this condition was mistaken for a dilatation of the abdominal aorta. In another case, as a result of congestion of the fused kidney, a compression was suddenly exerted upon the underlying vessels, which caused a thrombosis of the large veins; death resulted from a complete arrest of the circulation. Previous to this time, although the isthmus lay over the abdominal aorta and inferior vena cava, it was raised with each arterial pulsation, thus lessening the pressure on the vena cava.

The anomalous form and position of the kidneys may, under certain circumstances, be responsible for interference with the escape of urine, with a fatal termination. Such a case has been reported by Koster—the pregnant uterus pressed upon the displaced ureter causing a pyelonephritis.

B. von Langenbeck has seen children die suddenly with brain symptoms, probably uræmic, in whom a horseshoe kidney was found at autopsy.

These kidneys are usually found in abnormal positions, even in the true pelvis, lying in the cavity of the sacrum. Sometimes they are on one side and may present nodular, irregular masses in which inflammation and suppuration may occur.

In any case of a single kidney the possibility of it being a fused kidney must be considered. Bachhammer gives the following means of distinguishing between them. In the single kidney with two ureters, they open either individually, or after fusion, into the same side of the bladder. A fused kidney has two ureters which open into the bladder normally.

Most cases of crossed dystopia occur in males. It does not predispose to disease. In sixty per cent. of cases the right kidney was displaced. The frequency of the anomaly is very small. Morris noted its occurrence only once in 14,318 autopsies performed at different London hospitals. It is due primarily to a fusion of the two metanephric blastemata, the dystopia being a result of this. Each ureter has its own clientèle of pyramids.

(3) *Absence of One Kidney.*—We sometimes meet with cases in which one kidney is entirely absent. These must not be confounded with cases in which there is great congenital atrophy of the kidney, dependent on rudimentary development of renal vessels.

When the kidney is entirely absent, the corresponding ureter will be invariably absent. It is a rare thing for the ureter of a solitary kidney to cross over to the opposite side before it opens into the bladder. There is usually no disturbance in the renal function. The kidney is hypertrophied and thus takes upon itself the work of two kidneys. Following a nephrectomy the remaining kidney undergoes hypertrophy. If, however, it should become involved in disease, or the ureter should be obstructed by a calculus or compressed by a tumor, very alarming symptoms supervene. In twenty-four cases in which the cause of death was given, in twelve it was due to the anomaly; in ten it was due to the presence of renal calculi plugging the ureter, in two to pressure on the ureter by cancerous tumors. In operations upon the kidney the surgeon should make certain that his patient has more than one kidney. The fused kidney is most likely to deceive the surgeon; it is enlarged and displaced and may be mistaken for a new growth. In Polk's case the organ was removed under the belief that it was a floating kidney. The patient lived eleven days, had complete anuria, and it was found post mortem that the patient's only kidney had been removed.

The cystoscope has proved of great value in diagnosing the presence of two kidneys. It could not, however, exclude the fused kidney, since in this variety the two ureters might open into the bladder in a normal manner.

Ballowitz found in two hundred and five cases of absence of one kidney that in fifty-seven per cent. it was the left which was lacking.

(4) Congenital absence of both kidneys is an extremely rare condition, occurring only with extreme defect of development. It is, of course, incompatible with continued existence.

(5) In a few exceedingly rare cases of fused kidney,

the supernumerary renal parenchyma is not united to the two kidneys, so that the middle piece constitutes an independent kidney, which receives blood from both lateral parts, but also possesses in part independent vessels.

C. Variations in Pelves, Ureters, and Blood-Vessels.—When only a single kidney is present there may be one or two pelves and one or two ureters. These may open separately or they may fuse and have but a single opening. It is a rare thing for the ureter of a solitary kidney to cross over to the opposite side before it opens into the bladder. In the different varieties of fused kidneys, the veins and arteries are usually abnormal, both in number and in position. Branches frequently pass to the kidneys from one or both of the common iliac arteries. The kidneys on the anterior surface in the majority of cases, consequently the ureters pass in front of the kidneys. In the different varieties of congenitally dislocated kidneys the renal and neighboring vessels have anomalous origins, their number is usually increased, and they are shorter than normal. The ureters are also shortened and run an anomalous course. As an illustration of an unusual abnormality may be mentioned the persistence of Müller's duct.

