The two latter are considered by some authorities to constitute "closed" tuberculosis, but the term does not appear to be quite accurate, as the pelvis is so extensively involved. It is difficult to place this particular specimen in the groups mentioned. If placed in Group 3 almost complete destruction of the whole kidney would be implied, and this is far from being the case. Such a specimen might be rightly considered as one of "closed renal tuberculosis," and be placed accordingly in a final and fifth group under that heading. The term "closed" could then be considered inadmissible in cases falling into Groups 3 and 4.

I am greatly indebted to Professor Seymour Barling for permission to publish this case and for his advice and help.

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NEW GROWTHS OF THE KIDNEY*

WITH ANALYSIS OF SIXTY-FIVE CASES

BY

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(With Special Plate)

The following survey is entirely limited to new growths occurring primarily in the kidney, and excludes all those renal enlargements due to hydronephrosis, calculus, or cystic disease. I have collected all my private and hospital cases, and, for the purpose of arriving at some statistical figures, have added some notes of specimens of renal tumours from the pathological museums of the Cancer Hospital and St. Paul's Hospital. I would like to thank my colleagues for their permission to make use of these cases. In this way I have fairly complete clinical and pathological records of sixty-five cases from which to draw my conclusions.

The subject of new growths in the kidney presents many features of great interest—interest to the pathologist, clinician, and surgeon alike. Pathologists have for several years expressed many and varied opinions as to the origin and the true nature of these tumours, and even at the present time no definite conclusion has been arrived at. The clinician has improved his methods of examination in so marked a degree that he is able to demonstrate almost with certainty whether a renal growth is present or not.

CLASSIFICATION AND PATHOLOGY

With the uncertainty that exists as to the true pathology of renal tumours it is somewhat difficult to attempt any exact classification, but for clinical purposes the following will cover all forms of growth:

- RENAL TISSUE
  - *Innocent* ... Adenoma; Fibroma; Lipoma; Angioma
  - *Malignant* ... Carcinoma—Papillary adenocarcinoma; Alveolar adenocarcinoma; Hypernephroma; Sarcoma, including embryonic tumours
- RENAL PELVIS
  - *Innocent* ... Papilloma
  - *Malignant* ... Villous carcinoma; Squamous epithelioma

* A clinical lecture delivered before the Fellowship of Medicine on November 24th, 1932.

The benign tumours of the renal parenchyma can be dismissed in a few words. They are usually found in post-mortem examinations, and very rarely give rise to any clinical symptoms. Adenomata are found as small rounded nodules, especially in a kidney affected with interstitial nephritis, and are of some interest in that they may form a focus in which carcinoma is likely to arise. Small fibromata and lipomata have been found in the kidney, and I[1] have described a case of cavernous angioma of the kidney which gave rise to such profuse haematuria as to necessitate nephrectomy as a life-saving measure. Only about twelve similar cases have been recorded.

The Neoplasms

It is the malignant new growths of the renal tissue that have caused so much discussion as to their true pathology. At one time they were variously classified as sarcoma and carcinoma, and the first great interest was kindled when, in 1883, Grawitz[2] published his paper describing these tumours as arising from "adrenal rests." The latter are small subcapsular islets of suprarenal tissue left in immediate conjunction with the renal cortex in the process of development. Grawitz based his opinion on the similarity of these cells to those of the suprarenal cortex—particularly in that they both contain fat and highly refractile particles—on the fact that the cells are quite unlike those of the normal renal tubule, and that the growths are found beneath the renal capsule, a place where adrenal rests are found. He also made a point of the occurrence of a fibrous core in the central part of the tumour, from which pass septa dividing the tumour into lobules. In 1893 Sudek[3] described these Grawitzian growths as actually arising from the renal tubules, and in 1908 Stoerk[4] showed that the tumours had a papillary basis, an opinion which was upheld by Wright[5] in 1922, after a very critical examination of nineteen specimens of renal growths, most of which were removed from patients under my care in the Cancer Hospital. Doubt has been expressed by some as to whether aberrant suprarenal rests do actually occur under the renal capsule, but Shaw Dunn[6] and Ewing[7] definitely state that they do exist. Arguments have been put forward from the embryological side that these adrenal rests may originate from the remains of the Wolffian ridge, from which the testes and ovaries are developed, and in which organs somewhat similar tumours have been rarely recorded. These tumours, however, differ from renal tumours in that they contain no fat and no haemorrhagic or necrotic areas, while they further possess a uniform structure. Though I cannot attempt to discuss the histogenesis of these tumours, there exists a very interesting specimen, which to my mind throws a good deal of light on the origin of them. This specimen was found in the kidney of a man aged 75 who died in the Cancer Hospital from advanced epithelioma of the tongue. He had no symptom referable to the urinary organs, but on post-mortem examination a small, rounded, yellowish tumour about 1½ cm. in diameter was found wholly embedded in the renal tissues. It was separated from the capsule of the kidney by a covering of normal renal substance quite 1 cm. in thickness, and thus could not possibly have originated from a subcapsular adrenal rest. In cross-section the growth is lobulated, necrotic in the centre, shows some small areas of haemorrhage, and is surrounded by a thin capsule. Histologically the structure is typical of a hypernephroma with large vacuolated cells.

