitomycin-C, and one patient had suffered from septicaemia. None of these patients had any previous renal diseases. The pertinent clinical data are summarized in Table 1. Twenty human kidneys with

normal function were used for control. These kidneys were removed with the full length of renal artery and vein 1-5 hr postmortem. Silicone rubber (Microfil, MV-112, Canton Biomedical Inc.) was injected into the renal arteries under pressure between 100 mmHg and 120 mmHg, after perfusion of saline added with heparin, and fixation with 2.5% glutaraldehyde solution. Injection was stopped at optimal filling of subcapsular cortex in order to make the microvasculature around the glomerulus readily visible. After vulcanization for 7 hr at room temperature, the kidneys were hardened and dehydrated in graded alcohol series, and cleared in methyl salicylate. Fine vascular arborization of silicone rubber cast was observed and photographed by the use of stereoscopy and profile projector V-16A (Nikon).

For histological examination, four blocks were taken from the right kidney, fixed in 10% Formalin, and stained with Haematoxylin and Eosin, Masson-trichrome, PAS, Yajima's PAM, and Watanabe's silver im-

Table 1. Clinical data of 10 cases of acute renal failure

Case No.	SN	Age (yrs)	Sex	Principal disease	Etiological factors of possible importance	Duration of oliguria (days)
1	²⁾ RSN 2237	55	F	Interstitial pneumonia	Cor pulmonale, hypotension	5
2	¹⁾ SN 4662	66	F	Carcinoma of the gall bladder	Obstructive jaundice, haematemesis, hypotension	3
3	SN 4669	24	M	Chronic myelogenous leukemia	Hyperuricaemia, Neocarzinostatin	7
4	SN 4687	23	F	Brain tumor	Surgery, hypotension	3
5		63	M	Hepatic cirrhosis	Surgery, hemorrhage, DIC	2
6	SN 4705	56	M	Carcinoma of the stomach	Mitomycin-C, obstructive jaundice	6
7	SN 4739	61	M	Emphysema	Cor pulmonale, haematemesis, hypotension	13
8	SN 4741	63	M	Hepatoma	Hypotension, jaundice,	5
9	RSN 2283	68	F	Carcinoma of the breast	Obstructive jaundice, hypotension	8
10	³⁾ NSN 812	34	F	Septicaemia	Hypotension, dehydration	3

1) Autopsy number of Tokyo Medical and Dental University.

2) Autopsy number of Japan Red Cross Medical Center.

3) Autopsy number of Nakano Sōgō Hospital.

pregnation methods.

2. *Experiments on acute anuria in dogs:*

Two mongrel dogs weighing 11~12 kg were used for experiments on acute anuria induced by ligation and clamping of renal arteries and intraarterial infusion of angiotensin II. The dogs were anaesthetized with Nembutal (20 to 25 mg/kg).

1) *Anuria induced by ligation and clamping of renal arteries:*

The renal arteries were exposed through a flank incision with minimal dissection in order to maintain normal innervation. The left kidney was removed 2 hr after ligation of the left renal artery, while the right renal artery was clamped for 2 hr, and then the right kidney was removed 1 hr after reflow of the blood stream.

2) *Anuria induced by infusion of angiotensin II:*

In a dog weighing 12 kg, the left femoral and renal arteries were exposed and a polyvinyl catheter was introduced into the vessels. Angiotensin II (1000 ng/kg/min) was infused into the left renal artery for 50 min and the left kidney was removed. Then the right renal artery was ligated for 1 hr and the right kidney was removed.

In each kidney, silicone rubber injection and histological examination were performed by the same methods as described above.