Movable Kidney.—The floating or movable kidney, both congenital and acquired, is extremely common, especially in women. In a large percentage of the cases the right kidney is dislocated. Both kidneys are dislocated a little more frequently than the left alone. In the vast majority of cases it is simply part and parcel of a much more extensive set of displacements involving most of the abdominal viscera, stomach, intestines, spleen, liver and genital organs—so-called Glenard's disease, in which the symptom complex is that of neurasthenia combined with digestive disturbances. It is highly important for the clinician to keep this in mind when considering the question of operation. Before performing nephrorrhaphy he must be absolutely certain that the symptoms come chiefly from the dislocation of the kidney. There is usually a symptom complex, due to involvement of most of the abdominal viscera. An attempt to correct *one* of many displacements *may* have absolutely no effect toward correcting the symptoms of the patient. In rare cases the kidney may alone be dislocated, or it may alone be the cause of the symptoms. In such cases operative interference may entirely relieve the patient. This condition is extremely common, occurring especially in slender, poorly nourished, emaciated women. In many cases it is congenital, these patients having always been poorly nourished and having always possessed long, lax peritoneal attachments. It was formerly thought to be due chiefly to frequent child-bearing, and the resulting lax abdominal wall, with diminished intra-abdominal pressure. We now know that it is frequently found in young women who have never borne children. Rarely it occurs as a result of trauma and heavy lifting. It frequently comes on after prolonged wasting illnesses, such as the acute fevers and dyspepsias, thus depending upon the disappearance of fat from about the kidney. Consequently there is a medical side to the question. The patient should be brought up to a normal state of nutrition if possible. The musculature, especially that of the abdominal wall, should be developed. If necessary, in the severer grades special abdominal supporters may be used, in which the pressure is exerted from below upward. The degree of movability is varied. In many apparently normal women the lower border of the kidney, especially of the right, can be felt. This is termed the *palpable kidney.* The next degree is the *movable kidney.* On deep breathing the organ is forced down between the palpating hands, but returns during expiration. The next degree is the *floating kidney.* It is extremely movable, can be pushed into different parts of the abdomen, and is very little influenced by breathing. It assumes different positions with varied positions of the patient, may even reach the true pelvis, or the opposite side of the abdomen. Such a kidney as this is nearly always associated with a marked splanchnoptosis. If the symptoms of Dietl's crisis (*i.e.*, paroxysmal attacks

of renal pain) and hydronephrosis are present, and predominate over the general symptoms of splanchnoptosis, the kidney should be stitched into place. Dietl thought these symptoms were due to strangulation of the kidneys, or to twists or kinks in the renal vessels due to the extreme mobility. The association of floating kidney with dilated stomach has been emphasized by many authors. The combination of gastroptosis and nephroptosis is very much more common, but they are both parts of the same pathological condition. It cannot be said positively that nephroptosis bears a causal relation to dilatation of the stomach. *James Rae Arneill.*

KIDNEYS, DISEASES OF: AMYLOID DISEASE.—

(Synonym: Lardaceous or waxy kidney.)

History.—The names of Rokitansky, Meckel, and Virchow were intimately associated with the early history of this degeneration. In 1842 Rokitansky described the gross pathology of the lardaceous kidney, and spoke of it as a separate form of Bright's disease. Before this time it was considered a simple form of Bright's. Later, Meckel emphasized its various etiological factors. He established its iodine and sulphuric-acid reaction, but thought the substance was identical with cholesterin. Virchow pointed out the errors in Meckel's demonstration. He believed that this substance belonged to the cellulose group, because of its reactions with iodine, and gave to it the name amyloid, which has clung to it ever since, notwithstanding the fact that it was soon proved that the substance was of an albuminoid rather than a starchy nature.

Etiology.—It was early recognized that this degeneration was secondary to the various cachexias. Prolonged suppurative processes, especially of bones, are the most frequent causes of amyloid degeneration. As illustrations of other causes in this class may be mentioned: empyema, bronchiectasis, pyelitis, psoas abscess, etc. There seems to be a peculiar etiological connection between chronic ulcers of the feet and amyloid degeneration. Syphilis and chronic pulmonary tuberculosis are fairly common causes. Rarely cases have been found following carcinoma. As one would expect from its varied etiology, this degenerative process attacks many tissues. It may, however, localize itself more in one organ than in another. We then speak of amyloid disease of the liver, spleen, or *kidneys*, etc.

Morbid Anatomy.—The gross appearance of the kidneys depends upon the extent of the degeneration and the variety of the associated nephritis. In the early stage, when the vascular loops of only a portion of the Malpighian corpuscles are affected, the kidney may appear apparently normal. But as soon as the degeneration is at all extensive the kidney enlarges. The change is seen especially in the cortex, which, with its light yellow color, contrasts strongly with the red pyramids. When of long standing it has a homogeneous appearance. The enlarged Malpighian tufts appear, in the words of Meckel, like sparkling dew drops. If the pyramids become extensively involved, they are pale. The capsule strips off easily. The microscopical study is greatly facilitated by the peculiar reaction which amyloid tissue shows to iodine. A watery solution of iodine, tincture of iodine, or Lugol's solution (pure iodine 0.5 gm., potassium iodide 5 gm., and water 200 c.c.) may be used. If the degeneration is well advanced, the iodine solution may be poured over the cut section and the amyloid tissue, by virtue of the color reaction, recognized with the unaided eye. For more accurate microscopical work, fine sections from the fresh preparation, thoroughly washed with distilled water, are placed in one of the diluted solutions of iodine. The amyloid tissue strikes an iodine color (mahogany or ruby red) which appears to be due to a special affinity which this tissue has for iodine—a sort of absorbing process. The addition of sulphuric acid changes this color to a black. This was formerly supposed to be blue, and to indicate the presence of starch. We now know that the acid acts upon the reagent and not upon the tissue. The amyloid

change is intertubular and almost entirely vascular. The Malpighian vessels first show the characteristic reaction, the capsule remaining unaffected to the last. The straight vessels of the cones are next attacked. The interstitial tissue apart from the blood-vessels is usually unaffected. The epithelium does not participate in this change. The iodine reaction has been noted by Dickinson in casts within the tubes.

