A. Anatomy

The renal vessels may show variations in their number and branches. If there is only one major renal artery on each side, both originate from the aorta at the level of the intervertebral disk between the first and second lumbar vertebra. The origin is often asymmetrical: the right renal artery usually takes off somewhat deeper. Close to the hilus, the renal artery gives off several branches. The majority of these enter the parenchyma in front of the renal pelvis. The suprarenal artery and the testicular or ovarian artery originate along the course of the renal artery from the aorta to the hilus of the kidney (see p. 19).

In about 25% of humans, the kidney is supplied by two or more arteries, directly originating from the aorta. These variant vessels can cause compression effects in the region of the renal pelvis and the ureters, with consequent impairment of urinary flow as well as the risk of accidental ligation. Renal hypertension may develop, depending on which region of parenchyma is no longer supplied.

Additional arteries may enter the kidney at the hilus itself, but also somewhat outward. They may be expected above as well as below the hilus. The origin of the right renal artery is covered by the venous crossing, which is formed where the two renal veins join the inferior vena cava; extensive dissection of this crossing is necessary for adequate exposure of the renal artery.

The Collateral System of the Kidney. The renal arteries are usually considered to be terminal arteries since in cases of acute occlusion, the existing collaterals are not sufficient for maintaining an adequate blood flow. However, when the renal arteries gradually narrow, owing to an occlusive disease, preexisting collaterals may develop so that at least basic metabolism is maintained. Collateral circulation may be developed by arteries to the adrenals via the junction to the superior suprarenal arteries, by the arteries of the capsule via the

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lumbar arteries, and by the ureter vessels. The ureter vessels are supplied by the renal artery in the upper region, by the ovarian or spermatic artery in the middle region, and by the common iliac artery in the lower region [12]. When serving as collaterals, the ureteral vessels show this very clearly on the intravenous pyelogram; these images serve as a sure diagnostic sign of a functionally significant stenosis of the renal artery.

B. Renovascular Hypertension, Indications for Operation

Renovascular hypertension is the most common reason for operations on the renal arteries. Therefore, procedures for the treatment of this condition will be discussed before those that are used in acute occlusion, in trauma, or following renal transplantation.

I. Definition, Incidence

Renovascular hypertension is a syndrome in which normalization of the blood pressure may be achieved by reconstruction of the appropriate renal artery lesions outside of the parenchyma [17]. Lesions of intraparenchymatous arteries and arterioles, on the other hand, are not included. However, the term “renovascular hypertension” is not used uniformly in the literature. Among all types of hypertension, renovascular hypertension may account for less than 1% [6].

II. Etiology

The most common cause of renal artery stenosis is arteriosclerosis [9] close to the aorta, in the first third of the renal artery. The stenosis is more or less concentric with a poststenotic dilatation. It is impossible to say whether the renal artery arteriosclerosis is caused by essential hypertension or vice versa.

The second most frequent disease is fibrodysplasia, occurring in four different forms [13]:

1. The rare, so-called fibromuscular dysplasia with a circumferential stenosis caused by thickening of the artery muscles
2. Intimal fibroplasia, causes also constricting lesions with poststenotic dilatation
3. Medial fibrodysplasia, characterized by a series of stenoses alternating with aneurysmal dilatations, resembling haustration
4. Subadventitial (perimedial) dysplasia

The lesions, typically located in the periphery, are often bilateral and combined with a so-called mobile kidney.

These lesions may also appear in other vascular regions, but are then not as widespread as in arteriosclerosis.

It is controversial to what extent the classification of the various forms, as already diagnosed by the angiogram may have therapeutic consequences. The forms of revascularization that are necessary may differ greatly from those required for arteriosclerosis [22].

III. Pathophysiology

A common feature in all lesions is the impairment of renal blood flow, resulting in characteristic changes in the affected kidney, as compared with the contralateral one, which is exposed to a higher systemic blood pressure [3]. Reduction of blood flow causes an increase in the concentration of the initial urine and a decreased sodium concentration of the final urine as a result of reduced filtration. Previously, the indication for operation was based on tests of the functional differences on both sides [14], but because these tests were too complicated and caused too much stress on the patient, they have been abandoned.

Moreover, decreased blood flow, resulting in an increase of blood pressure via the renin–angiotensin system, is probably perpetuated via the aldosterone system by changes in the sodium metabolism. Administration of the converting enzyme inhibitor captopril may reveal the significance of the renin–angiotensin system by decreasing the blood pressure [20]. Care must be taken in the case of a single kidney because of the possibility of acute renal failure. Only in exceptional cases should the decision to operate be based on the results of this test.

IV. Diagnosis

History and clinical examination will not reveal peculiarities in patients with renovascular hypertension. The only basis for a clear suspicion is