**Arteriovenous Fistula of the Kidney**

**New Observations and Report of Three Cases**

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Arteriovenous fistulas of the kidney or renal vessels are probably not so rare as the small number of reported cases suggest. The fact that all but 2 of the total of 12 cases appearing in the literature are recently reported (within the past 11 years) lends support to this contention.

To this total of 12, we should like to add the following 3 cases that were observed at the Mayo Clinic between the years 1954 and 1957.

**REPORT OF CASES**

*Case 1.* This patient was a man 42 years of age when last seen at the clinic in 1955. The patient was first seen here in 1950, at which time he complained of fatigue, sweating, palpitation, and a feeling of stimulation and nervousness. He was clinically euthyroid, and his blood pressure was 150 mm. of mercury systolic and 90 mm. diastolic. His history indicated that he had been shot at close range in a hunting accident at age 14 years. The 22-caliber bullet entered his trunk anteriorly in the region of the liver. Gross hematuria occurred for 2 weeks, during which time the patient was in bed with drains in the hepatic region. A roentgenogram of the lumbar vertebrae showed the bullet lodged between the bodies of the first and second lumbar vertebrae with post-traumatic ankylosis bridging the area.

Since the roentgenogram of the chest in 1950 showed cardiac hypertrophy and the history of trauma was known, evidence of an arteriovenous fistula was sought in an attempt to explain the enlargement of the heart. A loud continuous bruit, loudest in the midaxillary line, was found over the upper part of the abdomen on the right.

The urine was normal, but an excretory urogram revealed marked distortion of the renal calyces. Results of the sulfobromophthalein (bromusulphalein) test of liver function were negative.

In an attempt to obtain evidence supporting the diagnosis of renal arteriovenous fistula, limited cardiac catheterization was carried out. Unfortunately, the catheter could not be made to enter the right renal vein, and evidence of arterialization of the blood was not encountered in the inferior vena cava, so proof of an arteriovenous fistula of the kidney could not be obtained (table 1). Nevertheless, the patient was considered to have an arteriovenous fistula of the right kidney and surgical exploration was advised with an alternative of continued periodic observation.

The patient returned 4 years later (1954) with the same complaints, but increased in severity. The enlargement of the heart had increased. The electrocardiogram showed changes consistent with early left ventricular hypertrophy. At catheterization, indicator-dilution studies showed recirculation time of 12 seconds as compared to a normal value of approximately 21 seconds. The cardiac output was increased as compared to the 1950 studies (table 1). An aortogram showed a greatly enlarged and tortuous renal artery terminating in what appeared to be an aneurysmal sac (fig. 1). Right nephrectomy was carried out.

Pathologic examination revealed an arteriovenous fistula with the renal artery and vein entering a common aneurysmal sac located at the hilus of the kidney. The narrowest portion of the arteriovenous fistula was 4 mm. in diameter (fig. 2).

Seven months later the patient returned stating that he felt normal for the first time in many years. At that time, cardiac catheterization yielded normal findings except for some slowing in the systemic recirculation time (table 1). Blood pressure was now normal, 128/86, and there was a rather striking reduction in size of the heart (fig. 3). The abnormal electrocardiographic pattern had disappeared.

*Case 2.* The patient, a 30-year-old woman, was admitted to the hospital in 1957 because an arteriovenous fistula of the left kidney was suspected. At the age of 4 years, she had been run over by the wheel of a wagon but apparently had sustained no significant injury. A heart murmur had been heard at an examination when she was in her early teens. She was married at 19 years of age and in the next 10 years had an unremarkable health record, which included 4 uneventful pregnancies.

From the Mayo Clinic and the Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

*A report on this case appeared in an exhibit at the meeting of the American Medical Association in June 1955.*
The present illness appeared to coincide with the beginning of her fifth pregnancy in September 1956. About this time she began to be weak and fatigued easily, and she had episodes of left upper abdominal pain. In January 1957, tachycardia, edema of her ankles, and an apical systolic cardiac murmur developed. The next month she was hospitalized for treatment of congestive heart failure. Continuous treatment for this condition was necessary until labor was induced and a normal child was delivered on May 22, 1957. During the last 4 months of pregnancy, a bruit had been heard over the left flank. Since delivery, the patient had remained virtually at rest in bed.