Symptoms and Signs.—There are no pathognomonic signs of this condition. Osler states that it is simply an event in the process of chronic Bright's disease, more commonly of the amyloid variety. The presence of albumin in the urine, and of œdema or diarrhœa, is very suggestive, especially if combined with the presence or a history of prolonged suppuration, syphilis, or tuberculosis. These patients are usually pale, emaciated, and cachectic. This degeneration may occasionally be found in well-nourished syphilitics. They are almost always anæmic, however. The detection of an enlarged liver or spleen not otherwise explained is extremely suggestive. Diarrhœa is a frequent and important symptom, and in some instances is due to involvement of the intestine by the lardaceous process. If persistent, it has much to do with the fatal issue. Dropsy, according to some authorities, is a fairly common sign, but is frequently absent. The variety of nephritis, associated with the amyloid degeneration, is of necessity after the sign. Dickinson states that apoplexy and albuminuric retinal changes are uncommon in amyloid disease, because of the absence of increased arterial tension and cardiac hypertrophy.

The urine shows important changes, but from these alone a diagnosis of this degeneration cannot be positively made. The quantity is usually increased, varying according to Stewart from 1,080 to 6,000 c.c. in the twenty-four hours. It is pale, clear, and of low specific gravity.—from 1.006 to 1.017. Albumin is usually present and often abundant. The sediment as a rule is scanty. Hyaline, granular, or fatty casts may be found. The amyloid reaction has been noted in the casts, but is rare. Intercurrent affections, such as pneumonia, pleurisy, pericarditis, and peritonitis, may occur, but are not so common as in the parenchymatous or interstitial forms of nephritis.

The duration of the disease is extremely variable, depending largely upon the associated nephritis. It may be very acute or prolonged indefinitely, as in certain syphilitic cases. It is difficult to tell at just what time in the course of the disease the amyloid degeneration begins.

Treatment.—An attempt should be made to remove the causative disease—suppuration, syphilis, etc.—before the development of the amyloid degeneration. If it is too late for this prophylactic measure, the treatment is that of the associated nephritis, together with attention to the causative disease, if still present. If this is syphilitic in origin the iodides should be administered. Grainger Stewart claims to have seen a case almost cured under the use of the iodide of iron. *James Rae Arneill.*

KIDNEYS, DISEASES OF: CONGESTION.—

Congestion or hyperæmia of the kidneys may be acute or chronic, active or passive.

Active congestion of the kidneys is a condition of acute or active engorgement of the blood-vessels of the kidneys unassociated with exudation. In most cases it represents the earliest stage of acute nephritis; in other cases it is part and parcel of this disease; and inasmuch as it is extremely rare for acute congestion of the kidney to exist without at least a slight amount of exudation, there can be no sharp dividing line between such acute congestion and acute nephritis. Indeed, the typical examples of "acute congestion" of the kidneys encountered after death are almost exclusively examples of acute Bright's disease. In the great majority of cases, therefore, the etiological factors of active congestion of the kidneys are the etiological factors of acute nephritis (*vide infra*). However, it is likely that in some cases congestion

may exist alone—*e.g.*, in conditions of mild toxæmia, such as occur in certain infective diseases; in mild poisonings by certain drugs and chemicals, such as cantharides, copaiba, turpentine, arsenic, carbolic acid, etc.; after the use of ether for anæsthesia, etc. It has been attributed also to sudden contraction of the blood-vessels of the periphery of the body following exposure to cold, and to loss of vaso-motor control. It probably occurs also in conditions of increased functional activity, such, for instance, as occurs in one kidney after extirpation of its fellow, and when one kidney has been rendered functionless by the blocking of its ureter with a calculus, etc. In acute congestion the kidney is enlarged, somewhat softened, and deep red in color, or reddish-brown and mottled; its capsule strips readily and reveals distended and congested vessels, and on section the organ drips blood.

In general, symptoms are wanting; at most, they are indefinite. In cases with definite symptoms there is more or less disturbance of the renal epithelium, and the condition, except in that it disappears with removal of the cause, is scarcely to be distinguished from acute toxic or degenerative nephritis, or the mildest grades of acute diffuse nephritis (*vide infra*).

Passive congestion (mechanical congestion) of the kidneys occurs most frequently in conditions of general venous stasis, and less frequently in cases of more or less obstruction to the return circulation from the renal veins, such, for instance, as may result from pressure on the renal vein or veins by a tumor, aortic aneurism, pregnant uterus, ascites, deformities of the vertebræ (causing angulation of the renal vein), and in cases of thrombosis of the ascending vena cava and renal vein or veins. In most cases it is associated with failing cardiac compensation due to valvular disease of the heart, fibroid heart, adherent pericardium, emphysema, interstitial pneumonitis, chronic adhesive pleuritis and mediastinitis, extensive tuberculosis of the lungs, etc. It occurs also in cases of cirrhosis of the liver.

