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Renal Artery Streching and Renovascular Hypertension Experimental Studies

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Summary. Renovascular hypertension was produced in rabbits by displacing one kidney to the iliac fossa in order to produce traction on the renal artery. Hyperplasia of the juxtaglomerular apparatus was seen in the kidney where the renal artery was streched, whereas no JGA hyperplasia was witnessed in the untouched kidney of rabbits. Whether traction on the renal artery is the sole or a contributing factor to the pathogenesis of fibrous dysplasia of the arterial wall remains a moot point.

Key words: Traction on renal artery, renal hypertension, hyperplasia of juxta-glomerular-apparatus.

The frequent co-incidence of nephroptosis and renal artery fibroplasia in patients with renovascular hypertension has aroused the interest of a number of investigators (1, 2, 6). Kaufman (2) suggested that the "accordion-like" action on the renal artery created by repeated ascent and descent of the mobile kidney might be of significance in the pathogenesis of fibromuscular dysplasia. Previous evidence has indicated that ptosis of the kidney may cause orthostatic hypertension (4, 5) and correction of the ptosis in some instances has resulted in significant improvement of hypertension. In 1938 Leadbetter and co-workers (3) reported a case of hypertension in a young male patient with an ectopic (pelvic) kidney. It was pointed out in this report that the renal artery and vein were under considerable tension and the artery was described as showing subintimal fibroplasia. Although there are experimental studies to suggest that traction of the renal artery may produce fibromuscular dysplasia in dogs (6) controlled investigations to determine whether renal artery traction per se may produce hypertension by impeding renal bloodflow have not been carried out to our knowledge.

Materials and Methods

Female rabbits weighing between three and five kilograms were anaesthetized with intravenous pentothal-sodium. A left femoral artery cutdown was performed and a polyethylene feeding tube (8 F.) was inserted as far as the aorta to monitor blood pressure. The anaesthesia was maintained at as a light level as possible to avoid hypotension, which was noticed when the animal received to much anaesthetic. Following the replacement of the catheter a mid-line incision was made and in most instances the left kidney was exposed and entirely mobilized. The animals were divided into two groups. Group I consisted of eight rabbits in which the renal artery was dissected free, an umbilical tape was slung around the renal artery and connected to a weight in order to stretch the artery for 15 seconds. A constant weight of 297 g. was found to be slightly less than that necessary to avulse an isolated common iliac artery of a rabbit. The abdomen was then closed and postoperative studies were conducted eight weeks later. Group II consisted of eleven rabbits, in which the kidney was mobilized and then fixed to the iliac fossa in order to produce a long term streching effect on the renal artery. (Fig. 1) After eight weeks the rabbits in both groups were anaesthetised and aortic blood pressure was obtained after insertion of a feeding tube. The feeding tube was then connected to a Statham P 23 Db transducer and recorder. Following this determination aortography was performed. Aortography was made by a single injection of 10 to 15 cc. of 60% Conray (Fig. 2). Subsequently the animals were sacrificed and the kidney and renal arteries removed for histological studies.

Results

Two rabbits in group I were lost because of technical failures. Six rabbits recovered and were

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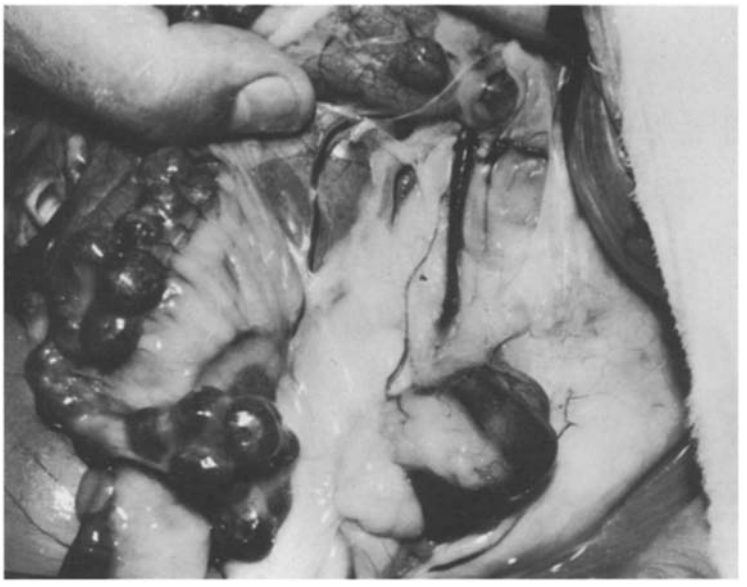