On examination the blood pressure was 180/80; the pulse rate during rest was 120 beats per minute. Funduscopic examination of the eyes revealed narrowing, grade 2, and focal constrictions, grade 1 (on a grading basis of 1 to 4), of the retinal arterioles, but no sclerosis was discernible. Examination of the heart revealed gross cardiac enlargement and a soft systolic apical murmur. A loud bruit and thrill were found in the left upper part of the abdomen anteriorly, laterally, and posteriorly. These were continuous and were accentuated during systole. The point of maximal intensity was immediately over the kidney posteriorly.

Laboratory examinations gave the following results: The urine contained albumin, grade 3; the blood showed 8.6 Gm. of hemoglobin per 100 ml. and 18 mg. of urea per 100 ml. An excretory urogram, cystoscopy, and a left retrograde pyelogram showed a nonfunctioning left kidney that appeared about half the size of its mate. A roentgenogram of the chest showed cardiac enlargement.

In order to delineate the nature of the suspected arteriovenous communication, a special catheterization procedure was carried out. The right atrial pressure was elevated, being 15/10 mm. Hg. The oxygen saturation of blood withdrawn from the superior vena cava was 61 per cent and that withdrawn from the inferior vena cava at the junction of the iliac veins was 64 per cent; both these values were below normal. The oxygen saturation of blood withdrawn from the inferior vena cava in the region of the orifices of the renal veins, however, was 95 per cent. The catheter was introduced into what was apparently the left renal vein, and blood withdrawn from this site had an oxygen saturation of 97 per cent, which was not significantly different from blood simultaneously withdrawn from the radial artery. A catheter was introduced into the right femoral artery and advanced to the level of the diaphragm. After injections of dye into the aorta at the diaphragm, dye-dilution curves were recorded simultaneously from the renal artery and from the “right-heart” catheter when its tip was in the left renal vein and after withdrawal to the inferior vena cava at the diaphragm. The contour of the dye curves indicated a rapid recirculation time (11 seconds), as is typical of patients with large arteriovenous fistulas. The dye curves recorded from the left renal vein and inferior vena cava indicated a large arteriovenous fistula in or near the left kidney. A selective angiocardiogram was made by injecting contrast media through a catheter, whose tip was in the right renal artery. The picture was consistent with an arteriovenous fistula at the hilus of the left kidney. Estimation of the size of the shunt based on saturation data indicated that 69 per cent of the inferior vena caval blood was from the shunt.

At the time of nephrectomy the left kidney was found to be about half normal size and adherent to surrounding structures. The renal artery approached the kidney in a downward direction and communicated directly with the renal vein in the

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**Table 1.—Cardiac Catheterization in Case 1**

<table>
<thead>
<tr>
<th></th>
<th>1950</th>
<th>1954</th>
<th>1955</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Output (L./min.)</td>
<td>Index</td>
<td>Output (L./min.)</td>
</tr>
<tr>
<td>On air</td>
<td>7.7</td>
<td>3.8</td>
<td>5.9</td>
</tr>
<tr>
<td>On oxygen</td>
<td>8.0</td>
<td>3.9</td>
<td>10.7</td>
</tr>
<tr>
<td>Recirculation time</td>
<td>—</td>
<td>12 sec.</td>
<td>31 sec.</td>
</tr>
<tr>
<td>Radial artery pressure (mm. Hg)</td>
<td>—</td>
<td>173/84</td>
<td>137/80</td>
</tr>
</tbody>
</table>

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**Fig. 1. Case 1. Aortogram in 1954 showing a greatly enlarged and tortuous renal artery terminating in an aneurysmal sac.**

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hilus of the kidney. There was a saecular dilatation of the renal vein about the size of a golf ball, and the remainder of the visible portion of the renal vein was at least three times normal size and thickened. The ostium of the arteriovenous fistula was 8 mm. in diameter.

The patient's postoperative course was uneventful. Her blood pressure in the immediate postoperative period was 130/80.

Case 3. A 39-year-old woman was referred to the clinic by her physician, Dr. James R. Machan of Fort William, Ontario, in April 1955, because of hypertension of 3 years' duration, cardiac enlargement with early cardiac failure, and an abdominal mass that was considered to be an arteriovenous aneurysm. She mentioned also severe frontal headaches, weakness, and easy fatigability. She denied urinary symptoms.