Ewing, in his last work on *Neoplastic Diseases*, gives his opinion that these tumours arise in the renal epithelium, and divides them into papillary and alveolar adenocarcinoma, reserving the term "hypernephroma" for the rare tumours arising from adrenal rests. This division

really confuses the issue, as the term " hypernephroma " has for many years been used to include all those renal tumours which show areas of necrosis and haemorrhage, and microscopically exhibit large vacuolated cells containing fat. It is by no means easy to classify these growths from their histological appearances, as various parts of the same tumour seem to approximate to more than one variety, while in some vacuolation of the cells may be due to fatty degeneration. After a critical survey of these growths at the Cancer Hospital by Dr. Piney and Dr. Hawksley, I have separated those showing a distinct papillary or alveolar structure into separate classes, and collected all those showing masses of large, clear, vacuolated cells into another group, for which I have retained the term " hypernephroma," as will be seen in the tabulated list of cases.

It should be recognized that these growths are really all carcinomata, and that the word " hypernephroma " is unfortunate, as it implies a growth arising in an adrenal rest; in fact, Hugh Young[8] suggests that it should be given up and the term " nephroma " used to cover all these growths. For the present I look upon them all under the generic term of " carcinoma," although I must admit that they differ very largely in their virulence towards metastatic spread, some remaining localized to the kidney for months or years, whilst others rapidly infiltrate the kidney and give rise to early metastases.

There remains the group which I have included in the classification as sarcomata. Sarcomata of the kidney form a small proportion of malignant tumours, and may exist as round, spindle-celled, or mixed-celled growths, sometimes including striped or even unstriped muscle fibres or other forms of developed tissue. In one particular form, described by Wilms[9] as embryonic adenosarcoma, there are, in addition to muscle fibres, cartilage, or fat, masses of tubules lined by cuboidal epithelial cells. The tumours occur usually in infants and children under 5 years of age, but may, like other sarcomata, be found in adults. Sarcomata rarely cause haemorrhage, but first attract attention by the appearance of a tumour in the loin, which rapidly increases in size and which is exceedingly malignant.

Hypernephroma

Carcinomata are growths of later age, and in my series of fifty-one cases the following table gives the ages in decades:

Decade	No. Cases	Percentage	Decade	No. Cases	Percentage
20 to 30 years	1	1.9	51 to 60 years	19	37.2
31 to 40 ,,	2	3.9	61 to 70 ,,	11	21.5
41 to 50 ,,	13	25.4	71 to 80 ,,	5	9.8

These figures approximate very closely with those given by Garceau[10] in 176 cases. The youngest case in my series was that of a woman of 27, who was under the care of my colleague Mr. Percival Cole for enlarged glands in the left supraclavicular fossa. On removal these presented the histological appearance of hypernephroma. Later on, the patient developed haematuria and died with further glandular metastases from a large hypernephroma of the left kidney. The oldest patient was aged 75—the one who died from epithelioma of the tongue, and in whom the very early carcinoma of the kidney was found. There were thirty-two men and nineteen women presenting hypernephromata, and, whereas most authorities record a larger percentage occurring on the right side, in my series there are thirty-two on the left to eighteen on the right. In one case the side has not been mentioned.

