Occlusion of a Renal Artery as a Cause of Hypertension

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This paper presents three cases and the summary of a fourth case, in which occlusion of a renal artery was the cause of hypertension. Three patients were relieved of hypertension by nephrectomy. The literature on nonembolic renal artery occlusion with hypertension is reviewed, including 21 autopsy reports and 16 cases in which the patients were relieved of hypertension by nephrectomy or thromboendarterectomy. When a patient is found to have renal hypertension, the possibility of renal artery occlusion should be considered. Translumbar aortography currently is the best means of demonstrating renal artery occlusion.

SINCE Goldblatt¹ demonstrated in 1934 that compression of one or both renal arteries with a metal clamp will produce hypertension in dogs, the clinical implications of experimental hypertension have been extensively explored. It is now generally accepted that a causal relationship exists between various lesions of the kidneys and hypertension in human beings. Unfortunately, the term “Goldblatt phenomenon” has been loosely applied to many clinical and pathologic conditions which do not simulate the state experimentally elicited by application of an arterial clamp.

Several recent reports have described patients with obstructive lesions of one or both main renal arteries who exhibited the syndrome of malignant hypertension and in whom the arterial occlusion seems to have preceded the onset of hypertension. Such cases represent a close parallel to experimental renal arterial hypertension. Correlation of clinical data with pathologic findings has facilitated accurate diagnosis of this type of renal hypertension and has clarified the urgent indications for early definitive treatment.

This paper presents reports of three patients who had renal hypertension and clinical and pathologic evidences of occlusion of one main renal artery or a major branch. Each of these patients was relieved of hypertension following nephrectomy. A fourth case, previously reported by Fisher and Corcoran,² is presented in summary. The aim is to emphasize the significance of renal artery occlusion as a cause of hypertension and to describe the recognition and treatment of this condition, with special emphasis on the value of aortography in demonstrating the lesion.

Case Reports

Case 1. In December 1952, R. V. S., a 30 year old white man, suddenly developed right loin pain that persisted for five days. An intravenous urogram revealed a nonfunctioning right and a normal left kidney. No opaque calculi were seen along the urinary tract. Four days later a second intravenous urogram showed prompt excretory function of both kidneys, although there was poor concentration of contrast medium in the right kidney. The urinalysis was negative for sediment and albumin. Blood pressure was not recorded at that time.

Four months later, the blood pressure was slightly elevated with a systolic of 146. A month later the blood pressure was 240/140 and the patient complained of frequent morning headaches. There was no family history of hypertension.

A month later, the blood pressure was 180/130 and the patient had lost 12 pounds in weight. The fundi showed generalized moderate angiospasms. The blood urea nitrogen content was 10 mg. per 100 ml.; urea clearance was 95 per cent of average normal. Urine culture produced Escherichia coli and streptococcus. Another intravenous urogram was interpreted as showing good bilateral excretory function. Retrograde pyelograms demonstrated a normal left kidney but gave indication of an abnormality of the lower pole of the right kidney. Indigo-carmine excretion from the right kidney was diminished as compared to that of the left.

The patient was referred to us in June 1953, by Dr. A. K. Hamp of Grand Rapids, Mich., who made the tentative diagnosis of renal hypertension and furnished relevant clinical data. On examination here, the blood pressure was 165/120 and the fundi revealed grade I constriction of vessels. The urea

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¹ From the Department of Urology, The Cleveland Clinic Foundation, and The Frank E. Bunts Educational Institute, Cleveland, O.
clearance was 100 per cent of normal in the first and 82 per cent in the second hour. An Addis test showed a volume of 610 cc.; maximum specific gravity of 1.015; and protein content of 0.7 Gm. per 24 hours. The sediment contained 1,200,000 red blood cells, 30,000,000 white and epithelial cells, and 102,000 casts in 12 hours.

The pyelographic abnormality was interpreted as a decrease in size and crowding together of the calyces in the lower pole of the right kidney (fig. 1A). A lesion of the renal artery was suspected; a translumbar aortogram (fig. 1B) demonstrated a narrow right renal artery. Arterial supply of the upper two thirds of the right kidney appeared normal but there was no filling of the arteries of the lower pole. The left renal artery and its branches appeared normal. These findings were attributed to occlusion of the artery to the lower pole of the right kidney with ischemia* of this area; nephrectomy was advised. The patient returned home for nephrectomy which was performed by Dr. J. A. Ryan on June 17, 1953, at the Blodgett Memorial Hospital, Grand Rapids, Mich.