RESULTS

1. *Studies in man:*

Macroscopically, the kidneys of acute

Table 2. Microscopic findings of the kidneys in acute renal failure

Case No.	Glomeruli	Tubules	Interstitium
1	Slight edema of mesangial matrix	Dilatation of DCT and PCT with eosinophilic precipitate, focal destruction of Bm (1)	Marked edema
2	Thickening of basement membrane, edema of mesangium	Dilatation of lumen with hyaline and bilirubin casts, desquamation of epithelium, disruption of Bm	Edema and lymphocytic infiltration
3	Slight mesangial proliferation	Slight vacuolization of PCT (2) epithelium, granular and hyaline casts in DCT and loop of Henle	Slight edema
4	Non-remarkable	Dilatation of DCT (3) and PCT, eosinophilic precipitate and granular casts	Edema and lymphocytic infiltration
5	Fibrin thrombi in capillaries	Dilatation of DCT and PCT with desquamation and vacuolization of epithelium, granular casts	Edema and fibrillosis
6	Non-remarkable	Marked hydropic change with fine vacuoles of PCT epithelium, eosinophilic precipitate	Moderate edema
7	Slight proliferation of mesangial cells	Desquamation of epithelium, granular casts in the loop of Henle and collecting ducts, destruction of Bm	Focal edema
8	Slight increase in mesangial cells	Dilatation of lumen, desquamation of epithelium, focal disruption of Bm	Edema and fibrillosis
9	Non-remarkable	Dilatation of lumen, desquamation, necrosis of epithelium, focal destruction of Bm	Edema
10	Non-remarkable	Desquamation of epithelium, granular and pigmented casts	Slight edema

(1) Bm; Basement membrane of urinary tubule.

(2) PCT; Proximal convoluted tubule.

(3) DCT: Distal convoluted tubule.

renal failure are swollen and enlarged. The cut section revealed widened and pale cortex and dark medulla with congestion. The vascular architecture after arterial injection of silicone rubber reveals regular and compact filling of the cortex (Fig. 1), and dense "reticular" and "fascicular" capillary plexus around the tubules in normal controls (Fig. 2). On the other hand, the kidneys of acute renal failure show definite reduction, irregular distribution and patchy deficit of filling in subcapsular cortex (Fig. 3), in sharp contrast with relative increase in juxtamedullary and medullary filling. The peritubular capillary plexus is very rarefied (Fig. 4).

Histological findings of renal parenchymal lesions of 10 cases of acute renal failure are demonstrated in Table 2. The glomeruli are essentially uninvolved except for edematous thickening of mesangial matrix in many cases and fibrin thrombi in the glomerular capillaries in Case 5 (Fig. 5). Tubular lesions are characterized by dilatation of distal and proximal convoluted tubules, flattened epithelium of distal tubules, desquamation of damaged epithelium, pigmented and hyaline casts, and dense eosinophilic precipitate in the tubular lumen (Fig. 6). Diffuse and uniform vacuolization of proximal tubular epithelium is observed in Cases 3 and 5 induced by the administration of Neocarzinostatin and Mitomycin-C (Fig. 7). Destruction of tubular basement membrane is focal and scattered at distal convoluted tubules, loop of Henle, and collecting ducts in Cases 2, 7, and 8 (Fig. 8). Interstitial edema is varied and irregularly distributed in the cortex in many cases. Slight intimal thickening of arcuate and interlobular arteries is found in high aged Cases 2, 5, and 7.

Microvasculature of silicone rubber cast

in normal controls is characterized by smooth and straight running of interlobular arteries and afferent arterioles, and compact peritubular capillary plexus (Fig. 9). In acute renal failure, interlobular arteries become tortuous and afferent arterioles are narrowest at the branching from interlobular artery and then irregularly narrowing in the subcapsular cortex. Peritubular capillary plexus is very rarefied (Fig. 10). In all the cases of acute renal failure, narrowing figure is observed in 50–70% of afferent arterioles in the subcapsular cortex. The efferent arterioles in the subcapsular cortex do not reveal significant changes. In Cases 5, 6, and 7, narrowing of afferent arterioles in the juxtamedullary cortex is found. Dilatation of afferent and efferent arterioles in the juxtamedullary cortex, augmentation of filling of vasa recta from aglomerular arteries in the medulla are commonly observed in acute renal failure (Figs. 11 and 12).

2. *Studies on dogs with experimental acute anuria:*

The left kidney removed 2 hr after ligation of left renal artery is flabby and rather reddened, with histological changes of dilatation of distal convoluted tubules and amorphous eosinophilic precipitate in the lumen. The right kidney removed after clamping of right renal artery and reflow of blood stream shows anemic cortex and darkened medulla with histological lesion of marked interstitial edema, focal necrosis of tubular epithelium, and hyaline casts in the lumen (Fig. 13).

