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HISTOCHEMICAL CHANGES IN THE RENAL PARENCHYMA AFTER COMPRESSION OF THE RENAL ARTERY IN THE DOG

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Introduction

The effect on the renal parenchyma of the compression of the renal artery has been examined, among others, by the Hungarian authors Zádor and Balogh [33]. They have studied rat kidneys after compression for 1 to 20 minutes, and dog kidneys after compression for 30 minutes. The kidneys were removed 10 to 60 days later and stained with haematoxylin-eosin. From those of foreign authors, the investigations of Montserrat [11] are remarkable, who observed capillary changes after compression of the renal artery. In the well-known surgical and pathological textbooks of Kirschner [22], Bier, Braun and Kümmel [8], Lichtenberg [23], Anderson [1] only brief mention is made of the lesions ensuing after temporary compression of the renal artery. Recently, numerous authors have discussed the problem e. g. Babics, Hamilton [21], Badenock [4], but no exhaustive examinations have been published.

Our own examinations have been based on the assumption that after compression of the renal artery for a short time, when the histological changes are still absent or not yet appreciable, alterations of a subtler nature, not demonstrable by clinical and histological methods, might occur, first of all in the physiological and enzymatic functions of the kidney. These alterations influence only the most delicate vital functions of the organ, without producing more conspicuous morphological changes. We have, therefore, sought an answer to the questions, 1. what is the duration of the compression following which no morphological changes occur, but functional disorders may already be observed histochemically? 2. In which parts of the organ are the enzymatic or histophysiological functional disorders most marked, and do they cease promptly, or only after a prolonged time, and are they irreversible?

In our examinations especial attention has been paid to the juxtaglomerular complex, this extremely sensitive functional regulating system of the renal parenchyma. Becher [6], Schloss [29], as well as earlier investigators, like Möllendorf [25], Zimmermann [34], Goormaghtigh [16, 17, 18], Appelt [2], Feyrter [12], Clara [9], Ludwig [24] have all emphasized the high reactivity

of the complex (manifesting itself with a swelling of the cells, an increase of pigmentation), as the sensitive indicator of renal function, acting by way of the regulation of the vascular lumina.

To study the tendency for regeneration after compression we have extended our investigation to the observation of capillarisation. Finally, to draw conclusions, we have compared the results of the above-mentioned examinations with the findings of other authors.

Methods

The examinations were performed on dogs of roughly identical weight and the same sex, kept on identical food. In six series of five dogs each, under Evipan anaesthesia laparatomy was made and the right renal artery was compressed with rubber clamps. The compression was maintained for 2 minutes in the first series and for 5, 15, 30, 60 and 120 minutes, respectively, in the further series, each consisting of five animals ; the first animal of every series was killed with gas one hour after operation, the others were sacrificed in the same way on the lst, 3rd, 7th and 14th day. The kidneys were removed and worked up histochemically. In order to avoid an eventual inflammation interfering with the histochemical evaluation, the animals were treated with 200 000 I. U. of crystalline penicillin daily for 3 days. At the same time care was taken to observe identical feeding conditions and fluid intake. In some cases, mainly with a view to comparing with observations of capillarisation, the PAH clearance was also examined, but its values, obtained photometrically with Kedvessy's method were not found reliable.

In all cases the left kidney served as control, and the right one was worked up. The removed organs were washed with physiological NaCl, and then placed in a fixation, corresponding to the reaction to be carried out, and then subjected to the histochemical reactions serving to demonstrate capillarisation, lipids, proteins, alkaline phosphatase, acid phosphatase, the juxtaglomerular complex. Staining was made with haematoxilin-eosin and Azan to reveal histological changes. The different procedures were as follows :

Capillarisation

After fixing in neutral formaldehyde, frozen sections 100 to 300 μ thick were prepared and treated with benzidine.

Lipids

The removed tissue was cut into slices 2 mm thick, and fixed in 10 per cent formaldehyde. Frozen sections 10 to 25 μ thick were cut and stained with a 0,1 per cent alcoholic solution of Sudan Black B for two hours. In order to control localisation, the material was treated for 20 minutes with a saturated aqueous solution of Nile blue sulphate, or with Romeis' colloidal Sudan III Solution.

Nucleoproteins

For demonstrating parenchymatous elements rich in nucleoprotein we have adopted the methylgreenpyronine method. After fixation in Carnoy's fluid, sections 6 to 10 μ thick were prepared from paraffin blocks and treated according to Pappenheim-Unna's method.

Alkaline phosphatase

The excised tissue was cut in 3 mm thick slices and fixed in 80 per cent alcohol at 5° C for 24 hours. Frozen sections 10 to 15 μ thick were prepared and incubated at 37° C for 4 hours, mostly in a mixture of x + beta glycerophosphates and in a few instances, in betaglycerophosphate ; as a buffer Veronal was used, with a pH optimum of 9,5. As a control, for each reaction 3 sections were examined, inactivated either by heat, or 10 per cent acetic acid, or non-incubated.