Five months postoperatively, the blood pressure was 114/76 and the patient was entirely well. Eighteen months later, the blood pressure still was reported to be normal.

Pathology. The kidney was reported to show a clearly demarcated pale and ischemic lower pole. The major branch of the renal artery to the lower pole was partly occluded; the arteries to the remainder of the kidney appeared normal.

* The term "ischemia" is used in this paper to indicate a decrease in arterial circulation as shown by aortography.

Slides were sent by Dr. C. A. Payne of the Blodgett Memorial Hospital, Grand Rapids, Michigan, and were reviewed by Dr. L. J. McCormack of our Department of Pathology. The artery to the upper part of the kidney appeared normal with a thin intima and normal elastic lamina and muscular coat. The lumen of the artery to the lower pole (fig. 2A) was approximately 70 per cent occluded by a mass of loosely arranged fibrous tissue covered by endothelium. The internal elastic lamina was intact and the muscular layer and outer elastic lamina were normal. On longitudinal section, the mass of collagenous tissue was partly recanalized by endothelial-lined vascular channels. Sections of the kidney demonstrated two distinct histologic features: (1) The upper part of the kidney showed normal renal parenchyma (fig. 2B); blood vessels entering the glomeruli appeared slightly thickened. (2) A junction line was present, which divided the normal parenchyma from an area in which the tubules were collapsed and atrophic, with slight increase of interstitial tissue, and in which the glomeruli appeared shrunken and showed a slight increase in tuft thickness.

The pathologic diagnosis was old, partial occlusion of lower secondary branch of the main right renal artery due to fibrous tissue, with renal atrophy distal to the occlusion.

Comment: This case is an example of renal hypertension caused by a partially ischemic kidney. It is believed that the initial vascular injury occurred at the time of the abdominal pain. Translumbar aortography five months later clearly demonstrated obstruction of the

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**Fig. 1.** Case 1. (A) Retrograde pyelogram of right kidney reveals small, compact lower calyces. (B) Translumbar aortogram demonstrates absence of branch of main renal artery to lower pole of right kidney. Zone of atrophy is sharply demarcated. Left renal artery and kidney are normal.
lower main branch of the right renal artery and nephrectomy was advised. The fact that the patient has remained normotensive for 18 months after nephrectomy is reasonable evidence that the arterial occlusion with resultant partial renal ischemia was the cause of the hypertension.

Case 2. W. J., a 52 year old white man, developed acute appendicitis in May 1954; a suppurative appendix was removed at operation. Recovery was complicated by ileus and evisceration. During his hospitalization the blood pressure was found to be elevated and albumin was detected in the urine. One month after discharge from the hospital he developed headaches, blurring of vision, nausea, vomiting, and nocturia. He had lost 35 pounds in weight. He was referred here on July 13, 1954, by Dr. Meyer Bloom of Johnstown, Pa., who had made a tentative diagnosis of renal hypertension.

The patient had been examined here in 1950 for episodes of syncope diagnosed as postural hypotension. The blood pressure at that time was 105/70 in the supine position. In late 1953, a hemorrhoidectomy was performed and the blood pressure was reported to be slightly elevated. There was no family history of hypertension.

The blood pressure was 178/118. The fundi showed grade II constriction and sclerosis of retinal arteriolo, numerous hemorrhages and exudates and about 3 diopeters of papilledema. The heart was enlarged to the left but the sounds were normal. Peripheral arterial pulsations were normal. Slight ankle edema was present; the liver edge was 3.5 cm. below the right costal margin.

The blood urea was 27, creatinine 1.0, and cholesterol 343 mg. per 100 ml. Urea clearance was 44 per cent of normal during the first and 37 per cent during the second hour. Urinalysis revealed 1 plus albuminuria, 2 to 5 red blood cells and a rare white blood cell per high-power field. Three urine cultures were sterile. Over a 10-day period, daily urinary protein excretion varied between 6.5 and 21.5 Gm. per 24 hours.