The microvasculature of silicone rubber reveals diffuse reduction in filling with widely distributed patchy deficit in subcapsular cortex, and increase in filling of vasa recta (Fig. 14). Narrowing of afferent arterioles is characteristic at the branching from the interlobular arteries. The left

Table 3. Histological findings of dog kidneys in experimental acute anuria

	Case	Procedure inducing anuria	Renal parenchymal damage
Exp. 1.	left kidney	Ligation of left renal artery for 2 hr	Dilatation of distal convoluted tubules with abundant eosinophilic precipitate, and granular casts in the lumen, slight interstitial edema
	right kidney	Clamping of right renal artery for 2 hr followed by reflow of blood stream for 1 hr	Marked interstitial edema, focal necrosis of tubular epithelium, granular and hyaline casts in distal convoluted tubules
Exp. 2.	left kidney	Infusion of angiotensin II (1000 ng/kg/min) into left renal artery for 50 min	Dilatation of distal convoluted tubules with vacuolization and necrosis of epithelium, granular and hyaline casts, interstitial edema
	right kidney	Infusion of angiotensin II and ligation of right renal artery	Dilatation of tubules with accumulation of eosinophilic precipitate, focal tubular necrosis

kidney infused with angiotensin II shows macroscopically anemic cortex and darkened medulla, and microscopically dilatation of distal convoluted tubules, slight interstitial edema, necrosis and desquamation of tubular epithelium without involvement of basement membrane, and deposition of eosinophilic precipitate in the lumen. The right kidney removed after infusion of angiotensin II and ligation of renal artery shows anemic cortex and congestive medulla, and histologically more aggravated tissue damage, i.e., marked dilatation of tubules due to accumulation of dense eosinophilic precipitate, and desquamation of necrotic tubular epithelium (Fig. 15).

Injection figures show irregular distribution and reduction in filling with patchy deficit in subcapsular cortex (Fig. 16), increase in medullary filling, prominent narrowing of afferent arterioles, remarkably at the branching from the interlobular arteries in the subcapsular cortex (Figs. 17 and 18). In these experiments on acute anuria in dogs, renal parenchymal damage is the mildest in the case of anuria induced merely by ligation of renal artery, and the severest in the case induced by infusion of angiotensin II and ligation of renal artery.

The vascular architecture of silicone rubber in the right kidney with anuria induced by infusion of angiotensin II and ligation of renal artery is the most similar to that of human cases of acute renal failure. The renal parenchymal changes in experimental cases are summarized in Table 3.

DISCUSSION

We first examined whether characteristic injection figures of acute renal failure have some artefacts due to postmortem change, or injection technique, or not. The same vascular architecture of irregular narrowing of preglomerular arterioles in acute renal failure cannot be detected in any of the control kidneys. Thus it is admitted that the functional vasoconstriction might survive later for several hours postmortem. Both in human and experimental acute renal failure, the vasoconstriction of preglomerular arterioles is confined to subcapsular cortex, in contrast to relative increase in filling of afferent and efferent arterioles in juxtamedullary cortex and dilatation of vasa recta in medulla. The difference of microvasculature between subcapsular and juxtamedullary cortex may depend upon not only the anatomical situation, but also

the different function of nephrons.^{14,15)}

From anatomical viewpoint, vas efferens of subcapsular cortex is of small calibre, being as wide as the corresponding vas afferens and forms reticular and fascicular capillary plexus around tubules. Vas efferens in the juxtamedullary cortex is larger in calibre than that of vas afferens, and forms reticular capillary plexus or vasa recta.¹⁶⁻²⁰⁾ This basic anatomical difference in postglomerular microvasculature may be related to the occurrence of vasoconstriction, as its own physiological characteristics of vascular response.

Although the measurement was not made regarding comparison between the response of vas afferens and that of vas efferens, only the former showed vasoconstriction and no significant changes were seen in the latter. The vascular response of preglomerular region in subcapsular cortex, more precisely at the proximal portion of vas afferens branching from the interlobular artery, would be dominant in haemodynamic alterations in acute renal failure.

The real etiological factors of these 10 cases of acute renal failure are manifold and difficult to be determined readily. Jaundice was noticed in Cases 2, 6, 8, and 9, but only Cases 2 and 9 revealed hyperbilirubinemia clinically and bilirubin casts histologically. These two cases are classified as "hepato-renal syndrome". Cases 6 and 8 have low bilirubin value and are thought to be due to ischemic origin, as are Cases 1, 4, 5, 7, and 10. Cases 3 and 6 seemed to be nephrotoxic in origin, for they showed a prompt allergic reaction, shock, and anuria immediately after administration of each drug.