The pertinent findings at physical examination included a precordial systolic murmur, grade I, and evidence of cardiac enlargement. The blood pressure averaged 192/105. A firm palpable mass was present in the left upper quadrant of the abdomen, and over the mass a thrill and a loud continuous bruit with systolic accentuation were noted. The bruit radiated from the mass and could be detected throughout the abdomen, the left loin, and the chest. Examination of the ocular fundi disclosed narrowing of the retinal arterioles, grade 2, and areas of focal constrictions and hypertensive sclerosis, grade 1.

Laboratory examinations of the urine and blood (including blood urea) yielded normal findings. Roentgenographic examination of the thorax disclosed cardiac enlargement, and the electrocardiogram was consistent with left ventricular hypertrophy.

Study of the upper part of the urinary tract by excretory urography and left retrograde pyelography disclosed a rounded enlargement of the upper pole of the left kidney, and the upper calyx appeared flat, elongated, and broad. The infundibulum leading to the upper calyx was broad and irregular with a hook-shaped deformity in a branch of the middle calyx (fig. 4). An aortogram disclosed irregular pooling of medium over the region of the left kidney (fig. 5). A clinical diagnosis of hypernephroma or benign vascular tumor with arteriovenous fistula was made, and surgical intervention was advised.

Surgical exploration revealed a vascular mass occupying the hilus and upper pole of the left kidney. The left renal vein was strikingly enlarged, and the renal artery was divided into several different branches. Nephrectomy was performed. Examination of the excised specimen (fig. 6) disclosed a hypernephroma, 10 cm. in its greatest diameter, occupying the upper pole of the kidney. The arteriovenous fistula consisted of branches of the renal artery and vein which entered an aneurysmal sac that measured 6 by 6 cm. and was situated in the region of the hilus. Two large venous pools that drained into the renal vein were situated within the neoplasm.

A roentgenogram of the thorax made on the tenth postoperative day disclosed a distinct decrease in size of the cardiac shadow. Hypertension persisted, however, and a value of 170/110 was noted.

In response to inquiry the patient's physician in her home locality stated that he had examined her in January 1958. She had remained well during the intervening 3 years, had gained 15 pounds and was free of symptoms. The blood pressure remained elevated to 200/100. Examination of the ocular fundi, however, yielded normal findings. The cardiac enlargement had decreased.

Classification

Because of the considerable inaccuracy and ambiguity in the titles of articles dealing with intrarenal or parenchymal shunts, fistulas of the renal artery and vein, and fistulas in the stump of the renal vessels following nephrectomy, the following simple classification is proposed. It is based on the location and the nature of the communication: I. Arteriovenous fistula of the kidney. II. Postnephrectomy arteriovenous fistula.

The term "arteriovenous fistula of the kidney" would include those fistulas occurring within the kidney substance itself as well as those of extrarenal origin involving the renal
artery and vein. An aneurysmal sac is commonly present as part of the fistulous tract.

Although the term, "arteriovenous fistula of the renal vessels," might seem satisfactory, it would include fistulas occurring in the stumps of the renal vessels following nephrectomy, and could therefore have two entirely different meanings. The term, "postnephrectomy arteriovenous fistula," of course, implies a fistula between the stumps of the renal artery and vein.

The need for some standardization can quickly be appreciated by reviewing the literature because of several, almost completely misleading titles. The term, "arteriovenous aneurysm," commonly has been employed,
but since the fundamental lesion is a fistula, not an aneurysm, ‘‘clarity is lost and confusion is served by maintenance of other terms which have been used to indicate some of the manifestations of abnormal arteriovenous communication.’’

INCIDENCE

The scarcity of reports suggests that arteriovenous fistulas in the kidney or the renal vessels when the kidney is present are rare. Several cases included in previous reviews of the literature and listed in the Quarterly Cumulative Index Medicus have not been included in this review for one reason or another. In the case reported by Hollingsworth the diagnosis was not confirmed. This case, along with 2 others, actually represents a postnephrectomy arteriovenous fistula. From the clinical and physiologic standpoint, such fistulas resemble peripheral rather than renal arteriovenous fistulas and should not be included in a discussion of the latter.