The kidney the seat of passive congestion (cyanotic induration, cardiac kidney) is enlarged and much firmer than normally; the capsule strips readily and reveals distended veins, and usually a smooth surface that is dark bluish-red in color. On section the cortex is increased in thickness, dark bluish-red in color, and often distinctly striated. The pyramids usually are still darker than the cortex, being purplish. After the congestion has persisted for some time the kidney may become reduced in size, may be slightly adherent, and the surface slightly granular (the congested and contracted kidney). Microscopically, in the early stages there is marked dilatation of the veins and capillaries. The parenchyma may be normal, though usually, especially if the process has lasted for some time, the epithelium reveals more or less degeneration—parenchymatous and fatty. In the early stages the interstitial connective tissue reveals no noteworthy changes. After the process has persisted for some time, however, with gradual destruction of the parenchyma, the connective tissue becomes hyperplastic and subsequently contracts. In reality, the condition is a diffuse nephritis.

Clinically, passive congestion of the kidneys manifests itself by lessening in the amount of urine (due to diminution in the arterial pressure and consequent retardation of the circulation of the blood)—the daily amount in some cases being less than 200 c.c. In addition, the urine is dark in color, turbid, of increased specific gravity (1.028–1.035), and usually it deposits a considerable sediment of uric acid and urates. It always contains a small amount of albumin—the amount depending upon the functional activity of the heart and the degree of associated changes in the renal epithelium, but scarcely, if ever, exceeding one-tenth or one-eighth by bulk (after the heat-and-acid test). In uncomplicated cases the sediment contains only a few hyaline casts, maybe no casts. In many cases a few epithelial or granular casts, some renal epithelium, a few leucocytes, and erythrocytes

may be encountered; but these are referable to some complication, such as concomitant nephritis, congestive hemorrhages, hemorrhagic infarcts, etc. In uncomplicated passive congestion of the kidneys uræmia is practically unknown.

The *diagnosis* of passive congestion of the kidneys usually is a matter of comparative ease. The symptoms of the primary disorder are always of much importance. Occurring then as part of a general venous congestion in failure of the circulation, congestion of the kidneys is readily recognized. Especial diagnostic value attaches to the association of the urinary changes with enlargement of the liver and spleen, dropsy, and cyanosis. The cyanosis and dropsy contrast strongly with the pallor and dropsy of Bright's disease. Further, the amount of urine voided daily, as well as the amount of the albuminuria, varies from day to day with the functional activity of the heart. The urine then furnishes trustworthy evidence of the condition of the venous circulation—whether it is free or more or less impeded. In some cases with marked venous congestion difficulty may be encountered in differentiating the condition from a condition primarily renal and secondarily cardiac (*vide* Chronic Interstitial Nephritis).

The *prognosis* depends wholly upon the primary disorder and upon whether the lesions of diffuse nephritis have developed. In uncomplicated cases complete restitution of the kidney's may occur.

The *treatment* is the treatment of the primary disorder. In most cases then it is the treatment of failing cardiac compensation—rest, a nutritious and readily assimilable diet, cardiac tonics and diuretics, especially digitalis, strychnine, caffeine, alkaline diuretics, sparteine, theobromine, diuretin, etc.
Aloysius O. J. Kelly.

KIDNEYS, DISEASES OF: NEOPLASMS. — Morbid growths of the kidney occur more frequently than is generally supposed. Clinically, any enlargement of the kidney which we detect by physical examination is spoken of as a tumor; several of these enlargements have already been considered, namely, hydro- and pyonephrosis, perinephritic and nephritic abscesses, tuberculous enlargements, etc.

Other tumors are now to be considered, and for purposes of description they may be taken up in one of two ways, or in a combined way: I. As to histological structure, or II. As to position of the growth with reference to the kidney.

I. Varieties as to pathological structure:
(*a*) Benign.
   Adenoma.
   Lipoma.
   Fibroma.
   Angioma.
   Myoma.
   Leukæmic tumors.
   Villous papilloma.
   Accessory adrenal (struma suprarenalis of Grawitz, often malignant).
   Osteoma.
   Enchondroma.
(*b*) Malignant.
   Adenoma.
   Sarcoma.
   Carcinoma.
   Lymphadenoma.
   Accessory adrenal (Grawitz).

Cysts. { Hydatid.
        { Dermoid.
        { Polycystic degeneration.
        { Simple or serous cysts.

A pure pathologico-anatomical division can be made which has as its basis the various tissues which form the foundations of the tumors:

1. Those arising from the preformed connective tissue: Fibroma and lipoma; osteoma and enchondroma; angioma and lymphangioma; sarcoma, angiosarcoma, endothelioma, and perithelioma and their mixed forms.

2. Those arising from the preformed epithelium: Adenoma, cystadenoma, carcinoma, adenocarcinoma.

3. Those arising from cells of a different kind from those in the vicinity of the tumor: Accessory adrenal (hypernephroma, rhabdomyoma).

4. Those arising from retention tumors: Polycystic degeneration, solitary cysts.

5. Those arising from parasitic cysts: Hydatids.

This division, however, is not a practical surgical one, as benign and malignant tumors are classified together; consequently we will adhere to the old useful division into benign and malignant.