Renal parenchymal damage is varied in these 10 cases of acute renal failure. Focal destruction of tubular basement membrane is found in Cases 1, 2, 7, and 8 of rela-

tively prolonged oliguria. Epithelial lesions varied from vacuolization to complete necrosis; vacuolization in Cases 3 and 6, desquamation of epithelium in Cases 1, 2, 5, 7, 8, 9, and 10. Interstitial edema is not so prominent in Cases 3, 6, and 10, and irregularly distributed in many cases. Thus the degree of parenchymal damage in acute renal failure is not necessarily correlated to the duration of oliguria, but rather to the strength of local vasoconstriction, reflected by patchy deficit of filling of silicone rubber cast.

Experiments on acute anuria induced by large quantities of angiotensin II in the dog demonstrated that angiotensin II may affect directly the preglomerular arterioles in subcapsular cortex and maintain vasoconstriction, although the possibility of direct effect of angiotensin II on the tubular epithelium cannot be excluded.

We think at present that an initial common pathway in acute renal failure induced by various causes is vasoconstriction which occurs and continues at the site of preglomerular arterioles in subcapsular cortex.

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EXPLANATION OF FIGURES

Plate 1

- Fig. 1. Vascular pattern of silicone rubber in normal kidney (42 years, female), showing compact and regular filling of the cortex.
- Fig. 2. Microvasculature of silicone rubber of cortex in normal kidney (42 years, female), showing compact "reticular" and "fascicular" peritubular capillary plexus. (Original magnification; $\times 20$). Scale bar indicates length of 500μ .

Plate 2

- Fig. 3. Reduction in filling of silicone rubber with patchy deficit in subcapsular cortex, in contrast with increased medullary filling in Case 10.
- Fig. 4. Rarefaction of peritubular capillary plexus and increase in medullary filling of silicone rubber in Case 10. (Original magnification; $\times 20$). Scale bar indicates length of 500μ .

Plate 3

- Fig. 5. Diffuse fibrin thrombi in the glomerular capillaries in Case 5. (Haematoxylin and Eosin stain; original magnification; $\times 120$)
- Fig. 6. Granular casts in the tubules and interstitial edema of the medulla in Case 10. (Masson-trichrome stain; original magnification; $\times 120$)
- Fig. 7. Marked hydropic change with fine vacuoles in the proximal tubular epithelium in Case 6. (Masson-trichrome stain; original magnification; $\times 120$)
- Fig. 8. Flattened epithelium of distal convoluted tubules, interstitial edema, and focal disruption of tubular basement membrane in Case 2. (Masson-trichrome stain; original magnification; $\times 120$)

Plate 4

- Microvasculature of silicone rubber. Scale bars indicate length of 100μ .
- Fig. 9. Smooth and straight running of interlobular artery and afferent arterioles and fine peritubular capillary plexus in normal kidney (26 years, female). (Original magnification; $\times 120$)
- Fig. 10. Serpiginous interlobular artery, narrowing of afferent arterioles, and rarefaction of peritubular capillary plexus in Case 6. (Original magnification; $\times 120$)