The case reported by Garritano and co-workers is excluded also because the same case was reported by Kirby and associates a year earlier.

The fact that 10 of the total 15 cases (including the present series) reported to date have been noted in the past 4 years indicates that arteriovenous fistulas of the kidney or renal vessels are much commoner than had previously been thought.

Since the bruits produced by these fistulas are easily heard, we should like to suggest that auscultation be carried out over the renal areas in patients who have unexplained cardiac enlargement, unexplained deformities of the kidney or renal pelvis or both, unexplained cardiac failure, and hypertension. In this way fistulas may be detected in their early stages.

ETIOLOGIC ASPECTS

The abnormal arteriovenous communications in the kidney and renal vessels in the 15 cases (including our 3 cases) were thought to be congenital in 6, and to be due to trauma in 6 and to malignant tumors in 3.

Concerning 5 of the 6 cases listed as traumatic in origin, there is little doubt as to the cause since, in 3, bullet wounds, in 1 a stab wound, and in 1 a surgical procedure involved the renal vessels. The cause was uncertain in only 1 of the 6 cases (table 2).

Of the 6 arteriovenous fistulas listed as being of congenital origin, 2 were intra-renal, and 4 were in the renal vessels. All 6 patients had fistulas that were aneurysmal in nature.

Simple aneurysm of the renal artery seems to be much commoner than arteriovenous fistula. To May 1955, 120 cases had been reported. It is easy to visualize how such an aneurysm may erode its way into the renal vein while in the process of rupturing, with a fistula between the renal artery and vein as the final result. Additional support is given to this thesis by the fact that 2 of the 6 patients with congenital fistulas were free of symptoms until they were 60 years of age or older.

It is well known that one of the factors leading to the development of symptoms from arteriovenous communications is the duration of the shunt. This makes survival until age 60 years most unlikely unless the fistula is of more recent origin.
### ARTERIOVENOUS FISTULA OF THE KIDNEY