II. Division with reference to the position of the growth in the kidney itself:
(*a*) New growths of the parenchyma.
(*b*) New growths of the calyces or pelvis.
(*c*) New growths of the renal capsule.

A. NEW GROWTHS OF THE PARENCHYMA OF THE KIDNEY.—These are overwhelmingly more frequent than those arising from the pelvis or capsule of the kidney. Out of 70 cases of kidney tumors observed by Israel only 2 arose from the pelvic mucous membrane, while 68 were situated in the renal parenchyma.

Of the relative frequency of the different forms, the following statement is quoted from Morris' "Surgery": of 154 cases collected from various sources there were: Sarcomata, 68; carcinomata, 41; cystic degeneration, 21; hydatid cysts, 11; adenomata, 10; papillomata, 3; myxomata, 2; lipomata, 2; dermoid cyst, 1; total, 154.

In 1,000 autopsies at the Presbyterian Hospital, New York, there were found in the kidney: Leukæmic tumors, 1; cystic kidney, 2; adenoma of kidney, 3; cancer of kidney, 7.

BENIGN TUMORS OF THE PARENCHYMA.—These are very rare, and form scarcely six per cent. (Morris) of kidney tumors. Aldibert, out of 51 collected cases, found only 3 benign. They are of more pathological interest than surgical because they, as a rule, reach only very moderate size and give few if any symptoms. This is particularly true of angioma, lymphangioma, osteoma, and enchondroma. Lipoma, fibroma, adenoma, and their mixed forms have occasionally reached large dimensions and become the object of surgical treatment.

Lipoma.—True lipomata are rare. They must be differentiated from lipomata which arise in the substance of, or outside, the capsule, as well as from fibro-fatty changes seen as the result of calculous inflammation, pyelitis, retention of urine, etc. Some of the so-called lipomata have been shown by Grawitz, Lubarsch, and others to be small subcapsular inclusions of aberrant adrenal tissue, or of fatty tissue mixed with muscular fibres. Malignant growths may undergo fatty transformations. Usually the tumors are of mixed forms, fibro- and myxolipomata, and are often found intermingled with smooth muscle fibres.

The true lipomata are small, rounded tumors, seldom larger than a cherry, situated usually immediately under the capsule and not in the region of the hilum where fat is normally present. They may be single or multiple; when they are numerous, although each growth may be small, the volume of the kidney may be much increased. They take their origin in most part from included, scattered portions of the fatty capsule, and from the fatty transformation of connective tissue or fibroblastic cells, the "heteroplastic lipomata" (as Virchow named them, because there is no true fatty tissue normally present in the renal parenchyma.

Fibroma.—True fibromata are very rare indeed. They are found usually as small whitish nodules near the bases of the pyramids, and, on a few occasions, a very large, simple, fibrous tumor has been met with in the kidney. Such tumors have been operated on and reported by Dickinson, Bristowe, Bruntzel, Peaslee, Wahl and Bardenheuer, and others. Most of the large tumors have undergone simple softening, resulting in extensive cystic transformation; in other instances they contain muscular tissue, or are myxofibromata. Small fibrous tumors of the size of a pea, or even smaller, are not very

rare and are said by Ebstein and Virchow to be formed in connection with diffuse interstitial nephritis, many of them resembling adenomata.

Adenoma.—These are usually whitish or yellowish nodules of the size of a pea, single or multiple, situated in the cortex of the kidney; they are usually encapsulated, although not invariably, and they occur in adult age. They are more frequently found in kidneys the seat of interstitial inflammation, but some regard these little bodies as due to glandular proliferation rather than as true new growths. Larger tumors may occur, from the size of a cherry to that of an orange. They may be situated just beneath the capsule, or buried in the parenchyma; they may be divided into distinct lobules by whitish bands, and surrounded by a distinct capsule. Within these there may be hemorrhagic extravasations, or there may be simple softenings and cystic formations of large or small size. These cystic forms of adenomata are especially prone to develop papillary ingrowths within them, either sessile or pedunculated.

Delafield, Ricker, Klebs, Ziegler, Thoma, and many others emphasize the fact that adenomata may develop into malignant tumors. Sabourin has divided these tumors into two groups according as the tubules are lined by (1) cuboidal or by (2) cylindrical cells. There is also a division by Weichselbaum and Greenish into (1) papillary and (2) alveolar adenomata. Both these classifications, however, seem unnecessary, because we see side by side in many of the same tumors cuboidal and cylindrical cells, as well as the papillary and alveolar forms; so that the varieties under each of these two classifications represent so new formations but are simply various stages of the same developmental processes, and are closely related to each other. The pathogenesis of these tumors is obscure. Some are undoubtedly of accessory adrenal origin, others arise from remnants of the Wolffian body, others still from isolated elements due to an error in development, etc.

Angioma.—True angioma is very rare. In structure it is practically the same as the cavernous angiomata, or erectile tumors, of the liver and parotid. It may or may not be encapsulated and is usually of a bright red color. It varies in size from a cherry pit to a walnut.