Plate 5

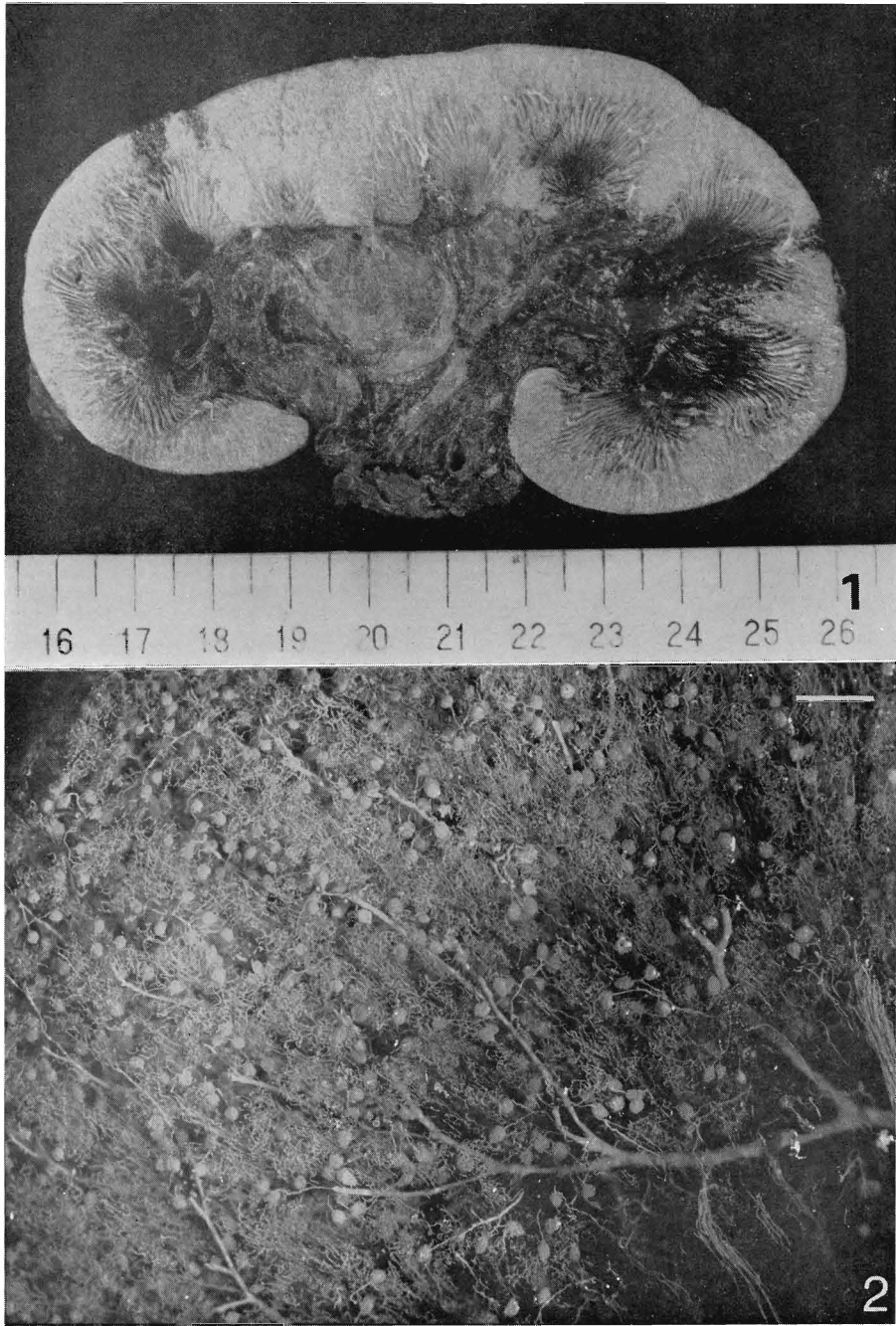
- Microvasculature of silicone rubber in juxtamedullary cortex. Scale bars indicate length of 100μ .
- Fig. 11. Efferent arterioles in normal kidney (31 years, female). (Original magnification; $\times 120$)
- Fig. 12. Dilatation of afferent and efferent arterioles, and increase in filling of vasa recta from aglomerular artery in Case 4. (Original magnification; $\times 120$)