Fig. 1. Left kidney eight weeks after displacement down to the ipsilateral iliac fossa

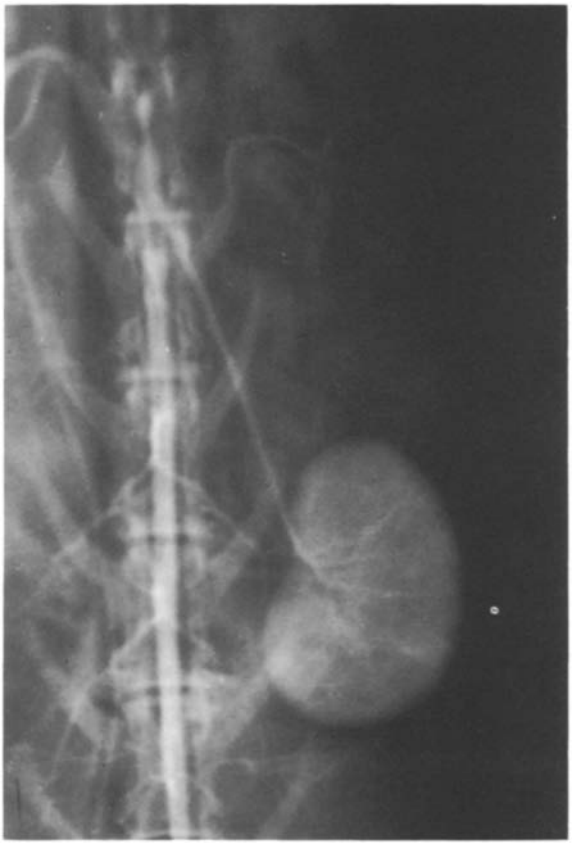

Fig. 2. Aortography eight weeks after displacing the left kidney to the iliac fossa. Significant elongation and narrowing of the renal artery

Table 1. Aortic blood pressure studies

	preoperative	8 weeks post nephropexy	elevation
no 1	90/60	160/100	50/40
no 2	100/70	rabbit died post operatively	
no 3	80/62	116/92	36/30
no 4	100/75	136/100	36/25
no 5	90/60	rabbit sacrificed	
no 6	80/60	136/88	56/28
no 7	95/60	140/100	45/40
no 8	112/80	died post anaesthesia induction	
no 9	108/96	128/108	20/12
no 10	85/55	108/96	23/41
no 11	100/60	112/104	12/44

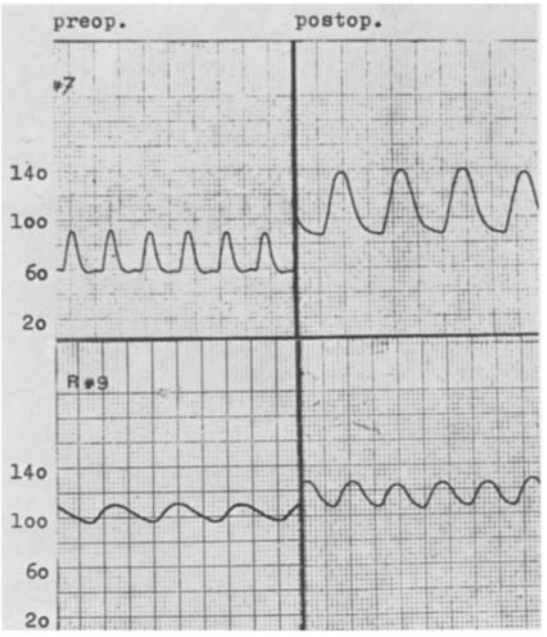

Fig. 3. Preoperative and postoperative blood pressure studies in rabbit no. 7 and 8