Myxoma.—Bezold and Hollen have each described a case of pure myxoma. Myxomatous changes occur in tumors of other kinds, and myxosarcoma sometimes occurs in the kidney.

Osteoma and Chondroma.—Roberts states that a fibrous tumor growing in the kidney may ossify, while the fibrous capsule of the kidney may also undergo ossification. Undoubtedly many of the so-called bony growths are in reality calcified inflammatory products, such as we find in pyelitis and pyonephrosis and in the transformation of hydatid cysts.

Cartilaginous growths are still more rare, and it is a question whether they ever occur.

Leukæmic tumors are small, scattered, roundish patches of lymphoid or leucocyte cells, which follow the course of the capillary vessels; they stuff the capillaries and transude into the surrounding tissues and give rise to hemorrhages. There is very little structure, the stroma consisting of coagulated fibrin and the normal connective tissue, the so-called growths being little more than collections of leucocytes. These tumors at times actively grow, so as to take on a truly malignant character.

Hypernephromas, which are tumors of an entirely benign character, have already been discussed in Vol. IV. Cysts are to be considered farther on in the present article.

MALIGNANT TUMORS.—These include some forms of adenoma, carcinoma, sarcoma (and myosarcoma or rhabdomyoma), and some forms of struma suprarenalis. Cancer and sarcoma are more frequently secondary than primary in the kidney, following cancer of testis, liver, stomach, pancreas, uterus, and breast. Usually both kidneys are affected as the result of a secondary infection, while as a primary disease only one kidney is first

affected, although the second may subsequently become diseased through metastatic deposits.

In 3,926 autopsies at the Middlesex Hospital, 69 cases of malignant disease of the kidney were found. Of these 54 were secondary and 15 primary new growths, and of the latter, 13 were carcinomata and 2 adenomata (Morris). In the Zurich Clinic of Dr. Krönlein in the past twenty years there have been 118 surgically diseased kidneys of various kinds. Among these there were 15 malignant tumors, 2 cases of cystic tumors, and 1 echinococcus cyst. Out of 184 operations in Schede's clinic, there were 18 cases of malignant tumors, i.e., 8 carcinomata, 5 sarcomata, 2 strumæ, 3 adenomata.

Formerly all malignant tumors of the kidneys were thought to be cancer. Extended observations have now taught us that this is erroneous.

Schede's statistics of 312 cases of malignant tumors of the kidneys, taken from the literature, give the following as to histological structure: 153 sarcomata, 112 carcinomata, 28 strumæ suprarenales, 8 perirenal sarcomata, 6 mixed tumors, 4 malignant tumors of the kidney pelves, 1 malignant degenerated teratoma.

Age.—Von Bergmann has given two periods of life which are most predisposed to develop malignant tumors, namely: 1. The earliest years of life, i.e., under five years. 2. The period between forty and fifty years and thereafter.

Schede has collected out of the literature 329 nephrectomies for malignant disease. Of these 117 were found in children under fifteen years of age, and the great majority of these were in the first year of life. Out of 148 collected by Morris 39 occurred under five years of age, 2 between the ages of five and ten; 6 between ten and twenty; 14 between twenty and thirty; 20 between thirty and forty; 30 between forty and fifty; 22 between fifty and sixty; 11 between sixty and seventy; 4 between seventy and eighty.

Israel's statistics concern 68 cases—of these, 61 cases, or 89.7 per cent., occurred between the fiftieth and eightieth years, 3 occurred under ten years of age. Israel remarks that it is also of theoretical as well as of practical interest that malignant kidney tumors show a strict contrast to kidney tuberculosis with regard to the predisposing age in life, as in tuberculosis 81 per cent. of all cases occur between the ages of thirty and fifty, while in kidney tumors only 1.4 per cent. occurred in the corresponding period of life.

Brodeur states that of 18 cases of cancer, 17 occurred in adults, 1 in a child; that of 27 cases of sarcoma, 15 occurred in adults, 10 in children, and, finally, that 80 per cent. of sarcomata in children appear during the first four years of life.

Sex.—In Israel's 68 cases, 45 (66.17 per cent.) occurred in men, and 23 (or 33.83 per cent.) in women.

Guillet's statistics of 99 cases give twice as great a frequency among men as among women. According to Israel's experience, there were twice as many inoperable tumors, on the first examination, among women (11 cases or 48 per cent.) as among men (9 cases or 20 per cent.). This is explained by the fact that the two symptoms in general which lead a person with a kidney tumor to the surgeon are (1) a tumor, and (2) hæmaturia. 1. A growing tumor causes less discomfort in women, particularly those who have borne children, than in men, because the former have more relaxed abdomens, and consequently a growing tumor gives less discomfort to them than to men. 2. Then, as regards the hæmaturia, it must be remembered that blood clots pass through a woman's urinary tract more easily than through a man's, while at the same time blood in the urine is often looked on as a menstrual anomaly. A man, on the other hand, is at once frightened by the appearance of blood in the urine, and hæmaturia is apt to cause a greater degree of discomfort.