Plate 6

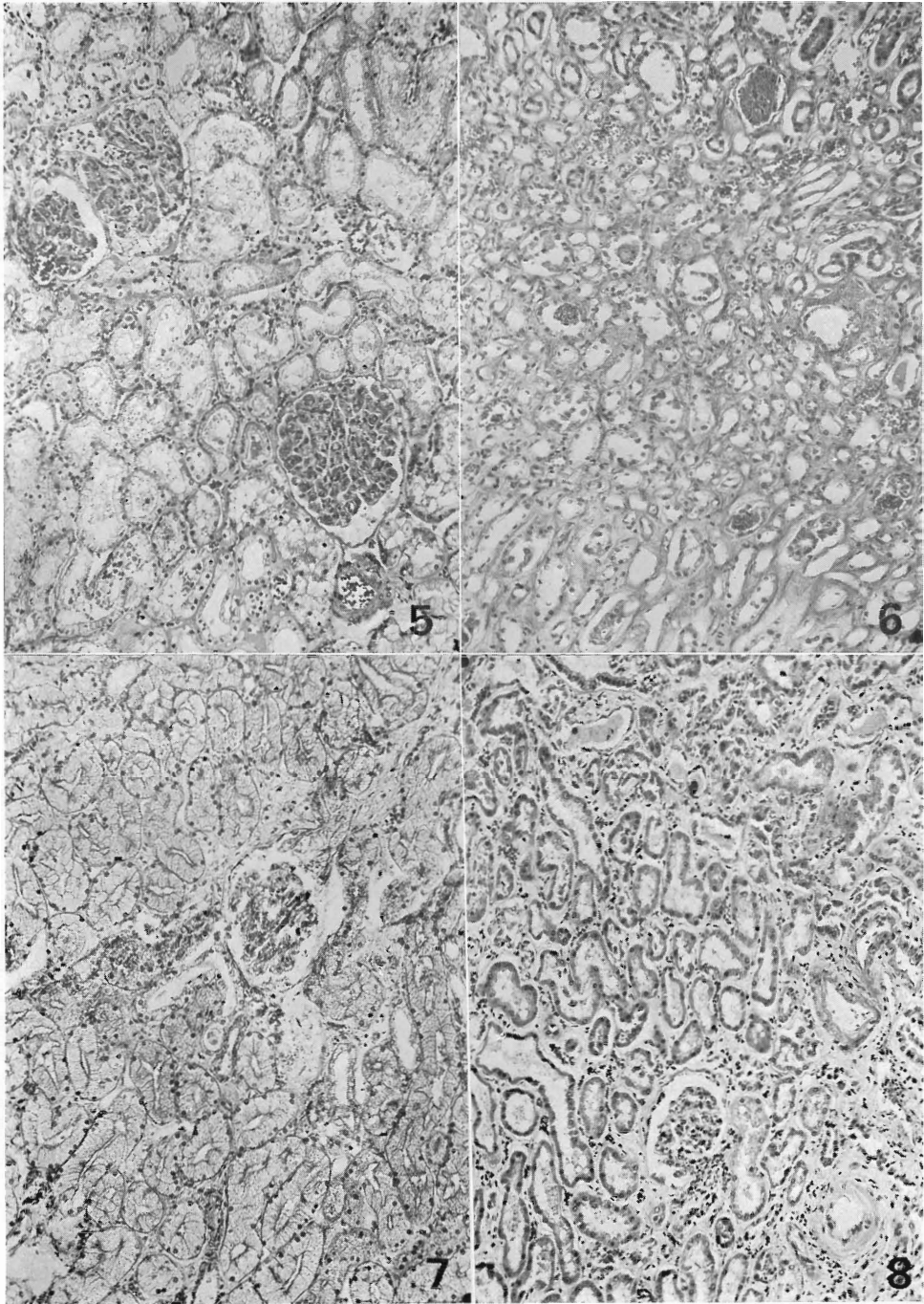
- Figs. 13 and 14 show the right kidney of a dog with anuria experimentally induced by clamping of right renal artery followed by reflow of blood stream.
- Fig. 13. Moderate interstitial edema, dilatation of distal convoluted tubules, focal necrosis of tubular epithelium, and hyaline casts in the lumen. (Haematoxylin and Eosin stain; original magnification; $\times 120$)
- Fig. 14. Microvasculature of silicone rubber showing diffuse reduction in filling with patchy deficit in subcapsular cortex and relative increase in medullary filling.
- Figs. 15 and 16 show the right kidney with anuria induced by infusion of angiotensin II and ligation of right renal artery.
- Fig. 15. Marked dilatation of tubules due to accumulation of dense eosinophilic precipitate, and desquamation of tubular epithelium. (Haematoxylin and Eosin; original magnification; $\times 120$)
- Fig. 16. Injection figure of silicone rubber showing irregular distribution and reduction in filling with patchy deficit in subcapsular cortex.