The electrocardiogram showed myocardial changes. By the Ungerleider-Gubner scale, the heart was calculated to be 6 per cent over normal size for the patient’s height and weight. Tiselius electrophoretic analysis of the plasma proteins revealed a total protein content of 4.38 Gm. per 100 ml., with 2.19 Gm. of albumin, 0.73 Gm. of α-globulin, 1 Gm. of β-globulin and 0.46 Gm. of γ-globulin.

Examinations of the urinary sediment on several occasions revealed 62,000 to 390,000 casts, 2,320,000 to more than 18,000,000 red blood cells, and 880,000 to more than 16,000,000 white blood cells per 12 hours. Cold pressor and Regitine tests were negative. The averages of 44 blood pressure recordings over a one-week period were 172/117 in the supine position and 166/117 mm. Hg in the standing position.

The intravenous urogram revealed a normal right kidney with good excretory function, but only a faint trace of dye was seen in the tip of the lower calyx of the left kidney (fig. 3A). A left retrograde pyelogram demonstrated normal renal pelvis and calyces with slight reduction in size of the kidney.

Fig. 2. Case 1. (A) Cross section of artery to lower pole, showing partial obstruction of lumen (X20). (B) Photomicrograph of lower pole of kidney showing demarcation between the normal renal parenchyma on left and the zone of atrophy on the right (X110).
(fig. 3B). Intravenously injected indigo-carmine appeared after 13 minutes in poor concentration from the left ureteral catheter. Translumbar aortogram showed only a small aberrant renal artery to the lower pole of the left kidney (fig. 3C); the left main renal artery was not visualized. The arterial distribution to the right kidney was normal. The left renal artery was considered to be completely occluded. This was believed to be the cause of his malignant hypertension. Left nephrectomy was performed on July 30, 1954. The main left renal artery was pulseless and occluded by a hard plaque midway between the aorta and the kidney.

On the day following operation, the blood pressure was 129/86, supine. Rapid, progressive improvement of the retinopathy occurred with absorption of hemorrhages, exudates, and papilledema.

Three months later he was feeling well; blood pressure was 130/88. The proteinuria varied between 5 and 9 Gm. per 24 hours, on three occasions. A mannitol-para-aminohippurate renal clearance test indicated a renal blood flow of 537, renal plasma flow of 317, and glomerular filtration rate of 36.7 ml. per minute with filtration fraction of 0.12; the respective preoperative values were 293, 176, 46, and 0.27.

Seven months later he was asymptomatic and the blood pressure was 130/85. The fundi had be-
come completely normal; proteinuria had diminished to about 1.5 Gm. per 24 hours.

Pathology. The left kidney (fig. 4) weighed 120 Gm.; its capsule stripped easily, revealing a smooth surface. The cut surface was uniformly blue-pink in color, except the lower pole where the cortex was pale. A hard clot, 1 cm. in length and 0.5 cm. in diameter, was visible and palpable in the main renal artery, completely occluding its lumen. A small patent aberrant renal artery led to the lower pole of the kidney. The renal veins and ureter were normal.

The microscopic pathologic description was prepared by Dr. J. B. Hazard of our Department of Pathology. Sections of the renal artery showed the lumen to be filled by blood clot, sufficiently old in the center to show complete loss of red-cell structure. No canalization was evident. The vascular wall was thin and distended by the clot. The internal elastic membrane and muscular layer appeared normal, except for moderate infiltration of lymphocytes and plasma cells, more evident in the adventitia.

Sections of the cortex from the upper pole showed areas of increased interstitial tissue with reduction in tubular tissue. Some of the tubules were dilated and contained granular coagula and hyaline casts. In the areas with greatest increase in connective tissue, the tubules that were present had a small caliber; and in such zones proximal, convoluted tubular epithelium could not be identified. Bands of well-preserved tubules were scattered throughout the kidney; no colloid-filled tubules were seen. The glomeruli were of normal size and their capillaries contained blood in the majority of tufts. A rare sclerosed glomerulus was present. The arcuate and larger vessels showed a moderate degree of intimal thickening.