#### Table 2.—Summary of Cases in Literature

<table>
<thead>
<tr>
<th>Author</th>
<th>Date, age, sex</th>
<th>Presenting complaint</th>
<th>Symptoms and findings</th>
<th>Blood pressure (mm. Hg)</th>
<th>Cause</th>
<th>How diagnosed</th>
<th>Outcome: heart and blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Varela</td>
<td>1928, 28 M</td>
<td>Severe dyspnea</td>
<td>Enlarged heart, failure, bruit rt. flank</td>
<td>160/80 Congenital</td>
<td>At necropsy</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td>Rieder</td>
<td>1942, 29 M</td>
<td>Palpitation, dyspnea</td>
<td>Enlarged heart, failure, bruit rt. flank</td>
<td>220/120 Congenital</td>
<td>Normal; 125/90</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pearse and MacMillan</td>
<td>1947, 60 M</td>
<td>Hematuria</td>
<td>Not given</td>
<td>160/60 Congenital</td>
<td>At operation</td>
<td>?; 125/75</td>
<td></td>
</tr>
<tr>
<td>Hamilton et al.</td>
<td>1953, 29 M</td>
<td>Severe dyspnea, cough, fatigue</td>
<td>Marked pulm. edema, heart failure, bruit in left lumbar region</td>
<td>180/110 Adenocarcinoma</td>
<td>At operation</td>
<td>Normal; 140/90</td>
<td></td>
</tr>
<tr>
<td>Pélot et al.</td>
<td>1954, 26 M</td>
<td>Dyspnea, cough</td>
<td>Heart greatly enlarged; gallop rhythm; bruit, grade 4 retinopathy</td>
<td>210/130 Bullet injury, 1952</td>
<td>At operation</td>
<td>Normal; 135/75</td>
<td></td>
</tr>
<tr>
<td>Vest</td>
<td>1954, 3 F</td>
<td>Hematuria</td>
<td>—</td>
<td>—</td>
<td>Nephrolithotomy</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Baron and Koenemann</td>
<td>1955, 5 F</td>
<td>Headaches</td>
<td>Hypertension, bruit in left flank</td>
<td>146/110 Stab wound</td>
<td>At operation</td>
<td>Normal; 120/80</td>
<td></td>
</tr>
<tr>
<td>Kirby et al.</td>
<td>1955, 22 M</td>
<td>Dyspnea, weakness</td>
<td>Enlarged heart, bruit</td>
<td>180/60 Bullet wound, 1952</td>
<td>By aortogram</td>
<td>Normal; 120/74</td>
<td></td>
</tr>
<tr>
<td>Slominski-Laws et al.</td>
<td>1956, 19 F</td>
<td>Dyspnea, edema</td>
<td>Heart enlarged</td>
<td>120/80 Congenital</td>
<td>By aortogram</td>
<td>Not stated</td>
<td></td>
</tr>
<tr>
<td>Myhre</td>
<td>1956, 66 F</td>
<td>Hematuria</td>
<td>Heart normal, bruit, pain in rt. flank</td>
<td>140/70 Adenocarcinoma</td>
<td>By cardiac catheterization and aortogram</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Bohne and Henderson</td>
<td>1957, 63 M</td>
<td>Dyspnea</td>
<td>Heart enlarged, bruit</td>
<td>? Congenital</td>
<td>From aortogram</td>
<td>Normal; (?)</td>
<td></td>
</tr>
<tr>
<td>This series</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 1</td>
<td>1954, 42 M</td>
<td>Pulpituation, fatigue, sweating</td>
<td>Heart enlarged, bruit</td>
<td>174/84 Bullet wound, 1926</td>
<td>At catheterization plus aortogram</td>
<td>Normal; 128/86</td>
<td></td>
</tr>
<tr>
<td>Case 2</td>
<td>1957, 30 F</td>
<td>Abdominal pain, dyspnea</td>
<td>Heart enlarged, heart failure</td>
<td>180/80 Injury (†) 1931</td>
<td>At catheterization plus angiogram</td>
<td>Normal; 130/80</td>
<td></td>
</tr>
<tr>
<td>Case 3</td>
<td>1955, 39 F</td>
<td>Hypertension, lump in abdomen</td>
<td>Heart enlarged, heart failure</td>
<td>206/110 Hyperephromia</td>
<td>From aortogram</td>
<td>Elevated; 200/100</td>
<td></td>
</tr>
</tbody>
</table>
Further evidence is seen in our case 2. This patient was in good health until age 29 years when symptoms developed, which, within 3 months, progressed to marked congestive failure. Such a picture is inconsistent with the concept of a life-long arteriovenous fistula.

From the standpoint of renal carcinoma, it is probable that considerably more tumors result in abnormal arteriovenous shunts than the present figures would indicate. Careful auscultation with the stethoscope over the site of a renal tumor may well disclose a significant percentage of arteriovenous bruits. In the 3 cases of malignant tumor of the kidney in this series, including our case 3, erosion of the renal vessels by the tumor was the cause of the shunt.

Clinical Features

Many of the clinical features noted in the 15 cases of abnormal renal arteriovenous communication, which make up the world's literature to date, are summarized in table 2. Our 3 cases are included.

The most notable features are hypertension and cardiac enlargement with varying degrees of myocardial insufficiency. These 2 features, particularly the hypertension, tend to set these fistulas apart from arteriovenous fistulas in other parts of the body. Peripheral arteriovenous communications do not result in hypertension and do not commonly result in heart failure. Hypertension is truly a hallmark of an arteriovenous fistula of the kidney. Dramatic relief of the cardiac failure, as well as of the hypertension following corrective surgical procedures, is equally characteristic.

The blood pressure, which was recorded in 12 of the 15 cases reviewed, was elevated in 11. Preoperative and postoperative blood pressures were recorded in 9 cases, and in all but 1 the elevated pressure returned to normal after operation.

The size of the heart was recorded in 12 of the patients, and of these 11 had cardiac enlargement. Lowered reserve or heart failure was present in 12 of the 15 patients. The cardiac symptoms were relieved in every patient whose fistula was removed.

In 6 cases the size of the heart was recorded both before and after operation. In 5 the heart regained its normal dimensions, and in 1 it returned to almost normal size.