Acid phosphatase

The material was prepared according to the Newman method ; after fixation as in the preceding method, it was incubated with lead nitrate ; the pH optimum of 5 was assured with an acetate buffer (0,05 M). For both the alkaline and the acid phosphatase reaction, ammonium sulphite was used.

The juxtaglomerular complex

For demonstrating the pigments of the juxtaglomerular complex we have applied the staining procedure described by Wilson [32]. After fixation in Bouin's fluid, sections 5 to 10 μ thick were prepared from paraffin blocks and stained with crystalviolet (30 drops of a 1 per cent solution to 20 per cent ethyl alcohol). As a counterstain, 0,4 per cent light green solution was used.

Parenchyma

The renal parenchyma was stained with haematoxylin-eosin ; for examination of the interstice, staining with Azan was made. The material was fixed in 8 per cent formaldehyde ; the sections 5 to 10 μ thick were cut from paraffin blocks.

Observations

Capillarisation

In series I, II, and III (duration of compression, 5, 10 and 15 minutes respectively), the number of active capillaries diminishes for 24 hours only. In series IV, V, VI (duration of compression, 30, 60 and 120 minutes respectively), there were ischaemic areas especially in the cortex, even after 7 days, particulary following compression for 2 hours. Temporary ischaemia was followed in each case by a compensatory hyperaemia. The relative capillary density is designated in the Table with +, ++ and +++, where ++ corresponds to the normal density.

It should be noted that in no instance have we observed the arteriovenous anastomoses, situated extremely densely in the cortex (360/cm^2), nor the renal sinusoids connected with them, as described by Spanner. In connection with this we agree with Trueta et al. that similar structures mentioned in the literature may be considered in all likelihood as artefacts due to injection.

Lipids

From among the methods applied, Sudan Black B gave the most expressive picture. In series I, II, III there was already a marked accumulation of lipids in the area of Henle's loop and of the upper convoluted tubes. In series IV, V, VI the changes were not more definite. The extremely massive lipid accumulation, which essentially may be considered as a fatty degeneration, persisted after the

regression of the other histochemical changes, particularly in series IV and VI, and complete regeneration did not ensue even after 14 days.

It was remarkable that from among the three kinds of stain, Nile blue sulphate brought about a vivid blue and Sudan Black a deep bluish-black colour in the lipids, a sign of predominant phospholipid accumulation. On the other hand, the fact that even the colloidal solution of Sudan III did not produce an appreciable picture, indicates that lipid accumulation was intracellular and not interstitial.

Alkaline phosphatase

Alkaline phosphatase activity declined perceptibly in series I, II and III. A considerable decline in activity was observable firstly in the more distally situated tubular segments (Henle's loop), and later on also in the area of the proximal tubules. Despite the rapid decline of activity, nonspecific phosphatases were soon regenerated even in series IV and VI, hence even after prolonged compression. Activity is resumed as early as on the 4th to 5th day. In tissues fixed for a longer period (1 week or more), we have observed a particulary marked decline of activity. We have, however, considered this the consequence of an autolytic breakdown of the desmoenzyme and did not assess it.

Acid phosphatase

The decrease of acid phosphatase activity became more definite after prolonged compressions, which shows that it is less sensitive than alkaline phosphatase activity, or else, that the pH, arising in the compressed kidney approximates better the optimum for acid phosphatase.

The juxtaglomerular complex

In the sections treated with the staining method of Wilson, there is a conspicuous increase in the amount of pigments in the juxtaglomerular cells. The presence of a great number of paravascular, intertubular and Goormaghtigh cell groups (Sockelplasmodium) is particulary marked. Less marked but relatively more numerous are the macula densa per visual field. The most sensitive reaction of the juxtaglomerular complex, the swelling of cells could not be assessed, owing to the swelling caused by parenchymal degeneration also present in the sections. It should be noted that methyl-green pyronine is a very selective stain of the granules in the juxtaglomerular complex ; these granules consequently become easier to recognize than after staining with haematoxilin-eosin.

Summary of histological and histochemical changes within the single series

Series	Duration of compression	Day of examination	Lipid	Alkal. phosph.	Acid phosph.	Capillarisation	Sign. of degeneration
I.	2′	1	N.	+	N.	++	∅
		3	N.	+	N.	++	∅
		7	N.	N.	N.	+++	∅
		10	N.	N.	N.	++	∅
		14	N.	N.	N.	++	∅
II.	5′	1	N.	++	+	+	∅
		3	N.	+	+	+	∅
		7	N.	N.	N.	+	∅
		10	N.	N.	N.	++	∅
		14	N.	N.	N.	+++	∅
III.	15′	1	N.	++	+	+	∅
		3	+	+	+	+	∅
		7	+	N.	N.	+	∅
		10	+	N.	N.	++	∅
		14	N.	N.	N.	+++	∅
IV.	30′	1	+	+++	++	+	+
		3	++	++	+	+	+
		7	++	N.	N.	+	+
		10	++	N.	N.	+	+
		14	+	N.	N.	++	+
V.	60′	1	++	++++	+++	+	++ — +++
		3	+++	++	++	+	++ — +++
		7	+++	+ (N)	+	+	++ — +++
		10	+++	N.	N.	++	++ — +++
		14	+++	N.	N.	+++	++ — +++
VI.	120′	1	++	++++	+++	+	++++
		3	++++	+++	+++	+	++++
		7	+++	++	++	+	++++
		10	+++	N.	N.	+	+++
		14	+++	N.	N.	++	+++