The pathologic diagnosis was occlusion of main renal artery by partly organized old blood clot (thrombus), with focal atrophy of the cortical tubules and moderate arteriosclerosis of intrarenal arteries.

Comment: In this case it is likely that thrombosis occurred during the patient's convalescence from appendectomy. Within two months he showed evidence of rapidly progressive hypertensive vascular disease with grade IV eye-ground changes, depression of renal function, cardiac enlargement and a mean resting blood pressure of 176/117. Intravenous urography revealed that only the lower part of the left kidney retained any function. Translumbar aortography demonstrated that the main left renal artery was blocked, but that there was a small patent aberrant artery to the lower pole of the left kidney. Since nephrectomy, he has regained his health and has remained normotensive; eye grounds have become normal and renal function has steadily improved, indicating regression of the hypertensive vascular disease. The left renal artery was completely occluded by a thrombus. The kidney showed ischemic tubular atrophy; it is of interest that the intrarenal arteries showed only moderate intimal thickening.

Case 3. F. R., a 51 year old white policeman, suddenly developed an attack of dizziness and epigastric pain while walking his beat. The attack was followed by a throbbing headache and blurring of vision. The blood pressure was found to be 250/150, and he was given hypotensive drugs.

Past history revealed that a slight elevation of blood pressure had existed for about six years; the actual values were not known. There was no family history of hypertension.

In March 1953, two months later, he was admitted to the hospital with a blood pressure of 200/130. The fundi revealed grade III eye-ground changes with constriction and sclerosis of retinal arteries, hemorrhages and exudates. Except for enlargement of the heart, the remainder of the physical examination was negative.

Blood urea was 42 mg. per 100 ml.; urea clearance was 70 per cent of normal in the first hour and 64 per cent in the second hour. The urine showed a trace of albumin and occasional white blood cells.

![Fig. 4. Case 2. The ischemic kidney. Note the demarcation (d) between the ischemic portion and the area supplied by the aberrant renal artery (c). The area of thrombosis (a) is visible in the renal artery. The ureter (b) is normal.](http://circ.ahajournals.org/}

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Fig. 5. (Case 3) Visible between the aortogram needles are two small arteries extending to a small, poorly vascularized left kidney. Right renal artery is normal.

The urine culture produced *Escherichia coli* on one occasion; a second culture was sterile. The Addis test showed a specific gravity of 1.017 with 0.36 Gm. of protein excreted in 24 hours; the sediment contained 84,000 casts, 126,000 red and no white blood cells in 12 hours.

Chest roentgenograms revealed left ventricular enlargement. A plain roentgenogram of the abdomen was normal except for a small left renal outline. Intravenous urography demonstrated prompt excretion from the right kidney but there was no evidence of function of the left kidney. A retrograde pyelogram made two months earlier at another hospital had shown a contracted left kidney. Translumbar aortography revealed a large right renal artery and kidney. Supplying the contracted left kidney were two very small arteries (fig. 5).

Blood pressure determinations over a three-week period averaged 193/120 supine and 179/125 upright.

A left nephrectomy with a left thoracolumbar sympathectomy was advised on the premise that, if the disease failed to respond to nephrectomy, only one stage of the recommended sympathectomy would still have to be done. On April 24, 1953, a left nephrectomy was followed by ablation of the left sympathetic chain from the tenth thoracic to the second lumbar ganglia; the splanchic nerves were removed from high in the chest and from the celiac ganglion.

Three weeks later the blood pressure was 160/107 and the patient was discharged from the hospital. Urea clearance at that time was 90 per cent of normal in the first hour and 65 per cent in the second hour.

One year later, he was readmitted for evaluation, although he was asymptomatic. The blood pressure was 144/90, the blood urea was 31 mg. per 100 ml., urea clearance was 105 and 85 per cent of normal, and the fundi showed grade I constriction and sclerosis but no hemorrhages or exudates. The amount of protein in the urine was within normal range. Eighteen months after operation, the blood pressure was 130/85.

Pathology. The left kidney was small and weighed 120 Gm. The capsule stripped with some difficulty, revealing a greyish-tan granular surface of uniform texture. The cortex measured 0.5 cm. in thickness. The calyces and ureter appeared normal.