The tumour may occur in any part of the kidney, though it is most frequently found in the upper or lower pole. In some cases it arises in the central portion extending into the hilum, and in two cases it seemed to cause hydronephrosis by direct pressure on the pelvis of the kidney. It is at first globular in shape, surrounded by a fibrous capsule of varying thickness and compressing the adjacent renal tissue—almost suggesting an innocent tumour—though in some cases this capsule is absent and the growth directly invades the kidney substance. On section the macroscopic appearance is fairly characteristic. The surface presents a mottled, lobulated, or mosaic appearance, divided into lobules by the fibrous septa. There will be seen areas of haemorrhage, some of fresh blood and others of organizing clot. Areas of necrosis are also found, occasionally to a fluid consistency. The growth gradually extends, invading a calix and causing haematuria ; it also tends to spread into the veins, reaching the renal vein and giving rise to metastases in the lungs via the vena cava. The growth may gradually distend the kidney, or may form a rounded projection from it, infiltrating the capsule and spreading into the perirenal fat. In this way it may, in advanced cases, become adherent to the diaphragm, liver, or colon. Extension may also occur in the lymphatic glands along the abdominal aorta, to the mediastinal glands, or even to the cervical or inguinal glands. The most common metastatic deposits are seen in the lungs, in the liver, and in the bones—especially in the bodies of the vertebrae—whilst in two cases secondary growth was present in the humerus and femur, causing a spontaneous fracture in the latter.

SYMPTOMATOLOGY

The prominent symptoms of renal growth are haematuria, tumour, and pain.

Haematuria is the most common symptom, and in the majority of cases is the first to attract the attention of the patient. It is present in over 90 per cent. of all cases, and occurs as the initial symptom in nearly 70 per cent. The blood may be slight from the surrounding nephritis, or it may more frequently be due to the direct involvement of a calix or the renal pelvis by the growth. It is often profuse and accompanied by the passage of worm-like clots, and sometimes seems to follow exertion or injury. In one case haemorrhage followed a fall and in another it occurred after mountain climbing. It is usually sudden in onset, may last several days, and then clear away to recur after a time varying from weeks to months. In one case under my care attacks of fairly profuse haematuria had been present for four years before nephrectomy was performed, and this patient still remains well seven years after his kidney was removed. More usually, however, the initial attack of haematuria has been within twelve months of the patient first coming under observation. Haematuria occurs in all forms of growth, but is infrequent with the sarcomata and the embryonal tumours of children. In the intervals between the attacks of profuse haematuria, microscopic blood may be present in the urine from haemorrhage in the compressed renal tubules surrounding the growth. In cases of profuse haematuria clots may be formed in the renal pelvis and ureter, the passage down which may cause pain very similar to renal colic. The clots are thin, rounded, and worm-like in shape, and if found in the urine are certainly indicative of a severe renal bleeding. It should be stated that the severity of the haemorrhage bears no relation to the size of the growth, for I have seen quite small growths give rise to profuse bleeding, and large growths which have caused no macroscopic blood in the urine.

In one case a man, aged 49, was found to have a palpable tumour in the left subcostal area, and was thought by a surgeon in South Africa to have a carcinoma of the colon.

On laparotomy a renal tumour was found, the incision was closed, and he was subsequently sent to England. He had no haematuria at any time, but a pyelographic examination showed considerable deformity of the renal pelvis and calices. Nephrectomy was performed, when an extensive growth was found, and the patient developed metastases in the abdominal glands after a year.

A small central growth may invade the pelvis of the kidney comparatively early in its spread, and so give rise to haemorrhage.

Pain is present in some form in the majority of cases. It may be severe from ureteric colic, when clots pass down the ureter, or there may be pain and frequent desire to pass urine from retention of clots in the bladder. There may be occasional attacks of lumbar aching, due to increased tension in the kidney from small haemorrhages into the growth, or more or less continuous aching in the loin from involvement of the tissues surrounding the kidney. In advanced cases it may exist as a nerve pain due to pressure on nerve roots from metastases in the vertebral bodies. I cannot trace any relation or difference in the pain experienced in growths of the upper, as compared with the lower, pole of the kidney.