N.: normal
\+ mild reaction (capillary ischaemia)
++ : medium reaction (normal capillarisation)
+++ : strong reaction (capillary hyperaemia)
++++ : extreme reaction

Although the multiplication of the juxtaglomerular cellular elements must be considered with some reservation because the specificity of the Wilson stain has not yet been established and statistically confirmed — nevertheless, on comparing all the facts, the increase in number and size of the juxtaglomerular structures appears incontestable. This seems to support the opinion of BECHER [6], that the distinctive features of juxtaglomerular cells are not always identical, but are the corollary of a temporary, functional state.

Nucleoprotein

No appreciable difference between compressed and control side has been obtained with Pappenheim—Unna's stain.

Histological observations

The findings have borne out the observations of ZÁDOR and BALOGH [33]. The changes became more conspicuous after compression for 30 minutes (group IV). Considering that we had compressed only the renal artery, the usual congestion did not appear in the sections. In the mildest cases (duration of compression, 30 minutes ; sacrifice after 3 to 7 days) first of all a faintness of nuclear staining, and a simultaneous swelling and granulation of nuclei were observable. The parenchymal degeneration characteristic of the majority of cases was preceded by the disappearance of the brush border of the tubular cells. The outline of the cells was indistinct or invisible ; the cells themselves were inflated, those of the tubular epithelium shifted apart ; some of these had become detached and remained in the lumen of a tubule. The interstice was usually rich, the glomeruli shrunken. In some cases (duration of compression 120 minutes) there were very grave changes tending to necrosis. Round cell infiltration or secondary granulation were absent, probably because of the comparatively short period (maximum 14 days), passed since the compression, in which regeneration could not yet have started. This accounts for the fact that in no case were there symptoms of regeneration. It must be noted, that the severe changes affected primarily the cortical area, while the medullary substance was more or less intact.

Discussion

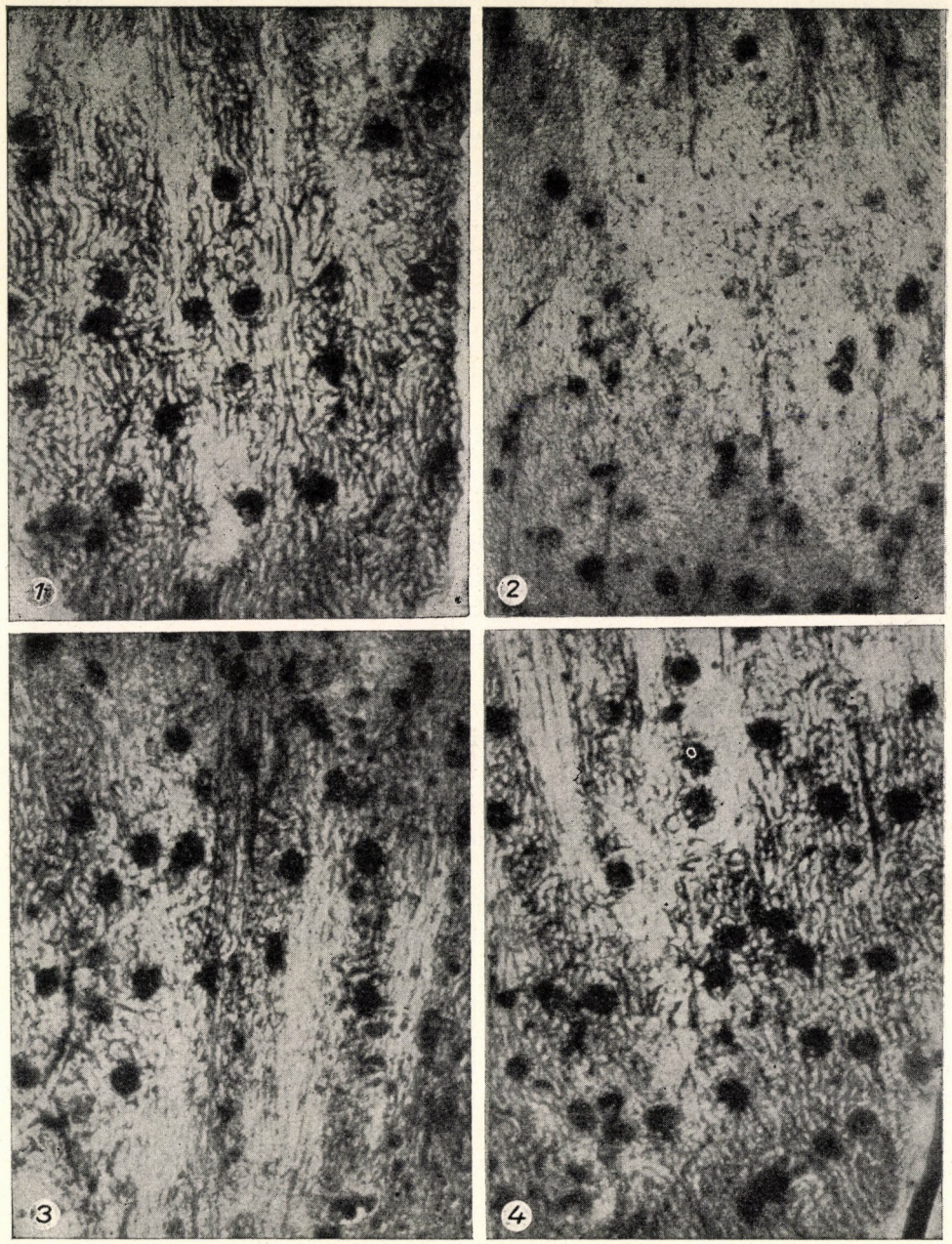
We again wish to emphasize that the examinations were made on dogs, so that the conclusions drawn from the results may not be valid for man. Our purpose has been to demonstrate the latent, most delicate functional disorders