The microscopic study was done by Dr. J. B. Hazard. Marked tubular atrophy was present, particularly in the outer half to two thirds of the cortex. A reduction in number of the tubules was evident, and the remaining tubules appeared small and separated by a relatively increased hyaline and fibrous stroma. In the inner portion of the cortex, the tubules appeared intact. The glomeruli were small but appeared well preserved and their capillaries contained some blood but were not distended.

An occasional sclerosed glomerulus and some clusters of hyaline glomeruli were seen. The capsular spaces were prominent and filled with granular coagula. No colloid-filled tubules were found. Major intrarenal arteries were not remarkable except for slight hypertrophy of their walls. Arterioles were patent and showed only a moderate amount of fibrous thickening of their intima.

The pathologic diagnosis was renal tubular atrophy and interstitial inflammation compatible with chronic ischemia associated with renal artery obstruction. No intrarenal vascular lesions were evident in the specimen.

Comment: This patient was found to have severe hypertension following an attack of epigastric pain. There were grade III eye-ground changes, depression of renal function, and cardiac enlargement. Intravenous urography showed no evidence of left renal function. Translumbar aortography revealed a striking contrast in the blood supply and size of the two kidneys: the right renal artery and kidney were normal, but there was no main left renal artery; instead, there were two little arteries coming directly from the aorta to the upper and lower parts of a small left kidney. While it was considered that this abnormal kidney could be the cause of the hypertension, left nephrectomy was combined with left dorso-lumbar sympathectomy to save the patient an additional operation if nephrectomy did not
lower the blood pressure. (These circumstances are similar to those in two cases reported by Thompson and Smithwick.⁵) The left kidney was contracted and showed changes characteristic of ischemia consisting of tubular atrophy and increased interstitial fibrosis without much change of the glomeruli, intrarenal arteries or arterioles. Following nephrectomy, his blood pressure gradually reached normotensive levels. He has remained asymptomatic and normotensive for almost two years. Since it is well known that unilateral dorsolumbar sympathectomy will not alter the course of hypertensive vascular disease, it is reasonable to assume that the ischemic kidney was the cause of this man's hypertension.

**Case 4.** This case has been reported by Fisher and Corcoran² and will be presented in summary only.

A 14 year old white boy was seen in June 1951 because of hypertension and attacks of abdominal pain. There was no family history of hypertension. The blood pressure was 238/160 in the left arm and 256/100 in the left leg. Tests of renal function gave normal results and the urinary sediment was normal. Neurogenic, endocrine, cardiovascular and intrarenal causes of hypertension were believed to be excluded. He gave a history of intermittent pain in the flanks and abdomen.

The intravenous urogram showed prompt, satisfactory excretion from both kidneys, although the right kidney was somewhat reduced in size. A translumbar aortogram was originally interpreted as showing normal renal arteries and vascularity but, reviewed in the light of later experience, showed poor filling of both renal arteries and their branches. At operation, the right kidney was smaller than the left, and no pulse was felt in the right renal artery. The right kidney was removed and since blood did not flow from the aortic stump of the renal artery, a probe was passed; it met resistance, apparently at the orifice of the renal artery. The boy died four days later. Autopsy revealed congenital coarctation of the abdominal aorta of the segment involving the origin of superior mesenteric artery and renal arteries. There was severe stenosis of the orifices of the renal arteries, superior mesenteric artery, and the celiac axis, due to arteriosclerosis of the fibrous intimal type. The kidneys, which had been protected from the impact of elevated blood pressure by the arterial stenosis, microscopically showed normal tubules, glomeruli and arterioles.

**Comment:** The fourth case report is summarized as an example of fatal renal hypertension resulting from stenosis of the aortic orifices of both main renal arteries. This was associated with coarctation of the abdominal aorta; the stenotic lesions were attributed to abnormal “jet” effects. Evidently the stenosis of the renal arteries allowed some blood to flow through, but in an impeded manner and with resultant renal ischemia, particularly of the right kidney as shown at operation. In spite of this, the intravenous urogram demonstrated prompt and satisfactory function of both kidneys. Pathologic examination showed no evidence of vascular disease or atrophy within the kidneys. Had the lesion been recognized, one autogenous renal graft might have been lifesaving.