A *tumour* may be palpable, and in children is usually the first sign of a renal sarcoma. If the tumour is large it causes a rounded swelling, which can be grasped bimanually and felt to descend on inspiration. The colon occupies a position anterior to it and may be palpated, or may produce a resonant note on percussion. The mass is usually smooth, but in some cases and in thin subjects rounded bosses can be detected on the surface. A tumour of the upper pole may not be palpable, but the lower pole of the kidney may be felt to descend to more than a normal extent on deep inspiration. In one case under my care a fairly large tumour of the upper pole of the right kidney dislocated the liver around a transverse axis, so that the anterior hepatic margin descended well below the costal margin. A renal tumour which appears fixed in the loin implies that perirenal infiltration has already taken place, and is a bad prognostic indication.

Albumin may be present in the urine, in some cases due to traces of blood, in others due to nephritis. Albarran states that it may result from toxic nephritis in the other kidney. Pyuria is rarely present unless the kidney is also the seat of calculous disease. In this series of cases calculus was present in two instances—one in which carcinoma was associated with pyonephrosis, and another where there was epithelioma of the renal pelvis. *Varicocele* has been stated to be frequently present, but I can only find a note of this in two out of fifty-one cases. If it is present and does not disappear upon lying down, it may be due to pressure on the spermatic vein by the growth or by glands, or possibly to extension of the growth into the renal vein. *Pyrexia* was present in two cases in the absence of any infection. It is stated by Israel[11] to be fairly frequent, but I cannot confirm this.

DIAGNOSIS

The combination of haematuria, localized lumbar pain, and the palpation of a tumour in the renal area should form fairly conclusive evidence of a new growth in the kidney. In calculous disease pyuria will be present, whilst in polycystic disease both kidneys will nearly always be palpable, although one may be much larger than the other. In tuberculous disease the kidney is rarely very much enlarged, pus is present in the urine, the average age is between 15 and 30 years, tubercle bacilli may be found, and increased frequency of micturition is a prominent symptom. Haematuria may occur with hydronephrosis.

In cases where haematuria is the only symptom it cannot be too strongly urged that further and complete urinary examination should be immediately carried out. So often do we see cases in which intermittent attacks of profuse haematuria have cleared up with rest, and the patient—and probably the doctor—has been lulled into a sense of false security and valuable time has been lost in which the growth is spreading or metastases have occurred. No one would leave a case of haemoptysis or haematemesis without full examination—why leave a case of haematuria? Haematuria should be looked upon as a symptom which may be of grave import, and therefore demanding complete examination.

Cystoscopic examination should be insisted upon. I am often asked if cystoscopy should be carried out during a period of haematuria, to which my reply is that I prefer it then. If the bleeding comes from the kidney, irrigation of the bladder will quickly produce a clear medium, blood may be seen to be emitted from one ureteric orifice, and the haematuria immediately localized to that kidney. On the other hand, should the bleeding come from a vesical growth or an enlarged prostate, careful irrigation with silver nitrate solution 1 in 4,000 or adrenaline will usually produce a medium clear enough for diagnostic purposes. The two most likely causes of painless intermittent haematuria are growth of some type in the bladder or in the kidney. I have seen cases in which haematuria has been accompanied by aching in the loin and by a renal tumour, in which cystoscopic examination showed the presence of a growth in the bladder obstructing the ureteric orifice and causing hydronephrosis.

In cases of renal growth in which haematuria is absent or has ceased, ordinary cystoscopic examination does not give any information, as the vesical wall and ureteric orifices are normal. In some cases the elimination of indigo-carmine from the kidney, after an intravenous injection of 2 c.cm. of a 0.4 per cent. solution, is absent or delayed, but in cases in which the renal tissue is not much destroyed the dye may be present within normal time. Segregation of the urines by ureteric catheter may show a low urea content on the affected side, especially if urea has been administered some one and a half hours before the examination. Personally I carry out these tests in order to ascertain the functional activity of the presumed unaffected kidney rather than for diagnostic purposes. No reliance can be placed on the presence of blood in a urine drawn off by ureteric catheterization, as it may be due to the passage of the catheter.