In most cases the hypertension was associated with typical hypertensive changes in the retina, and in 1, the retinopathy was of the group 4 category. A complete return of the ocular fundi to normal followed nephrectomy in every case.

The bruits associated with these arteriovenous fistulas have the classic auscultatory features of fistulas in other parts of the body but differ somewhat in being widespread and diffuse and not sharply localized. A thrill is commonly palpable. So far as is known now, an easily detectable bruit is present in all such arteriovenous fistulas.

Pain low in the abdomen was an occasional symptom, and gross hematuria was the presenting symptom in 3 cases.

The urine may be normal, or it may contain considerable albumin, casts, and erythrocytes. Except for the first patient whose case is recorded in the literature none of the patients had a tendency to elevated blood urea; the 1 exception was in terminal condition at the time of diagnosis.

Diagnostic Procedures

Excretory urograms were made for 12 of the 15 patients and showed abnormalities in 11. In the twelfth case that was reported by Kirby and associates, the urogram showed nothing abnormal.

The first instance in which the diagnosis was confirmed and the lesion was demonstrated without the benefit of operation or postmortem examination did not occur until 1954 when an aortogram demonstrated the presence of the lesion (our case 1). In the last 7 cases, including our cases, an aortogram or angiogram has demonstrated the arteriovenous fistulas described.

Special catheterization procedures were carried out in 3 instances. The demonstration of arterIALIZATION of the inferior vena caval blood, and particularly of arterial blood from the renal vein, should be considered diagnostic. A marked reduction in the recircula-
tion time as shown by the indicator-dilution test is characteristic of arteriovenous shunts. Increased cardiac output should be demonstrable.

The indicator-dilution curves in arteriovenous fistulas of the kidney were studied recently by Fox and Wood. Preoperative and postoperative curves shown in figure 7 are part of the catheterization study done in our case 1.

**Pathophysiologic Features**

The overloading of the circulation because of high cardiac output is characteristic of arteriovenous fistulas from the renal as well as from the peripheral vessels. The blood volume is increased. The pulse pressure becomes widened through lowering of the diastolic pressure in peripheral arteriovenous fistulas and through increase in systolic pressure and a relative lowering of the diastolic pressure in arteriovenous fistulas of the kidney (table 2).

The degree of alteration of the circulatory dynamics is proportioned to the size of the fistula, the gradient of pressure, and the length of time the fistula has been present. The factor of size is demonstrated by a patient who had a large fistula between the abdominal aorta and the inferior vena cava. Only 3 weeks elapsed between the trauma that caused the fistula and the development of severe intractable heart failure.

There are several reasons for the commonly noted cardiac enlargement and heart failure in cases of renal arteriovenous fistulas. The pressure as well as the pulsatile character of the arterial blood in the renal artery distal to the fistula is decreased and with it the rate of flow to the kidney. This gives an effect similar to that seen in a Goldblatt kidney, and hypertension almost invariably results. An atrophic renal cortex is characteristic, and, at times, has been associated with hydropnephrosis.

With the development of hypertension, the pressure gradient across the fistula is increased, and a vicious cycle is established. The hypertension augments the flow through the fistula, which tends to cause further increase in the cardiac output. The latter, in turn, presents more blood at increasing pressures to the fistulous tract.

Of great significance in illustrating the importance of the renal factor in the production of hypertension, cardiac enlargement, and heart failure is the return of the blood pressure and the size of the heart to normal in almost every case following surgical removal of the kidney along with the fistulous tract.

**Summary**

Only 5 reports of cases of renal arteriovenous fistula appeared before or in 1953. Subsequently, 7 isolated reports brought the total to 12, and the 3 that we are reporting make 15.

In reviewing the subject, the classification, incidence, causation, clinical features, diagnostic procedures, and pathophysiologic features were considered. Good evidence supports the thesis that in cases of congenital arteriovenous fistula an eroding renal arterial aneurysm is etiologic.

Outstanding clinical features were hypertension (to group 4) and myocardial insufficiency; these contrast markedly with features of the usual peripheral arteriovenous fistula. Dramatic relief of heart failure and lowered reserve followed corrective operation in every case in which these features were recorded; relief of hypertension followed corrective operation in 11 of 12 cases in which
this feature was mentioned. A loud, diffuse, continuous bruit invariably was present.