**Discussion**

**Retrospect.** That hypertension can be associated with unilateral renal disease was first pointed out by Ask-Upmark⁴ in 1929. Further attention was called to this relationship in 1937 by Butler⁵ who was the first to report the beneficial effect of nephrectomy upon hypertension in unilateral pyelonephritis. Meanwhile, experimental production of renal hypertension in dogs¹ and other animals,⁶,⁷ by constriction of renal arteries, stimulated great interest in hypertensive patients with seemingly unilateral renal disease. Nephrectomy frequently was done in the hopes of removing the cause of the hypertension. Results often were disappointing. Actually, removal of abnormal kidneys in hypertensive patients favorably altered the course of the disease in only one out of four or five for a year or longer.⁸,⁹ This experience indicated that unilateral renal disease was not commonly a primary cause of hypertension, at least, at the time of nephrectomy. The best results of nephrectomy were obtained in patients who had rapidly progressive hypertension of recent onset and in whom there was satisfactory demonstration of disease in only one kidney. Common among the urologic diseases for which nephrectomy was done in the hope of reducing blood pressure were pyelonephritis, pyonephrosis, calculous disease, tuberculosis, aplasia and hydronephrosis.

Gradually, over the last two decades, an increasing number of reports have appeared describing unilateral renal artery occlusion as a
cause of persistent hypertension. Some of these cases have been successfully treated by surgery. This situation, in contrast with many other renal diseases, closely resembles experimental renal hypertension elicited by partial compression of a renal artery.

Etiology: Yuile\(^9\) reviewed occlusive lesions of the main renal arteries in hypertensive patients and classified their etiologies as either extrinsic or intrinsic. Extrinsic causes of renal artery occlusion are aortic or renal artery aneurysms, hematomas, compression by adjacent cyst or tumor and twisting of the renal pedicle. Intrinsic causes of narrowing or occlusion of the renal artery are arteriosclerotic plaques, syphilitic arteritis, thrombosis, embolism and congenital or arteriosclerotic stenosis of the aortic orifice of the renal artery.

Arteriosclerotic narrowing of renal arteries is a common finding at autopsy and usually is associated with widespread arteriosclerosis. Several investigators\(^11, 12\) attempted to show a correlation between arteriosclerotic constrictions of the renal artery and hypertension, but others\(^13, 14\) were unable to demonstrate any relationship between the caliber of the renal artery and the level of blood pressure. Yuile\(^9\) pointed out that arteriosclerotic lesions of main renal arteries usually were associated with intrarenal arteriosclerosis and that pre-existing hypertension probably was a cause of both these lesions, rather than a result of the one.

Several cases have been reported of rapidly fatal, malignant hypertension caused by embolic occlusion of the renal artery in which the autopsy findings were carefully correlated with the clinical data.\(^15, 16, 17\) Transient hypertension has been reported to result from renal vascular injury due to embolus.\(^15, 18, 19\) The longest reported duration of hypertension following renal infarction of this type is 21 days. The size of the embolus and the extent of the renal infarction determine the course following such a vascular injury.

It should be pointed out that renal infarction can occur without causing hypertension. Regan and Crabtree\(^20\) reviewed the literature and summarized the important clinical and pathologic data of 94 patients who had arterial, venous, or traumatic infarction of the kidney. Hypertensive vascular disease was not reported to be present in this group of patients. Most were considered to have complete aseptic infarction of the kidney, and many were subjected to surgery shortly after the onset of symptoms. In others, the diagnosis was presumptive, the patients recovering without surgery.

Infarction of the kidney usually results in complete loss of excretory function and destruction of the kidney. Partial infarction, however, produces areas of atrophy in the renal cortex surrounded by normal tissue. Decrease in renal size results, but excretory function may seem to remain unchanged unless accurately measured and compared with the opposite kidney. Cases of patients with hypertension resulting from such a renal injury have been reported.\(^21, 22\)

Of particular clinical interest are the unilateral thrombotic renal artery lesions which cause sudden onset of renal hypertension. A number of case reports have been published which present the clinical history and pathologic findings at autopsy. Reports have appeared of 16 patients successfully treated by nephrectomy or thromboendarterectomy, with restoration of normal blood pressure. These are summarized in table 1.