Pyelography is of great value in these cases, and should always be carried out whenever any doubt exists. A plain *x*-ray may show an increased area of density in one renal area, but it is rarely to be relied upon. Pyelography may be carried out by intravenous injection of uroselectan B or by direct injection of the renal pelvis and calices by a solution of sodium bromide after ureteric catheterization, or it may be necessary to confirm or supplement the findings of the first method by the second. In a case of growth in the kidney there may be marked deformity or even absence of some of the calices, or of the pelvis of the kidney, depending upon the position and extent of the growth. No definite configuration can be laid down as there are very considerable variations, and in an early case there may only be elongation of one calix, with possibly a concavity of one aspect suggesting the rounded, bulging periphery of a tumour. In polycystic disease all the calices are elongated into spidery processes, and in one case in which I diagnosed a growth from pressure upon, and deformity of, a lower calix of an enlarged kidney, I found on operation the lower pole occupied by a large unilocular cyst.

In those cases in which tumour is the only symptom—as usually occurs in renal growths in children—there may be difficulty in diagnosis from tumours of the spleen, liver, or gall-bladder, or of the colon, but a pyelographic

INNOCENT NEW GROWTHS OF KIDNEY

	Initials	Sex	Age	Side	Initial Symptom	Operation	Histological Examination	Remarks
1	A. S.	M	19	L.	Profuse haema-turia	Nephrectomy	Cavernous angioma	Recovery

MALIGNANT NEW GROWTHS OF KIDNEY

	Initials	Sex	Age	Side	Initial Symptom	Operation	Histological Examination	Remarks
	SARCOMATA							
2	A. L.	M.	54	L.	P. and H.	Nephrectomy	Mixed-celled sarcoma	Recurrence in scar and in lungs in 8 months
3	J. D.	M.	71	L.	H.	,,	Leio-myosarcoma	Well after 12 months
4	E. Y.	M.	6 mos.	R.	T.	,,	Emb. adenosarcoma (Wilms)	Died 6 weeks later. Metastases in chest
5	J. R.	M.	6	—	T.	,,	Mixed-celled sarcoma	—
6	J. P.	M.	2	R.	T. no H.	,,	Emb. adenosarcoma (Wilms)	Well 2 years 7 months after
7	L. S.	F.	10	L.	T. no H.	None	—	Clinical diagnosis: sarcoma
8	J. C.	F.	2	L.	T.	,,	Adenosarcoma	P.M. specimen: Adherent colon; metastases in lung and portal fissure
9	T. A.	M.	55	L.	H.	Exploration and biopsy	Spindle-celled sarcoma	Capsule infiltrated; not removable
	CARCINOMATA:—1. Papillary Adenocarcinomata (Ewing)							
10	A. F.	M.	50	L.	H.	Nephrectomy	Papillary adeno-carcinoma with gran. cells	Well after 6 years
11	M. C.	F.	53	L.	T.	,,	,,	Died pneumonia 5 days after operation
12	C. N.	F.	55	R.	P.	None	Papillary adeno-carcinoma	P.M. Metastases in abdominal glands and lumbar spine
13	H. L.	M.	72	L.	H.	Nephrectomy	,,	Well for 7 years; died of influenza
14	Mrs. P.	F.	59	R.	H.	,,	,,	Died after 6 months. Metastases in lung
15	A. S.	M.	35	L.	H.	None	,,	P.M. specimen
	2. Alveolar Adenocarcinomata (Ewing)							