Preoperative and postoperative catheterization and indicator-dilution studies, angiograms and aortograms are diagnostic measures emphasized in our 3 cases. Arterialization of blood in the vena cava, greatly increased cardiac output, and markedly shortened recirculation times of the dye were found. Excretory urograms were abnormal in 11 of the 12 cases in which they were made.

The mechanism producing hypertension appears related to loss of pressure, decrease in flow, and loss of pulsatile character in the renal artery distal to the fistula, giving a Goldblatt-kidney type of end result. A vicious cycle of increasing hypertension followed by increasing flow through the shunt makes heart failure inevitable.

Since 10 of the 15 cases were reported in the last 4 years, indicating that the lesion is commoner than previously supposed, we advise auscultation over the renal regions in patients with cardiac enlargement or failure of unknown causation, unexplained deformities in urograms, and renal tumors.

SUMMARIO IN INTERLINGUA

Solmente 5 reportos de arterio-venose fistula renal esseva publicate ante o in 1953. Subsequentemente, reportos isolate augmentava le total a 12. Le 3 casos del presente reporto completa un total de 15.

Es presentate un revista del thema, incluse le classification, le incidentia, le causation, le characteristicas clinic, le technicas diagnostic, e le aspectos pathophysiologic. Il existe forte indications in supporto del theses que in casos de congenite fistula arterio-venose, un erodente aneyrysma renalarterial es le factor etiologic.

Eminente aspectos clinic essea hypertonie (a gruppo 4) e insufficientia myocardial. Istos contrasta maratemente con aspectos del usual fistulas arteriovenose peripherie. Un dramatic alleviamento del disfallimento cardiae e del reducite reserva sequve le chirurgia corrective in omne casos in que le mentionate aspectos essea presente. Alleviamento del hypertension sequve le chirurgia corrective in 11 del 12 casos in que iste aspecto essea mentionate. Un forte, diffuse, e continue ruito essea presente in omne casos.

Studios de catheterismo pre- e postopera- tori e studios a dilution de indicatores si ben como angiogrammas e aortogrammas es mesuras diagnostic de importantia sublineate in nostre 3 casos. Esseva constatate arterialisation del sanguine in le vena cave, marcate augmentos del rendimento cardiae, e multo reduceite tempores de recirculation del tincitura indicatori. Urogrammas excretori essea anormal in 11 del 12 casos in que illos essea effectuate.

Le mecanismo que produce le hypertension pare esser relationate a perdita de tension, reduction de fluxo, e perdita de character pulsatil in le arteria renal distal al fistula, resultante in un effecto termin 1 del typo de ren de Goldblatt. Un circulo vitioso de crescente hypertension sequite per un fluxo crescente via le derivation rende le supervenientia de disfallimento cardiae satis inevitabile.

Viste que 10 del 15 casos esseva reportate in le passate 4 annos, lo que indica que le lesion es plus commun que previemente supponite, nos recommenda auscultation supra le regiones renal in patientes con allargamento o disfallimento cardiae de etiologia obscure, con non-explicate deformitates del urogramma, e con tumores renal.

REFERENCES

I think this truth must needs follow and be apparent to all men.
1. First, That the blood is continually, and without any intermission, transmitted out of the vena cava into the arteries, in so great abundance, that it cannot be recruited by those things we take in, and insomuch that the whole mass of blood would quickly pass through.

2. In the second place, that continually, duly, and without cease, the blood is driven into every member and part, and enters by the pulse of the arteries and that in far greater abundance than is necessary for nourishment, or than the whole mass is able to furnish.

3. And likewise thirdly, that the veins themselves do perpetually bring back this blood into the mansion of the heart.

These things being prov'd, I think it will appear that it doth go round, is returned, thrust forward, and comes back from the heart into the extremities, and from thence into the heart again, and so makes as it were a circular motion.—William Harvey. De Motu Cordis, 1628.
Arteriovenous Fistula of the Kidney: New Observations and Report of Three Cases
CHARLES H. SCHEIFLEY, GUY W. DAUGHERTY, LAURENCE F. GREENE and JAMES T. PRIESTLEY

Circulation. 1959;19:662-671
doi: 10.1161/01.CIR.19.5.662
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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