PATHOLOGY.—*Adenomata.*—It was pointed out in 1875 by Sturm that renal cancer may take the form of adenoma, consisting of a true proliferation of convoluted tubules, which ultimately may be transformed into cancer. Grawitz, Sudeck, Sabourin, and Oettinger have reported cases of adenoma of the kidney followed by secondary deposits of like nature in the lung. Pilliet, Sottas, and Albarran all agree that certain renal adenomata behave like malignant tumors, both in their local and in their distant effects, i.e., metastases, and that distinction between adenoma and carcinoma is sometimes most difficult; that it is impossible, either from the microscopical or from the macroscopical characters, to establish precise limits between adenoma and epithelioma; and, further, that an adenoma is sometimes only the beginning of an epithelioma. Adenomata are spheroidal in shape, encapsuled, often of considerable size, forming a prominence on the outer surface of the kidney as large as an orange, or larger. There may be many cysts, and also hemorrhages seen on section. The capsule is often thick and fibrous, and sends processes across the tumor. The stroma is frequently very scanty.

Carcinomata.—Primary cancer of the kidney is more common on the right side than on the left. In Morris' collection of cases from English literature (1884 to 1893) it was in the proportion of 10 to 6, and, in American journals during the same period, 4 to 3. Guillet's cases showed a proportion of 7 on the right side to 3 on the left. It is more frequently met with in men than in women; as 13 to 3 in the English cases; in the American, on the other hand, as 2 to 5. Guillet's 99 cases show 64 men to 35 women. (Taken from Morris.)

Primary cancer occurs in the kidney in two forms: 1. The nodular, with or without a capsule. 2. The diffused.

According to the preponderance of cell elements over the connective tissue, or vice versa, we have all varieties of cancer, from the hard scirrhus forms to the softest medullary cancers.

The growth destroys a more or less considerable part of the organ, although there is usually left some portion of the renal parenchyma. The fibrous capsule of the kidney usually covers the surface of the tumor. On section there may be a regular outlined edge to the growth, frequently surrounded by its own capsule; or, when the growth infiltrates the tissue, this independent capsule will be absent, in which case the new growth may have no regular outline. In some cases there may be two or more encapsulated masses in the kidney. The cancer may increase the volume of the kidney without altering its shape, or the kidney may be much deformed. The fibrous capsule may rupture at one or more points. The growth may extend along the pelvis and ureter. The size of the growths varies from that of a nut to tumors larger than an adult head. They do not usually reach such large sizes as the sarcomata. There may be one or more cysts in the growths, with blood extravasations or calcareous deposits within them.

The nodular variety forms the larger number of cancerous tumors. The diffuse or infiltrating form shows its character in its disposition to replace more or less of the renal tissue, spreading itself in such a way that at the time when a tumor is demonstrable the perirenal tissues, and the lymph glands are infected, as well as the neighboring structures, such as the peritoneum, aorta, or vena cava. For these reasons the diffuse form can seldom be the object of surgical interference, while, on the other hand, the nodular form is the one which has been the more frequent subject of operation, because it only very late infiltrates or breaks through the capsule propria, and may reach a very large size before infection of the lymph glands takes place.

Sarcomata.—Simple sarcomata furnish few peculiarities. There are spindle-celled, large and small round-celled tumors, which occur as single or multiple growths. Occasionally there is a formation of giant cells. Mixed forms, as fibro-, myxo-, and chondrosarcomata occur as elsewhere, and the malignancy varies very much according to the richness of the cells, as we find in some cases hard, relatively benign forms, while in others we meet with soft, medullary, very malignant types. There may be hemorrhagic extravasations and cystic formations. Sarcomata may infiltrate the whole kidney without changing its form. Usually, however, the sarcoma forms a tumor which develops at one point, and rapidly destroys the rest of the organ. At its outset the sarcoma appears, in certain cases, separated by a capsule from the kidney, but often there is no capsule; and where it exists the capsule often bursts at some small point, and the tumor infiltrates, through this point of rupture, the kidney tissue. The sarcomata may form enormous tumors which fill more than half the abdomen. They are lobulated and of uneven consistency, being firm in some places and soft in others. They reach especially large size in early childhood, in most cases proving fatal before the fifth year. They usually rapidly invade other tissues, both far and near. In a small proportion of the cases sarcomata are bilateral. Sarcomata occur at any period of life. Melanotic sarcomata are almost invariably secondary in the kidney.

The round-celled vascular variety is soft and grows rapidly. It is the most common form of renal new growth, constituting twenty-three out of forty-three tumors in children operated upon since 1890. The spindle-celled form is firm, much rarer than the round-celled, of slower growth, and of less marked malignancy. Myxosarcoma rapidly attains a large size without much pain and often without metastasis. It occurred in five cases out of forty-three sarcomata operated upon in children since 1890. Angiosarcomata represent a class of growths which have very lately received a great deal of attention because they owe their origin to the cellular elements of the blood and lymph vessels, i.e., they are endotheliomata. We give the views of Manasse, to whom we are indebted for an exhaustive work on this subject, although there is no great unanimity of opinion as to these growths. He divides angiosarcomata into: 1. Blood-vessel endotheliomata; 2. Lymph-vessel endotheliomata, and 3. Perivascular sarcomata. As to the histology: 1. The blood-vessel endotheliomata. These are not nearly so frequently seen as the lymph-vessel endotheliomata, and are rare. They proceed from the capillaries and from the small veins by fine sprouts which end blindly and are filled with blood to the very end of the vessel, which may be drawn out into a fine point, represented by an endothelial cell. The capillaries and veins are much dilated and their endothelial cells multiply very greatly, projecting into the lumina which may become obliterated. As the lumina become obliterated, there is formed a tangle of strings made up of double rows of cells which have no order in their relation to each other, and are without any connective tissue. Hyaline degenerations and necroses take place, forming great or small cysts which may have papillary projections from their walls.