16	C. B.	M.	63	R.	F.	Exploration	Alveolar adenocarcinoma	Kidney contained 7 calculi and was distended with pus and soft growth. Died 6 hours a ter operation
17	M. S.	F.	58	R.	H.	Nephrectomy	,,	Cerebral embolism 5½ years later. No recurrence
18	M. I.	F.	48	L.	T.	,,	,,	Well 2½ years later
19	A. S.	M.	54	R.	H.	,,	,,	Well 1 year 7 months after operation
20	W. P.	M.	67	R.	H.	,,	,,	Well 6 months after
21	E. V.	M.	50	L.	H.	,,	,,	Recurrence in scar, iliac fossa, and lung. Died after 4 months
22	I. F.	M.	63	L.	P.	Exploration	,,	P.M. specimen. Metastases lumbar vertebrae
23	J. C.	M.	57	L.	P.	None	,,	Spontaneous fracture of femur. Later haematuria and death 3 years after fracture. Metastases in lung and heart
24	J. B.	M.	50	L.	H.	,,	,,	P.M. specimen
	3. Hypernephromata							
25	A. H.	M.	63	R.	P.	Nephrectomy	Hypernephroma	Died 13 days after operation. Nephritis
26	W. W.	M.	43	R.	H.	,,	,,	Adherent to diaphragm. Recurred
27	A. W.	M.	73	L.	H.	,,	,,	Growth in renal vein
28	M. B.	F.	52	L.	H.	,,	,,	Well after 10 years. Renal dilatation from pelvic pressure
29	J. S.	M	61	L.	H.	,,	,,	Well after 9 years
30	L. U.	M.	49	L.	H.	,,	,,	—
31	A. R.	F.	41	R.	H.	,,	,,	Growth in renal vein. Recurred abdominal glands
32	M. J.	F.	47	L.	H.	,,	,,	—
33	A. I.	F.	58	R.	P.	,,	,,	Hydronephrosis from pressure on pelvis
34	G. J.	M.	49	R.	P.	,,	,,	Died from shock after operation
35	R. C.	M.	49	L.	P.	,,	,,	Died after 1 month
36	Ford	M.	53	L.	T.	,,	,,	Growth in renal vein
37	J. S.	M.	51	L.	H.	,,	,,	Capsule infiltrated. Recurrence 8 months
38	J. T.	F.	68	R.	P.	,,	,,	—
39	C. J.	M.	57	L.	H.	,,	,,	Well after 9 years
40	J. H. T.	M.	59	L.	H.	,,	,,	Local recurrence. Died 1 year after
41	J. S.	M.	55	L.	H.	,,	,,	Well after 12 years
42	W. P. G.	M.	63	L.	H.	,,	,,	Well after 7 years
43	J. B.	M.	53	R.	H.	,,	,,	Died 2 years later; recurrence in liver
44	M. V.	F.	47	L.	H.	,,	,,	Died 1 year 9 months later; recurrence in lung
45	J. F. D.	M.	49	L.	T.	,,	,,	Died with abdominal recurrence 1 year later
46	Mrs. T.	F.	70	R.	H.	,,	,,	Alive 10 months after, but has nephritis
47	W. L.	M.	53	R.	H.	,,	,,	Only 3 months since operation
48	A. C.	F.	53	R.	T.	Exploration	No section	Too extensive to remove
49	M. M.	F.	60	L.	H.	None	,,	Clinical diagnosis
50	M. C.	F.	27	L.	P.	,,	Hypernephroma	P.M. specimen. Metastases cervical and abdominal glands
51	F. H.	M.	61	R.	H.	Exploration	No section	Adherent to liver and colon: irremovable
52	E. W.	F.	73	L.	H.	None	Hypernephroma	P.M. Metastases liver, thoracic glands, and vertebrae
53	C. C.	F.	59	L.	H.	Exploration	,,	Aortic glands involved
54	W. N.	M.	74	L.	H.	None	,,	P.M. specimen. Metastases in lung and liver
55	P.	F.	47	L.	P.	,,	,,	P.M. specimen. Deposit in pelvic bones
56	McL.	M.	75	—	—	,,	,,	Very early growth found on P.M. Death from cancer of tongue
57	A. C.	M.	62	L.	—	,,	,,	P.M. specimen. Death from cancer of rectum
58	E. T.	M.	53	R.	T.	Exploration	,,	Inoperable; metastases head of humerus
59	P. C.	M.	61	L.	T.	None	No section	Clinical diagnosis
60	A. G.	F.	40	L.	H.	Exploration	,,	Clinical diagnosis; too fixed to remove