Renal Pathology: A review of the vascular and renal lesions, found in three patients with unilateral renal artery occlusion and with the syndrome of malignant hypertension, was presented by Laforet.\(^23\) In two instances the renal artery was occluded by a thrombus; the renal artery of the third patient was obstructed by a metallic foreign body. Microscopic examination of the ischemic kidneys supplied by the occluded arteries showed tubular atrophy, increase in interstitial tissue, and a benign type of nephrosclerosis. The nonischemic kidneys, which were supplied by patent renal arteries, all showed necrotizing arteriolar lesions, characteristic of malignant nephrosclerosis. This accords with the view that renal artery obstruction protects arterioles of the ischemic kidney; whereas, under the impact of hypertension, the kidney with the normal renal artery develops malignant nephrosclerosis.

In animals, the severity of experimental renal hypertension depends in part upon the proportion between ischemic and nonischemic renal tissue. That this may hold true in human
beings is illustrated by the course of the first patient, who had a small area of ischemic atrophy in one kidney. Five months after the presumed arterial occlusion, his blood pressure was 240/130; a month later the fundi revealed moderate angiospasms but renal function was unimpaired. In contrast, the second and third patients developed hypertension within three months of the time when renal artery occlusion probably occurred. Both manifested severe hypertensive vascular disease as shown by eye-ground changes and depression of renal function. The extent of renal ischemia and atrophy in the second patient was subtotal, while the third patient had involvement of the entire kidney. The fourth patient did not show ischemia pathologically but had incomplete occlusion of both renal arteries and good evidence of intermittent grossly deficient renal blood supply.

Diagnosis

It is important to bear in mind the possibility of renal hypertension in a patient who has sudden onset of nonfamilial hypertension. Of particular interest are those patients who have abdominal pain or disease and soon after develop progressive hypertensive vascular disease. The abdominal symptoms may be brief or prolonged and investigation often fails to reveal the source of disease. The symptoms may not resemble the usual type of pain associated with renal disease. Intravenous urography usually will show absence or severe diminution of renal function in the ischemic kidney with complete renal artery occlusion. If only part of the arterial system is occluded, the kidney may show slight diminution of function or even appear normal.

Retrograde catheterization of ureters should be done and specimens collected from each kidney. The ischemic kidney will show reduction in function consisting of diminished urinary flow, delayed indigo-carmine excretion and decreases of mannitol and para-aminohippurate outputs and urea and creatinine concentrations. Retrograde pyelograms may show a normal pelviocalyceal system or there

Table 1.—Nonembolic Renal Artery Occlusion with Renal Hypertension

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<th>Autopsy Reports</th>
<th>Relief of Hypertension by Nephrectomy or Thromboendarterectomy</th>
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<td>Author</td>
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* Later evidence showed that this was not an occluded renal artery.
† Two additional cases are included in this report: one case was first reported by Boyd and Lewis; the other was found to have a ganglioneuroma pressing on the renal artery with no intraluminal arterial disease.
‡ Thromboendarterectomy.
may be some reduction of the size of the system with decrease of renal mass.

Translumbar aortography is the one definitive method by which occlusive renal artery disease can be demonstrated. It should be utilized in patients suspected of having this type of lesion as the cause of hypertension. It may demonstrate absence of filling of a main renal artery, filling defects in the renal artery, absence of important branches of the main renal artery and reduction in the vascular system of the affected kidney. After complete main renal artery occlusion, blood may be seen to enter the kidney only through aberrant arteries.

With proper safeguards translumbar aortography is safe and is relatively simple. The technic has been previously described and the procedure usually is done under local anesthesia. The technic consists of the insertion of a long needle into the aorta at the level of the first lumbar vertebra in the vicinity of the renal arteries. A preliminary injection of 10 ml. of 30 per cent contrast medium is made to be sure that the needle is inserted correctly. After inspection of this film, 10 ml. of 70 per cent medium is injected for the final film. The iliac arteries are occluded by a pressure pad to prevent down-flow during the injection. Excellent films are obtainable when the procedure is done under local anesthesia since the patient can cooperate by holding his breath during the injection and film exposure. A brief sensation of warmth is felt in the lower abdomen and legs. Approximately 250 translumbar aortograms have been done here by this method without serious complications. We have used Urokon, but we are now utilizing 50 per cent Hypaque as a contrast medium.

