ON THE COMPENSATORY HYPERTROPHY OF THE ISCHEMIZED KIDNEYS

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In 1959 one of the authors of this article [1] developed a method of producing a standard degree of stenosis in the arteries of rats in chronic experiments by means of a stenotizing spiral. Experimental test of this method showed that after the application of a spiral with a spire diameter of 0.3-0.35 mm upon one of the renal arteries the corresponding kidney underwent atrophy, and the contralateral — intact — kidney underwent hypertrophy. If, however, a spiral with a spire diameter of 0.3-0.35 mm was pulled over one of the renal arteries and stenosis of the artery in the second kidney was simultaneously produced by the application of another spiral with a spire diameter of 0.26 mm, the first kidney (in which the stenotic artery had a diameter of 0.3-0.35 mm) did not undergo atrophy, but hypertrophy.

It was the aim of the present investigation to confirm this observation experimentally.

EXPERIMENTAL METHOD

We carried out 3 series of experiments. In the first series, stenosis of the renal artery was produced in one kidney; in the second series, stenosis — of different degree — was produced in both renal arteries; and in the third series, stenosis was produced in the renal artery of a kidney which had earlier undergone hypertrophy. In all experiments, the stenotizing spiral was left on the arteries until the end of the animals’ life.

White rats of both sexes and of coinciding weight (between 158 g and 375 g) were used for the experiments. The following parameters were used to judge the degree of renal hypertrophy and atrophy: the size of the kidneys, the size of the glomeruli and of the tubules, and the general histological picture (in sections stained with hematoxylin-eosin, picrofuchsin and Sudan III). The size of the glomeruli and of the tubules in the proximal part of the nephron was measured by means of an eyepiece-micrometer. The size of the glomeruli was measured within the limits of the renal corpuscles, summing up their total size along the greater diameter, and the size of the tubules was measured at the basal membrane. In each section the diameter of 30 glomeruli and of 30 contorted tubules of the first order was measured in 3 different parts of the kidney (in the upper, middle and lower third of the organ). In each kidney the measurements were carried out in 3 sections after which the average values were calculated.

To assess the functional state of the kidneys the non-protein nitrogen level was estimated in the blood of arbitrarily selected animals.

EXPERIMENTAL RESULTS

I. Experiments Involving the Production of Stenosis in One Renal Artery.

Stenosis in one of the renal arteries was produced in 18 rats: in 11 rats with the aid of a spiral with a spire diameter of 0.3-0.35 mm, and in 7 rats by means of a spiral with a spire diameter of 0.4-0.46 mm.

If the arteries were narrowed by means of a spiral with a spire diameter of 0.4-0.46 mm, atrophy of moderate degree developed within 45 days in the corresponding kidney, without compensatory hypertrophy of the contralateral kidney.

If the arteries were narrowed by means of a spiral with a spire diameter of 0.3-0.35 mm, marked atrophy of the ischemized kidney and compensatory hypertrophy of the intact kidney could be observed, beginning from the 12th-14th day. At later stages the signs of atrophy and sclerosis in the ischemic kidney became more marked and...
by the 200th-300th day the organ was completely replaced by fibrous, frequently petrified tissue. In these cases the hypertrophy of the intact kidney reached a high degree. The atrophy of the ischemic kidney became manifest in the diminished size of the glomeruli and the contorted tubules.

The average size of the kidney, the artery of which had been rendered stenotic throughout the duration of the experiment (12-300 days), reached 12.6 x 8 x 5 mm; as compared to 18 x 10 x 8 mm in the controled animals. (All control values described in this paper were obtained on 10 intact rats). The average size of the contralateral kidney reached 19.3 x 12.3 x 9.3 mm.

The diameter of the glomeruli in the ischemic kidneys decreased to 60 - 65 μ, compared to 68 - 76 μ in the control animals (i.e. a decrease of 8-11 μ) and the diameter of the tubules reached 23- 28 μ instead of 30-38 μ (i.e. a decrease of 7 - 10 μ). In the contralateral kidney the glomeruli increased to a diameter of 80 - 125 μ (on the average to 99.6 μ) and the contorted tubules of the first order reached a diameter of 36 - 45 μ (on the average 40 μ).

Investigation of the non-protein nitrogen in the blood of arbitrarily selected animals revealed in 4 rats with a stenotized renal artery, and in 6 control rats that the non-protein nitrogen levels are very similar in both groups in the experimental animals the level varied between 21 and 36 mg% (on the average 29 mg%) and in the control rats between 21 and 46 mg% (on the average 37 mg%).

If one generalized the results of the first series of experiments one comes to the conclusion that experimental stenosis of the main artery in one kidney leads to the atrophy of the kidney and to the hypertrophy of the contralateral kidney. The nitrogen excretion by the kidneys remains on the whole unimpaired.

II. Experiments Involving Simultaneous Stenosis of Different Degree in both Renal Arteries

In the second series of experiments simultaneous stenosis of both renal arteries was produced in 17 rats. The artery of one of the kidneys was narrowed with the aid of a spiral similar to that used in the first series of experiments with a spire diameter of 0.3 - 0.35 mm and the artery of the other — contralateral — kidney was narrowed by means of a spiral with a smaller spire diameter equal to 0.26 - 0.28 mm. In various groups between one third and two thirds of all animals used for the experiments survived. The highest mortality could be observed within the first 10 - 14 days of the experiments.