NEW GROWTHS OF THE RENAL PELVIS

	Initials	Sex	Age	Side	Initial Symptom	Operation	Histological Examination	Remarks
	INNOCENT:—Papilloma of pelvis							
61	G. P.	M.	68	R.	H.	Nephrectomy	Papilloma, non-malignant	—
	MALIGNANT:—1. Villous-covered carcinoma of pelvis							
62	—	F.	53	R.	H.	„	Villous carcinoma of pelvis	Recurrence in lumbar glands 15 months later
63	A. C.	M.	58	—	H.	„	„ „ „	Recent case
	2. Epithelioma of Renal Pelvis							
64	E. J.	M.	54	R.	P.	„	Epithelioma involving middle calix and infiltrating kidney	Calculus in pelvis of kidney
65	G. W.	M.	64	L.	H.	„	Squamous-celled carcinoma	—

NOTE.—H. denotes haematuria. P. denotes pain. T. denotes tumour.

examination will usually determine a lesion in the kidney or prove it to be normal. In every case in which a carcinoma is suspected an *x*-ray examination should be made of the chest and of the skeleton to search for any metastatic deposit of the growth.

GROWTHS OF THE RENAL PELVIS

New growths arising in the renal pelvis are distinctly uncommon, and are very similar to the growths seen in the bladder. A villous papilloma of the renal pelvis may give rise to intermittent profuse haematuria, and from its position cause recurrent renal distension, with pain and a tumour of varying size in the loin. A significant feature of these growths is their tendency to multiplicity. Thus, small tumours may be present in the ureter or in the bladder, and small tufts of papillomatous growth have been seen to be extruded from the ureteric orifice upon cystoscopic examination. A more common form of growth in the renal pelvis is the villous-covered, or papillary, carcinoma, in which malignant infiltration occurs in the pelvic wall and also directly into the renal tissue. Squamous epithelioma also occurs, probably in cases of old-standing infection of the pelvis, and in the only case to which I can refer, a calculus was present in the pelvis of the kidney.

All these forms of growth may cause hydro- or haematonephrosis from obstruction to the pelvic outlet, and in the malignant forms spread to the lymphatic glands about the renal vessels seems to appear early. They all cause haematuria, but a diagnosis is rarely made before operation, though a pyelographic examination may show an absence of, or filling defect in, the renal pelvis.

TREATMENT

Nephrectomy, with removal of the perirenal fatty tissue and the lymphatic glands along the aorta and vena cava, is the only procedure which gives prospect of success, but before any operation is undertaken a careful examination must be made for metastatic spread in other organs and also of the functional capacity of the other kidney. Immobility of a kidney which is the seat of a growth implies that perirenal infiltration has occurred, and it must be remembered that the removal of a renal tumour may be a very difficult operation, trouble being frequently encountered from profuse bleeding from the dilated, thin-walled vessels covering the growths. The operation often commences as an exploration of the kidney for unilateral haematuria, when the renal fatty tissue is necessarily opened, but if a firm swelling is palpated in the kidney it is better to proceed immediately to nephrectomy and subsequent removal of the fatty tissue and glands than to incur the strong probability of early local recurrence by direct incision of the renal cortex. If a diagnosis of growth has been made the kidney should be removed together with the fatty capsule, lymphatic, and suprarenal glands in one mass. The question of operative route varies with individual surgeons, but the transperitoneal operation holds advantages for large tumours. A preliminary exploration will show the extent of glandular invasion, or may even show that the peritoneum is directly involved, but it has the additional advantage that haemorrhage may be more effectually controlled by early ligature of the renal vessels. Greater freedom is also found in separating the upper pole of the kidney, which may be so difficult in these cases where large veins are lacerated early in the operation by the lumbar route. Gregoire has described an extraperitoneal operation from an anterior incision which is very useful in these cases. The chief danger in the operation is haemorrhage from the dilated veins, whilst the renal vein may be easily lacerated if it contains growth. Injury to the vena cava can also occur, and may require lateral suture.

RESULTS

Of fifty-one cases of carcinoma operation was not advised in six cases owing to the extent of the disease or because of the presence of metastases. A total of thirty-nine underwent operations, but in seven the growth was found too extensive for removal. The remaining thirty-two had nephrectomy performed. Of these, five died as the result of the operation—two from shock, one from pneumonia, and two within a month of the operation, one death being due to nephritis. Ten have subsequently succumbed from recurrence of the disease at intervals varying from four months to over two years, the most common seat of recurrence being the abdominal glands, in the lungs, the liver, and the bones. The fate of four is unknown. Thirteen patients recovered from the operation, but of these two have subsequently died from intercurrent disease—one from acute post-influenzal pneumonia after seven years, and one from cerebral haemorrhage after five and a half years. Including these two cases, the interval since the operation has been: twelve years, 1 case; ten years, 1 case; nine years, two cases; seven years, 2 cases; six years, 1 case; five and a half years, 1 case; two and a half years, 1 case; under two years, 4 cases.

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FIG. 1.—Case 14. Papillary adenocarcinoma involving upper two-thirds of kidney with irregular infiltration into lower pole. Renal pelvis and vein invaded.