TREATMENT

The treatment of hypertension associated with ischemic atrophy of the kidney, resulting from unilateral renal artery occlusion, is nephrectomy (table 1). Function of the kidney with intact blood supply must, of course, be reasonably good. Relief of hypertension has consistently followed nephrectomy for this disease, including the first three cases reported here. Once the condition is recognized, nephrectomy should not be delayed. Considerable evidence indicates that the type of hypertensive vascular disease associated with unilateral renal artery occlusion is rapidly progressive, as in the second and third patients (cases 2 and 3). If untreated, malignant nephrosclerosis will occur in the nonischemic kidney and death will result from renal insufficiency.

There has been one report of a patient with partial occlusion of the left renal artery and thrombosis of the aorta, who was relieved of hypertension by thromboendarterectomy of the aorta and renal artery. Preoperatively, the affected kidney was said to have had normal excretory function; unfortunately it was not biopsied, but it is unlikely that atrophy had occurred. In a personal communication, DeBakey reports a similar instance of a patient relieved of hypertension by thromboendarterectomy. Perhaps more patients will be found who have hypertension resulting from partial occlusion of a renal artery, with no loss of function or atrophy, who can be successfully treated by thromboendarterectomy.

SUMMARY

Reports of three cases of unilateral renal artery occlusion associated with renal hypertension are presented. In all three patients, the hypertensive vascular disease was relieved following nephrectomy. The affected kidneys revealed ischemic atrophy, particularly of the tubules; the extent of atrophy depended on the amount of normal blood supply that remained intact. These three cases closely simulate experimental hypertension induced by a renal arterial clamp. A summary is also presented of the case of a patient who had stenosis of the aortic orifice of both renal arteries associated with renal hypertension.

Important points in the diagnosis of this lesion are: abdominal or flank pain followed by sudden onset of hypertension in a patient with

* Urokon sodium, Mallinckrodt Chemical Works.
† Hypaque sodium, Winthrop-Stearns, Inc.

Since preparation of this article, two additional patients, having occlusion of a renal artery associated with malignant hypertension, have been treated by nephrectomy. The findings in these cases will be reported in a subsequent publication.
no family history of hypertension; demonstration of unilaterally diminished renal function by intravenous urography or by comparison of samples of urine from each kidney; and most importantly, aortographic visualization of partial or complete occlusion of a main renal artery or one of its major branches.

Hypertension, associated with primary occlusion of a renal artery and renal atrophy, is treated by nephrectomy; thrombendarterectomy may be the treatment of choice in cases in which recent thrombosis of a renal artery has resulted from propagation of a primary aortic thrombosis.

SUMMARIO IN INTERLINGUA

Es presentate tres casos de occlusion unilaterale del arteria renal associate con hypertension renal. In omne le tres casos le morbo vascular hypertensive eseva alleviato post le nephrectomia. Le renes afficite revelava atrophia ischemic, specialmente del tubulos. Le grado del atrophia dependeva del portion intacte del apport normal de sanguine. Iste tres casos simulava molto directemente le typo de hypertension que es inducite experimentalmente per le application de un clamp al arteria renal. Es etiam presentate summarimente le caso de un patiente qui habeva stenosis del orificio aortic de ambe arterias renal associate con hypertension renal.

Importante aspectos in le diagnoste de iste lesion es: (1) Dolor del abdomen o del flanco sequite per le subitanea declaration de hypertension in un patiente sin historia familiar de hypertension. (2) Demonstration de diminution unilateral del function renal, effectuate per urographia intravenose o per un comparation de specimen de urina ab cata un del renes. E (3)—lo que es le plus importante—visualisation aortographic de occlusion partial o completo de un arteria renal principal o de un de su principal brancas.

Hypertension associate con occlusion primari de un arteria renal e atrophia renal es tractate per nephrectomia. Thrombendarterectomy pot e esser le tractamento seligite in casos in que thrombosis recente de un arteria renal ha resultate ab le propagation de un primari thrombosis aortic.

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Circulation. 1956;13:37-48
doi: 10.1161/01.CIR.13.1.37
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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