brought about by compression of the renal artery, disorders not to be established by clinical or common histological methods. The duration of compression required for inducing manifest changes was found to be 30 minutes. After compression for a shorter time, no definitely appreciable morphological changes arose ; still, there appeared extensive functional alterations in the parenchyma, which could be demonstrated by histochemical methods.

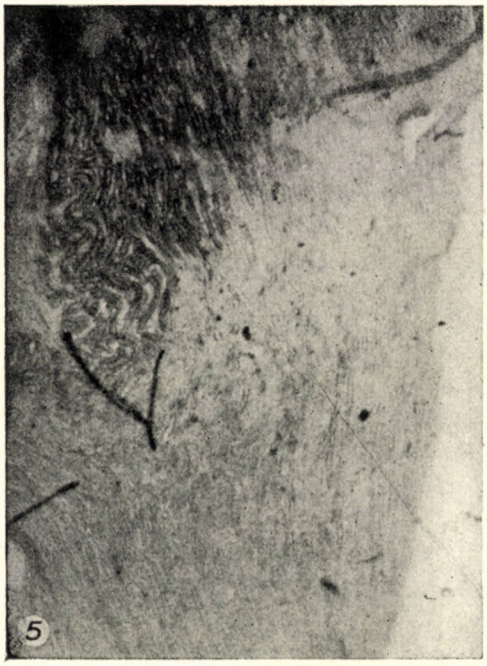
The most sensitive part of the renal parenchyma is apparently the upper tubular system ; it was here that the first changes appeared, and it was here that subsequently they were most conspicuous. In case of slighter changes demonstrable solely by histochemical methods, the functional disturbance of balance was soon normalized, in 3 to 5 days. On the other hand, extensive morphological changes revealed also histologically did not regenerate within the 14 day period examined.

The great sensitivity of alkaline phosphatase is of a certain physiological interest. If we accept the interpretation of ANDERSON, i. e. that enzymes, with the help of the dephosphorylation of hexosephosphatases, play the chief part in tubular sugar resorption, and, moreover, if we add the well-known phenomenon that the activity of alkaline phosphatases considerably declines in disturbances of tubular function (e. g. hydronephrosis) — the conclusion suggests itself that a disturbance of sugar resorption is the earliest symptom induced by compression of the renal artery.