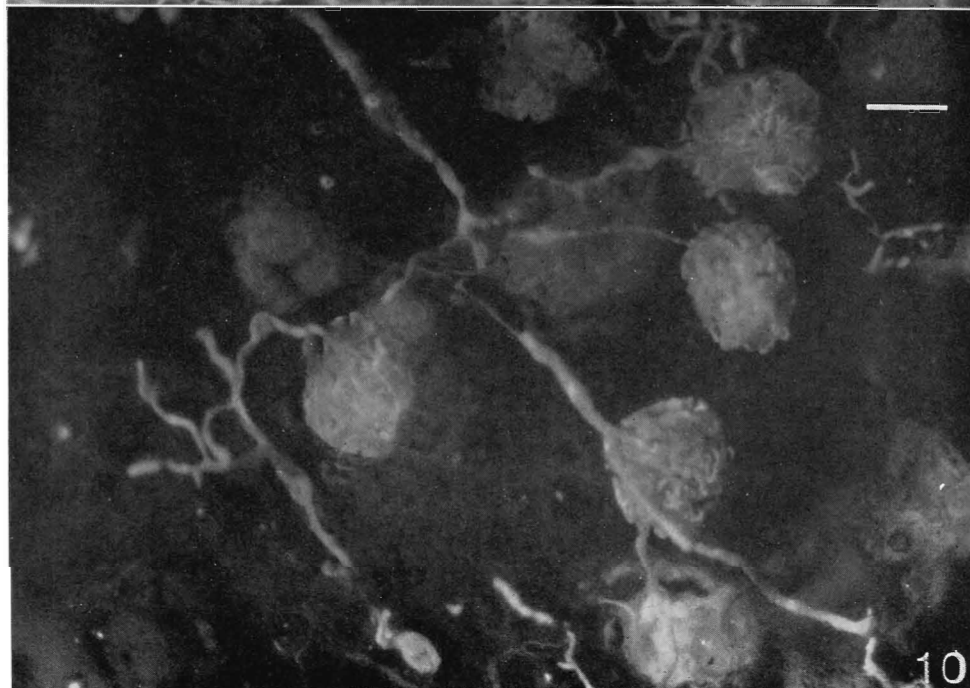
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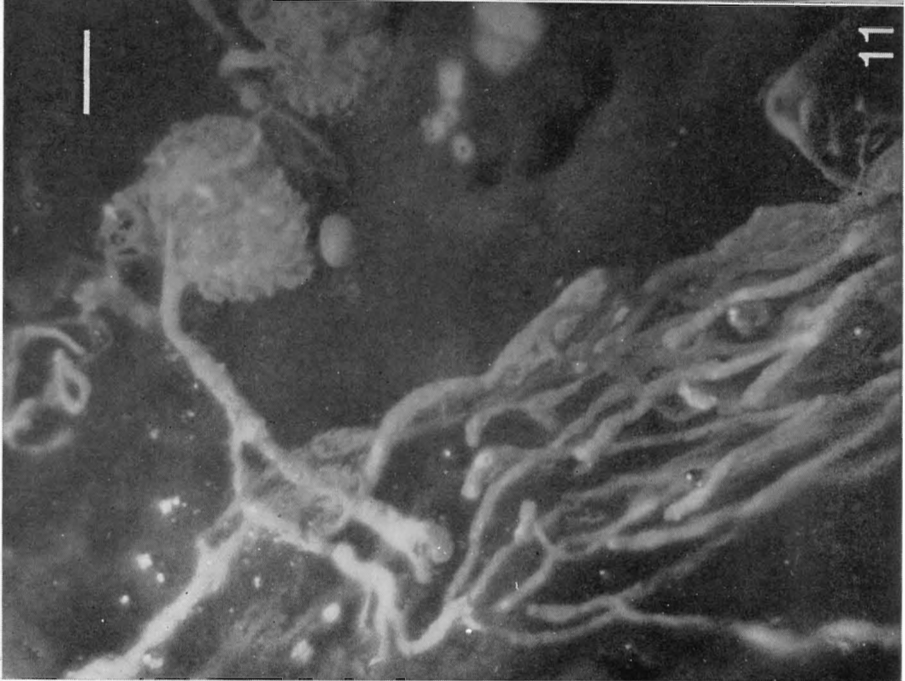
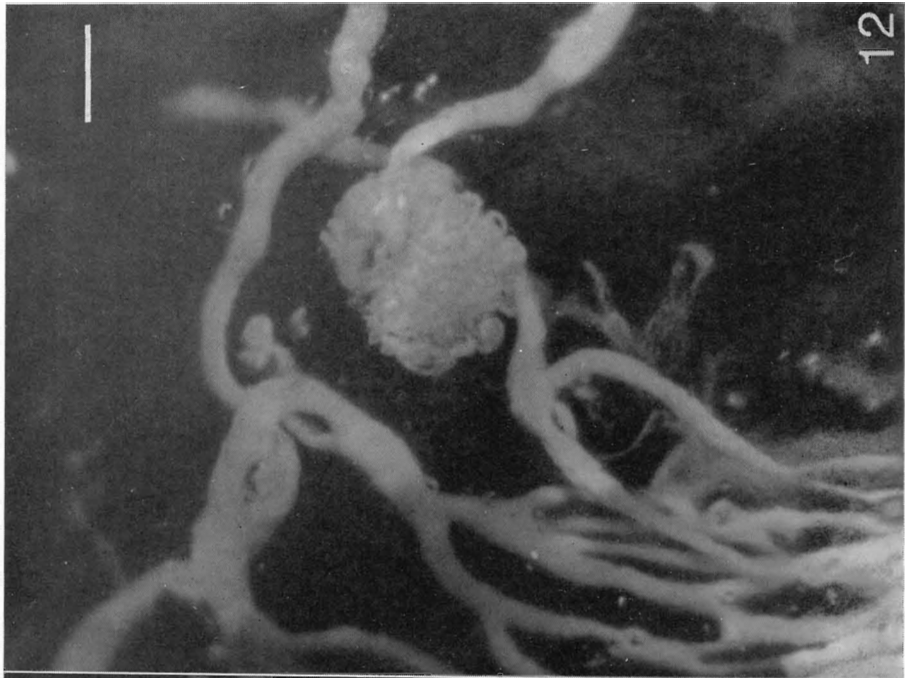
- Microvasculature of silicone rubber in dog kidney. Scale bars indicate length of 100μ .
- Fig. 17. Smooth running of afferent arterioles and fine peritubular capillary plexus in normal dog kidney. (Original magnification; $\times 120$)
- Fig. 18. Prominent narrowing of afferent arterioles and rarefaction of peritubular capillary plexus in subcapsular cortex of the right kidney in the case of anuria induced by infusion of angiotensin II and ligation of right renal artery.