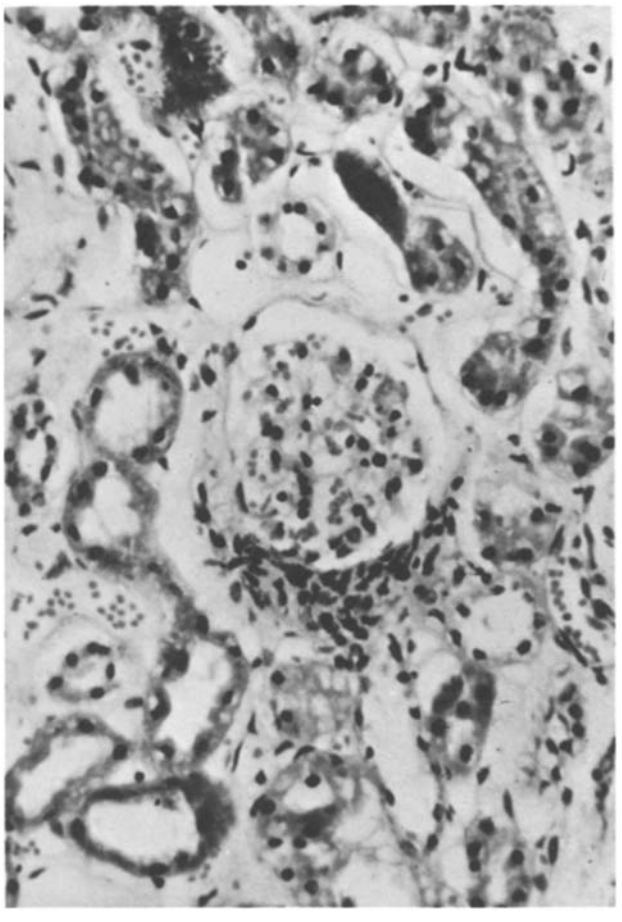

Fig. 4. Histological section of the displaced kidney showes hypertrophic J. G. A. 285 times magnification

kept alive without complications. Blood pressure determinations in this group did not show any changes of significance. Histological examinations of the arteries and kidneys showed no evidence of microscopic alteration. A renal artery thrombosis was found in one rabbit.

In group II consisting of eleven rabbits, two animals died one following induction of anaesthesia and the other two weeks after the experiment, because of necrosis of the displaced kidney and secondary infection. One rabbit (no. 5) was sacrificed after streching the artery while formalin (10%) was poured over the vessel to abtain the specimen for immediate histological examination. Histological sections did not show significant lesions with light-microscopy. Eight rabbits recovered from operation and were kept alive for eight weeks. At that time the blood pressure was significantly elevated in all animals (Table 1, Fig. 3). The average rise in blood pressure was 35 mm Hg. systolic and 21 mm diastolic. The histological sections of the renal arteries did not show lesions which could be interpreted as fibrous displasia or fibromuscular hyperplasia. However, the kidney sections disclosed a significant increase in number and size of the juxtaglomerular apparatus (JGA) of the displaced kidney compared to the untouched contralateral kidney (Fig. 4).

Discussion

The initial aim of this study was to determine whether streching of the renal arteries of rabbits would incite a fibroplastic response within the media or subadventitial layers. This was not achieved. However, a significant elevation of blood pressure in these animals was consistantly observed and therefore another model for producing renovascular hypertension experimentally has been found. The blood pressure elevation correlated with the histological finding of increase in size and number of the juxtaglomerular apparatus on the treated side. Further studies are in progress to determine whether hyperreninaemia will be found in the venous effluent from the displaced kidney compared to the untouched side. Several reports (4, 5) have suggested that renovascular hypertension may occur because of the abnormal mobility of the kidney and traction and torsion of the renal artery. In clinical studies we are convinced that in a certain number of women with nephroptosis and renal artery traction with or without stenosing lesions orthostatic hypertension or aggravation of their hypertension will occur in the upright position. It has been demonstrated clinically that in certain instances nephropexy resulted in an improvement of the hypertension (4, 5). We have previously postulated (2) that marginal stenoses of the renal artery could be made critical when traction was placed on the vessel. This condition occurs as an experi-

ment of nature in patients with severe nephroptosis and renal artery streching. In rabbits, we have found that traction on the renal artery produced sufficient narrowing of the vessel and subsequently renal ischemia and hypertension. We were not able to produce histological changes in the wall of the renal arteries as a result of traction. This model did not duplicate the condition seen in patients with nephroptosis since in the latter there is frequent streching and relaxation of the vessel which possibly produces structural changes in the arterial wall. Accordion-like action on the renal artery was not produced in this experiment, nor in those of Rothfield (6).

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