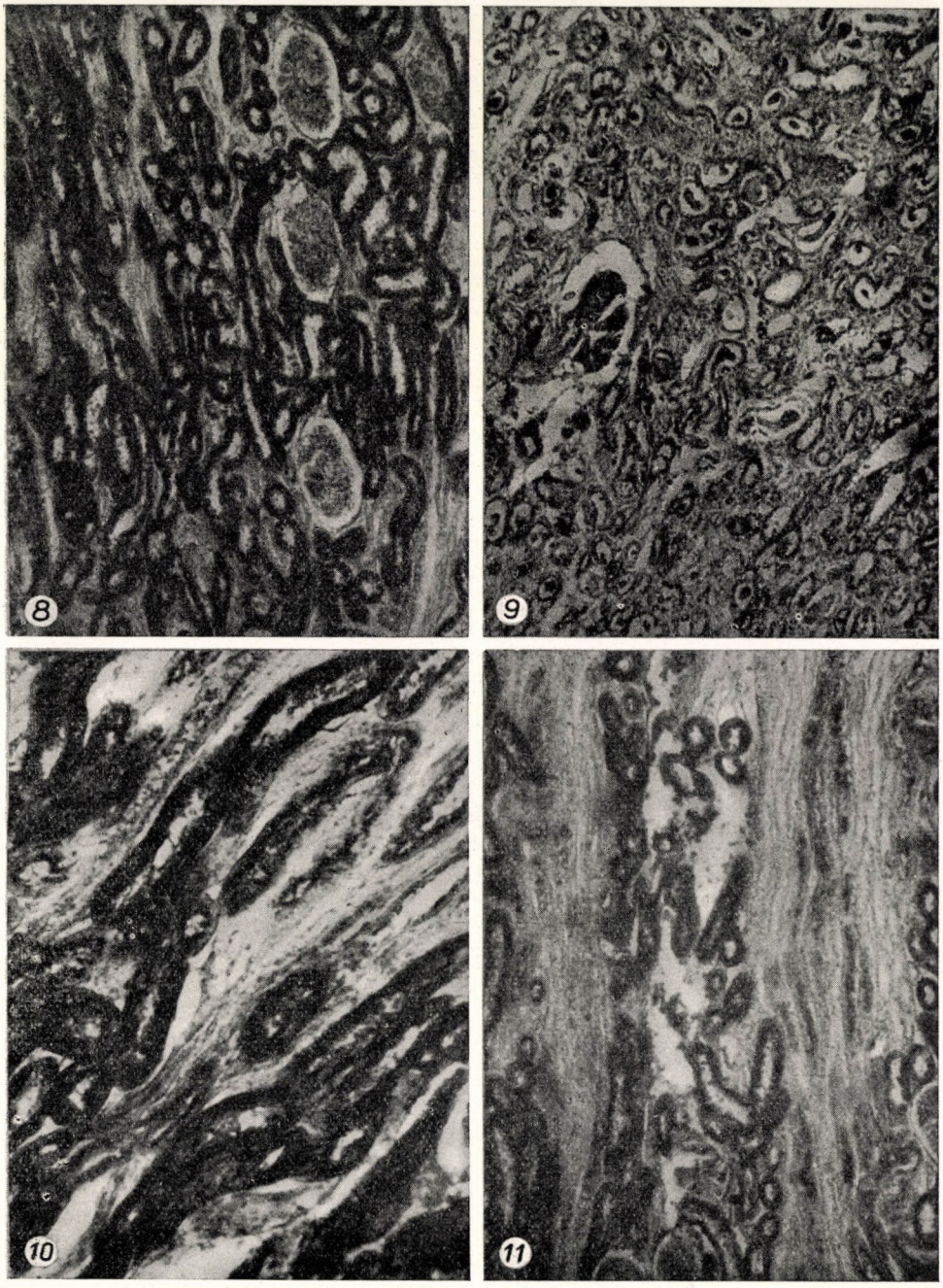
When reviewing the lipid stains, we have already mentioned that on the basis of their staining properties we consider as phospholipids the intercellular lipid accumulation observed in the upper tubules. This is indicated, in addition, by the very marked reaction to Sudan Black B — although, according to GÖMÖRI and his school, this stain is said to react to phosphatides with a special colour (the result was controlled with Baker's method specific to phosphatides) — as well as the fact that with Nile blue sulphate a colour characteristic of acid lipids develops. The decline of alkaline phosphatase activity occurring parallel with the increase of lipids suggests the possibility that the increase in the amount of phospholipids is actually a secondary process consequential to the decline of activity of the enzyme that effects the breakdown.



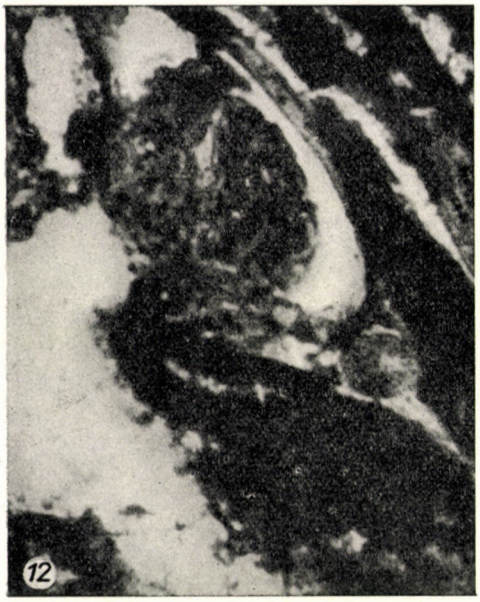
Changes of capillarisation in the renal cortex. *Fig. 1.* Normal renal cortex. x 120 Normal capillary density. *Fig. 2.* Renal cortex, 72 hours after arterial compression for 120 minutes. Magnification as above. Few shrunken glomeruli. In the central part of the picture marked capillary ischaemia. *Fig. 3.* Renal cortex, 72 hours after arterial compression for 30 minutes. Magnifications as above. In the marginal part of the cortex, relative capillary hyperaemia; in the medullary rays, relative capillary ischaemia. *Fig. 4.* Renal cortex, 14 days after arterial compression for 30 minutes. Magnifications as above. Complete regeneration, relative capillary hyperaemia in some areas