2. Lymph-vessel Endotheliomata are more frequent than the above, and are characterized by their clear-cut formation. They show a connective-tissue stroma, through which are drawn regularly branching, net-like strings of cells, which correspond to the lymph vessels filled with tumor cells. Now and then one finds typical, isolated lymph vessels which are likewise filled with grown epithelial cells, and which one can easily recognize as lymph vessels by the sinus-like dilatations, by their valves, and by their position with relation to the arteries. The lymph spaces are also commonly filled with tumor cells. The proof that these growths are not carcinomata but truly consist of proliferated endothelium can be conclusively shown only by examining the youngest forms of the new growth. Here one sees in a lymph vessel the normal endothelial coating gradually becoming transformed into the cells which we see in the remainder of the tumor. The endothelial cells become continually thicker till they finally liken themselves to epithelial cells. In carcinoma one is in the position to demonstrate, outside of the epithelial filling-up masses in the lymph vessels, the normal coating of epithelial cells still present. Such an occurrence of tumor cells and endothelial cells beside each other is impossible. In the later stage of these endotheliomata there may be an alveolar arrangement, and great and small cavities may arise, corresponding to lymph cysts. From these walls papillary outgrowths may project. The differential diagnosis from cancer may be very difficult—the net-like arrangement of cell strings, the facts that the tumor cells have no similarity to the epithelium of the uriniferous tubules and that they do not lie together in such unformed cell trabeculæ as in the carcinomata, will render it possible, in most cases, for the observer to make the right diagnosis.

3. Perivascular Sarcomata, or Peritheliomata. In these also the characteristic arrangement is the net-like disposition of the tumor cells. The trabeculæ of the net are formed of thin-walled, partly blood-containing, partly obliterated vessels, which in a very characteristic way upon their outer side are covered by a mantle of cells which at times consist of a simple double row of cells, at other times of several layers. These cells have again either the character of small endothelial-like, spindle-shaped cells, or more that of a cylindrical or club shape. Through the fact that this vascular network, with its cellular covering, is not embedded in a basement tissue but makes up the tumor mass, there is furnished to us an explanation of the decided spongy appearance which it presents. As now the adventitia cells further proliferate, the spaces below the trabeculæ of the network gradually become more or less filled up. One sees then isolated cells which may retain the character of the adventitia cells, or which may become changed to an almost epithelial-like character, giving to the tumor an alveolar, almost carcinomatous appearance. In older portions of the tumor the picture is more complicated. In place of the delicate blood-vessels with their cell mantles, which formed the trabeculæ of the network, we find broad processes, of light-colored, sometimes hyaline connective tissue, poor in nuclei, which in many case is covered on its outer side by the already described cells; while the meshes are empty, or are partly filled with cells. Occasionally we find in the midst of these connective-tissue trabeculæ double rows of endothelial-like cells, which correspond to obliterated blood-vessels. Besides hyaline degeneration in the tumor, there may occur amyloid degeneration, and rich amounts of glycogen may be made out in the tumors. The perivascular sarcomata occur sometimes pure, often partly mixed with other forms of sarcomata.

Embryonal Adenosarcoma.—While the so-called hypernephromata (struma suprarenalis), which arise from included adrenal remains and consequently rest on anomalies in congenital formation, develop first in the later years of life, at the same time there are other forms of tumors in the kidney which also take their origin from failures in development, which are characterized by a very rapid growth and marked malignancy, and which are observed at times not only in the new-born, but also in the first four years of life. They have been described partly as sarcomata, partly as carcinomata, partly as rhabdomyosarcomata, without corresponding clearly to the types of carcinomata or sarcomata. They consist of a proliferation of the elements of embryonal tissue—i.e., there is an increase of the glandular elements as well as a sarcomatous proliferation proceeding from the connective tissue. The relative quantities of these two elements are very different in different tumors or parts of the same tumor, i.e., sections taken from one part of a tumor may show adenoma or carcinoma, those from another part spindle-cell sarcoma, and those from still another part may be transition forms from adenomatous to sarcomatous tissues. The greater part of these tumors represents a combination of epithelial growth of adenomatous arrangement with varying degrees of development of smooth and striated muscle fibres in the stroma. Many authors consider it unwarranted to attempt a systematic division according as to whether the one or the other element preponderates. It seems better, instead of classifying the greater part of these malignant tumors of early childhood as carcinoma, adenocarcinoma, sarcomatous carcinoma, adenosarcoma, adenochondrosarcoma, and myosarcoma, to put them into one single group, and to designate them as the embryonal or sarcomatous adenoma of the kidney re-