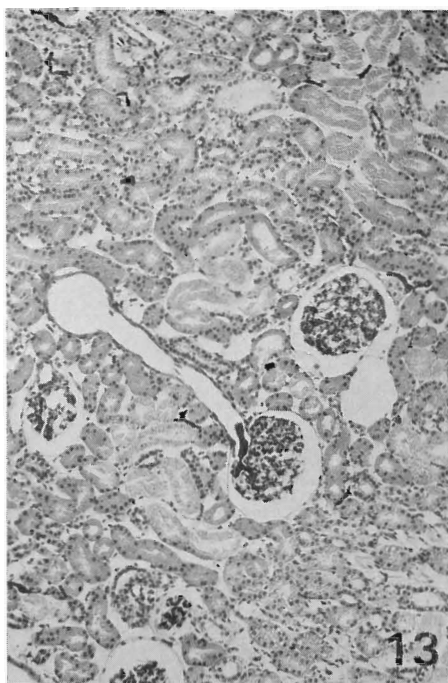








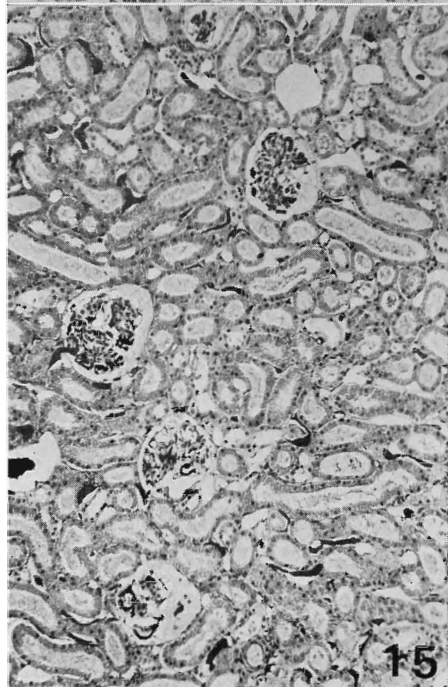




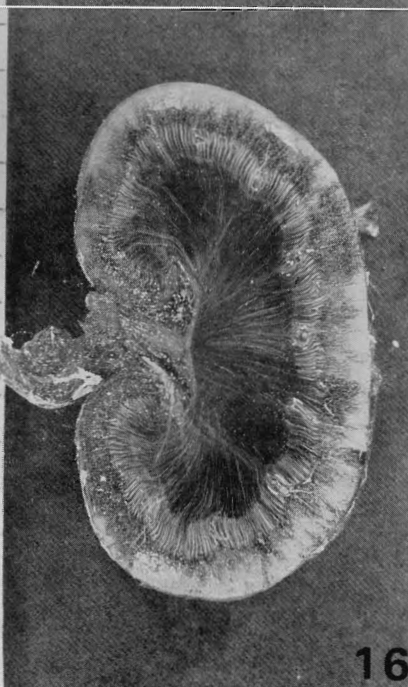
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