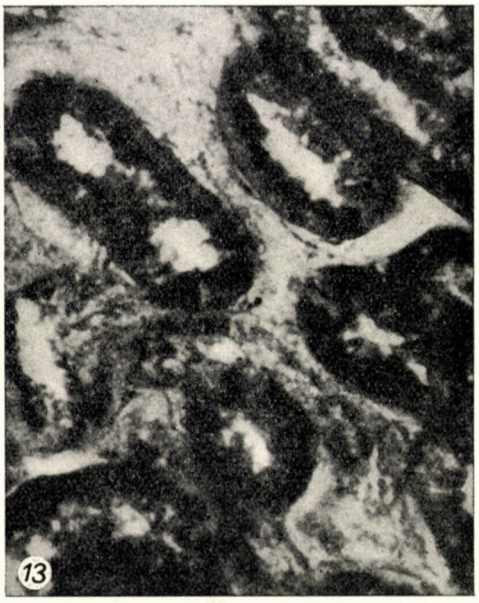


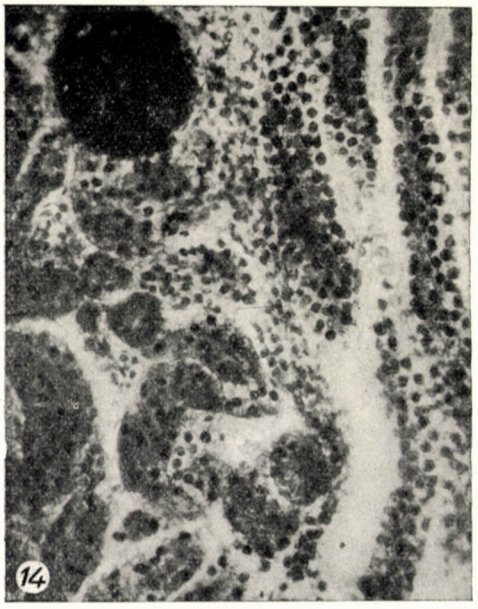
A) Lipid degeneration in the proximal tubules. *Fig 5.* Normal kidney Sudan Black B staining. × 12 Physiological lipid accumulation in the region of the proximal tubules. *Fig. 6.* Kidney, 7 days after arterial compression for 60 minutes. Staining, magnification, as above. Considerable lipid accumulation in the region of the proximal tubules chiefly in Henle's loop. *Fig. 7.* The same as No. 6, under high power (× 250). Granula degeneration in all tubular parts. Degenerative symptoms along with intracellular lipid accumulation

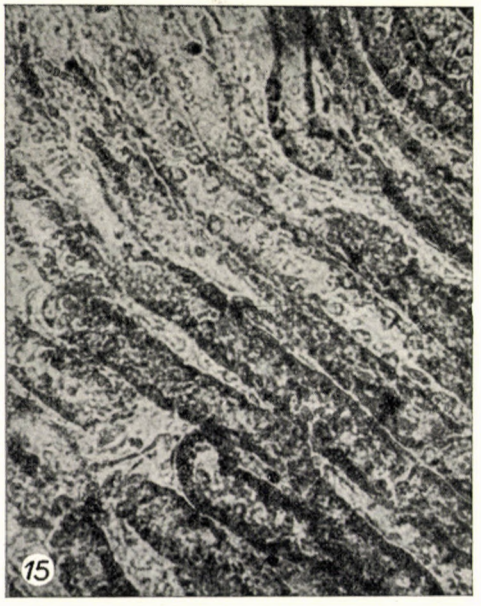


Changes of alkaline phosphatase activity. *Fig. 8.* Normal kidney. × 250 Normal enzymatic activity. *Fig. 9.* Kidney, 24 hours after arterial compression for 120 minutes. Technique as above. Considerable decline of activity. *Fig. 10.* The same as No. 9, under high power (× 500) enhanced enzymatic activity in Bertini's columns, slighter activity in the medullary rays. *Fig. 11.* Kidney, 7 days after arterial compression for 15 minutes. Technique as above. × 250. Complete regeneration. The enzymatic activity in the medullary rays in normal