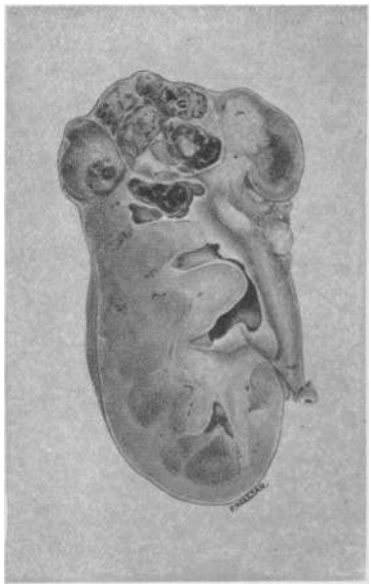

FIG. 2.—Case 29. Hypernephroma. Involvement of upper calyx by growth.

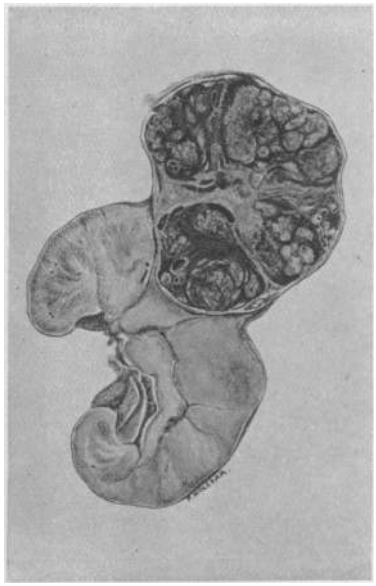

FIG. 3.—Case 39. Unusual type of hypernephroma forming rounded projection from kidney and surrounded by much fatty tissue. Growth appears to be encapsulated, but involves upper calyx. Renal tissue shows chronic interstitial nephritis.

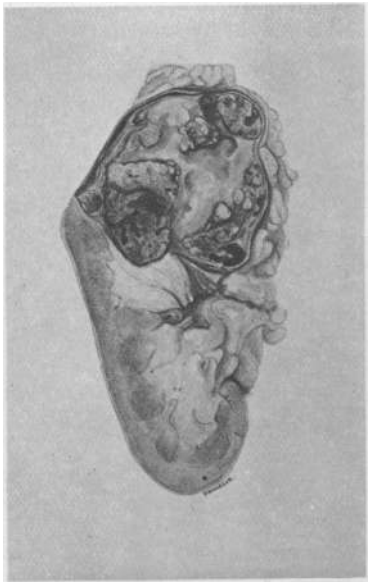

FIG. 4.—Case 41. Typical hypernephroma of upper pole of kidney invading renal pelvis. Area of necrosis and haemorrhage in growth.

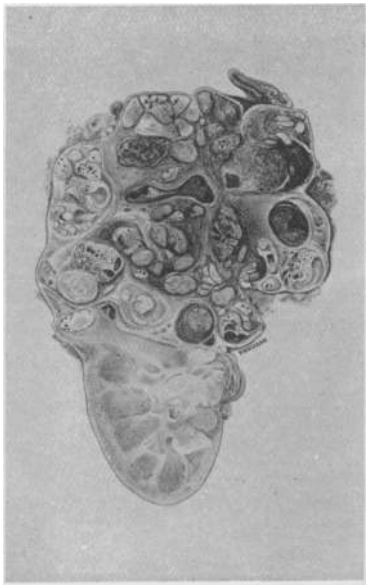

FIG. 5.—Case 42. Large hypernephroma of upper pole of kidney invading renal pelvis and showing partial encapsulation with areas of necrosis and haemorrhage.

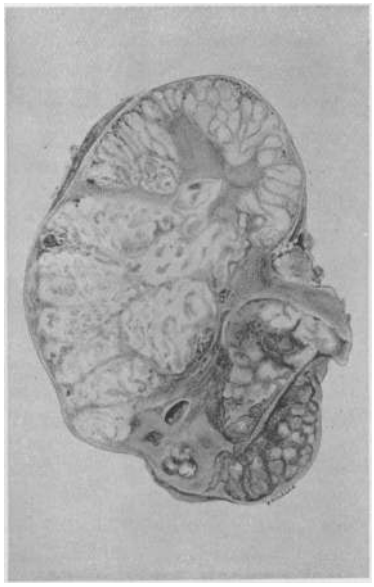

FIG 6.—Case 46. Hypernephroma invading practically the whole kidney.