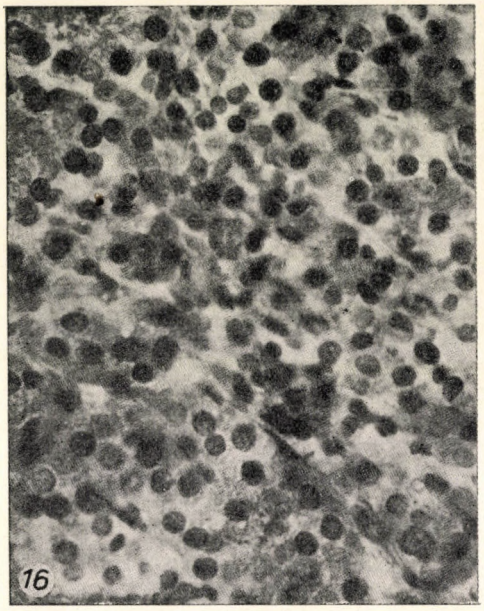


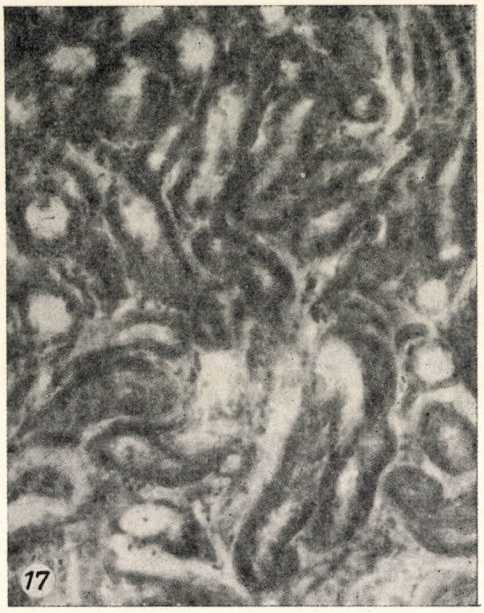


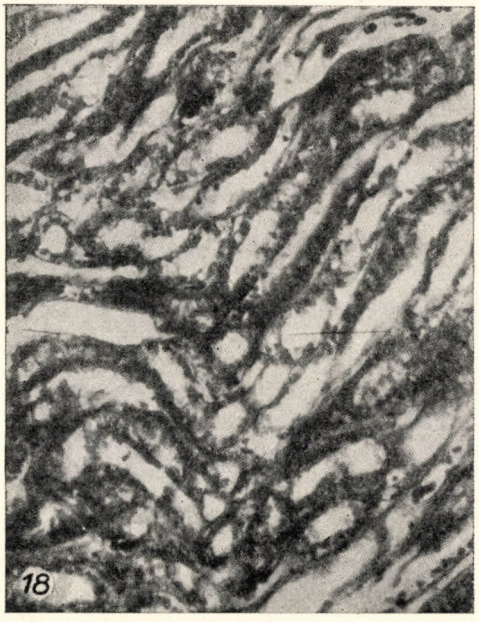


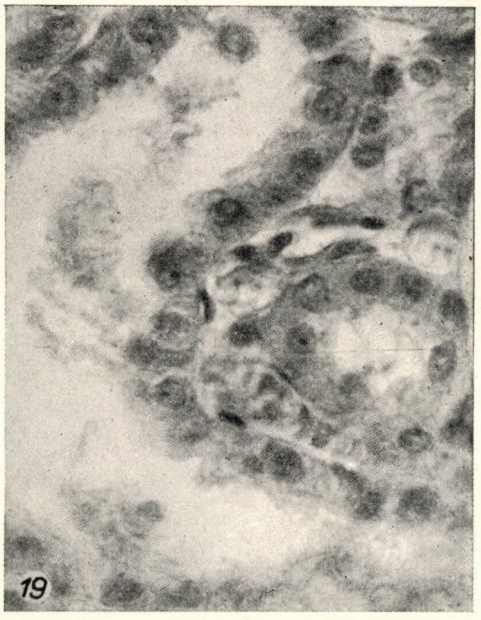


A) Alkaline phosphatase reaction. *Fig. 12.* Kidney on the 14th day after arterial compression for 60 minutes. Beside the glomerular pole a paraportal cell is'et with weaker reaction. Above the dark homogeneous dens ty is Sockelplasmodium × 600. *Fig. 13.* The same as Fig. 12, × 800 Completely restored enzymatic activity in the tubu'es which still show signs of degeneration. *B)* Changes of acid phosphatase activity. *Fig. 14.* Normal kidney × 250. Intense nuclear reaction. enzymatic activity specially marked in the glomerulus. *Fig. 15.* Kidney, 24 after arterial compression for 60 minutes. Technique and magnification as above. Decreased enzymatic activity in all areas. Signs of degeneration in the tubular epithelium









A) Acid phosphatase reaction. *Fig. 16.* Kidney, 7 days after arterial compression for 30 minutes. × 800. Completely restored enzymatic function *B)* histological changes. *Fig. 17.* Kidney, 3 days after arterial compression for 30 minutes. Haematoxylin-eosin stain. ×250. Swollen tubular epithelial cells. Faint nuclear staining, contours indistinct. Parenchymal degeneration. *Fig. 18.* 3 days after arterial compression for 60 minutes. Technique, magnification, as above. Grave degeneration. Epithelial cells shifted apart, some of them necrosed. Tissue structure has disappeared. Cell fragments in the tubular lumina. *Fig. 19.* Fig. 18 under high power (×800). Brush border disappeared, nuclear staining faint. The nuclei are inflated, in some cellls missing. A number of detached epithelial cells in the lumina

Summary

Temporary compression of the renal artery is a procedure frequently adopted in urological surgery for rendering the operative area ischaemic. The purpose of our examinations has been to demonstrate the histochemical changes not yet attended by clinically and histo-pathologically apparent symptoms that occasionally occur with arterial compression.

To study these changes, experiments in dogs have been carried out. A shorter or longer time after, the renal artery has been compressed for from 2 to 120 minutes, the right kidney was removed and subjected to ten different kinds of histochemical and histological examinations.

It has been found that compression for less than 30 minutes elicited changes which soon regenerated, and were observable only by means of finer histochemical methods. After compression for more than 30 minutes, a more severe condition was produced, demonstrable by usual histological methods. A characteristic feature was a marked decline of the alkaline and acid phosphatase activity suggestive of a disturbance in tubular sugar resorption. Lipid degeneration, the product of which has been considered to be phospholipids, ensues soon after prolonged compression. In contrast with the histochemical changes, morphologically apparent changes (e. g. parenchymatous degeneration, necrosis) did not regenerate within the 14-day examination period. The changes affected first of all the renal cortex whereas the medulla was left comparatively intact.

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ГИСТОХИМИЧЕСКИЕ ИЗМЕНЕНИЯ В ПОЧЕЧНОЙ ПАРЕНХИМЕ ПОСЛЕ ПРИЖАТИЯ ПОЧЕЧНОЙ АРТЕРИИ У СОБАК

Е. ШОМОДЬИ, Т. ДОНАТ и Й. БАЛИНТ

В урологической хирургии для обескровливания операционной области часто применяется временное прижатие почечной артерии. Целью опытов, проведенных авторами, было выявление гистохимических изменений, проявляющихся внутри обратимого времени прижатия, при которых еще не наблюдаются клинически и патологически выявляемые симптомы. Для создания изменений авторы проводили операции животных. У собак более или менее долгое время после прижатия правой почки в течение от 2—120' они удалили почки и обработали последних десятью различными гистологическими способами.

Результаты исследований показывают, что прижатие в течение менее 30' вызывает изменения, которые можно выявить только с помощью тонких гистохимических способов, и регенерация которых происходит сравнительно быстро. В случае почек, прижатие которых проводилось в течение свыше 30' проявляется уже более серьезное, патогистологически выявляемое состояние. В первом случае *характерным является сильное уменьшение щелочно- и кислотно-фосфатазной активности, что позволяет сделать заключения о возможности нарушения тубулярной ресорбции сахара.* В случае прижатия в течение более длительного срока рано появляется липоидное вырождение трубчатой системы, причем авторы того мнения, что химическим веществом этого вырождения можно рассматривать фосфолипоиды. В противоположность гистохимическим изменениям более грубые морфологические изменения (паренхиматозная дегенерация, некроз) в течение 14 дневного срока исследования не проявляли регенерации. Изменения наблюдались в первую очередь в корковом веществе, в противоположность сравнительно неповрежденному состоянию мозгового вещества.

HISTOCHEMISCHE VERÄNDERUNGEN IM NIERENPARENCHYM NACH KOMPRESSION DER ARTERIA RENALIS VON HUNDEN

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In der urologischen Chirurgie wird zur Entblutung des Operationsgebietes häufig eine zeitweilige Kompression der Nierenarterie angewandt. Es wurden Versuche zur Untersuchung der eventuell innerhalb der reversiblen Kompressionszeit auftretenden histochemischen Veränderungen durchgeführt, bei denen klinisch oder pathologisch nachweisbare Symptome noch nicht in Erscheinung treten. Zur Erzeugung solcher Veränderungen wurden kürzere oder längere Zeit nach 2—120' dauernder Kompression der rechten Nierenarterie des Hundes beide Nieren herausgenommen und nach zehn verschiedenen histochemischen, bzw. histologischen Verfahren bearbeitet.

Laut den Befunden ruft eine weniger als 30' dauernde Arterienkompression nur mit feineren histochemischen Methoden nachweisbare Veränderungen hervor, die verhältnismässig schnell regenerieren. Bei Nieren, deren Arterie länger als 30' komprimiert wurden, tritt ein schwererer, auch pathologisch definierbarer Zustand auf. Im ersten Fall ist *die starke Verminderung der* Alkali- und Säurephosphatasen-Aktivität charakteristisch, *woraus auf eine Störung der tubulären Zuckerresorption gefolgert werden kann.* Bei längerer Kompression tritt eine lipoide Degeneration des oberen tubulären Systems früh auf; als chemischer Stoff dieser Degeneration können die Phospholipoide betrachtet werden. Im Gegensatz zu den histochemischen Veränderungen zeigen die gröberen morphologischen Veränderungen (parenchymatöse Degeneration, Nekrose) während der 14 tägigen Untersuchungsdauer keine Regeneration. Sie betreffen in erster Reihe die Rindensubstanz, während die Marksubstanz verhältnismässig intakt bleibt.

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