slight flocculent sediment that contains a few, sometimes a very few (often very narrow) hyaline and pale granular casts, and a few epithelial cells, mostly from the bladder. In many specimens of the urine the casts are so few that it is only after centrifugalization that several very pale hyaline casts may be detected. In some cases a few erythrocytes may be found. The solids of the urine are usually diminished.

The lesions of the cardio-vascular system characteristic of chronic nephritis consist of hypertrophy of the heart, especially of the left ventricle, and of increased arterial tension. The hypertrophy of the heart is sometimes obscured by coexisting emphysema of the lungs, but usually it may be recognized by the increased force and displacement downward and outward of the apex beat, extension of the cardiac dulness downward and to the left, and accentuation of the aortic second sound. Increased arterial tension, which may be expected from the beginning of the disease, is one of the most valuable signs of chronic indurative nephritis. The pulse of high tension is quite characteristic—it is hard and resisting to the palpating fingers, it remains persistently full between the heart beats (this being the important feature of the pulse), and it requires considerable pressure with the fingers completely to arrest the pulsations. The sphygmographic tracings are also quite characteristic. While this hypertrophy of the heart and increased arterial tension may be found in all forms of Bright's disease (but not in all cases), in none of the forms do they so dominate the clinical picture as they do in the primarily contracted kidney—primary chronic interstitial nephritis. They may be expected from the time of the onset of the contraction or atrophy of the kidney, be this of the primary or of the secondary variety. Thus they develop early in the primary form, but they may be (but usually are not) much delayed in the secondary form. They may develop also in the chronic diffuse non-indurative nephritis, and they (more especially the increased arterial tension) have been observed in a considerable number of cases of acute nephritis, more especially in the acute nephritis following infections, such as scarlet fever, etc. In a few chronic cases they are not observed. Their absence may be due to faulty nutrition—the heart not being sufficiently nourished to enable it to hypertrophy; to sclerosis of the coronary arteries; to the faithful performance by the skin and the intestine of their vicarious action whereby they remove those excrementitious products ordinarily eliminated by the kidneys, etc.

The existence of the secardio-vascular changes, as also the kidney lesions, may for a long time remain entirely unknown to the patient and be absolutely unsuspected by the physician. Whatever be their exact mode of development (and many theories have been advanced, but as yet the correctness of none has been conclusively demonstrated), it is certain that they arise in response to excessive demands on the part of the general economy. So long as the heart continues to perform its increased work well no symptoms occur. Should, however, the increased quantity of urine or the development of slight swelling about the eyes or the ankles attract the patient's attention; or should he suffer from epistaxis (usually of serious moment), or from dimness of vision (due to neuro-retinitis), or from severe muscular cramps in the calves of the legs (which are quite common), or from any of the symptoms of chronic uræmia, inquiry on the part of the physician may elicit the information that for months, possibly years, excitement and exertion have been provocative of breathlessness and palpitation of the heart; but having an obvious cause they were regarded as of no particular significance. As the disease advances, however, the nutrition of the heart no longer keeps pace with its enlargement, degenerations ensue, and evidences of embarrassed circulation result. It is now that symptoms referable to the cardio-vascular system dominate the clinical picture. To the hypertrophy of the heart, if it were simple, there supervenes dilatation, and to disease of the left ventricle is added disease of the right. There result cardiac asthma, congestion and œdema of the lungs, bronchitis, gastro-intestinal disturbances, generalized œdema and effusions within the serous cavities, and alterations in the urine. The urine lessens considerably in amount, and it becomes of high color and increased specific gravity; albumin increases in amount and casts become more numerous; dark granular, epithelial, and even blood casts and free erythrocytes may be encountered. The symptoms referable to the heart and lungs, at first transitory and nocturnal, become permanent, the other symptoms mentioned are added, and if relief is not afforded, death ensues with cardiac failure or uræmia or both dominating the final scene. Toward the end of life, concurrently with the cardiac failure, the pulse, which had been of high tension, may become of lessened tension; the second aortic sound becomes less accentuated; with embarrassment of the right heart the second pulmonary sound becomes accentuated; signs of dilatation, and gallop rhythm, always of serious moment, supervene.

In some cases the first suggestive symptom may be œdema of the lungs or of the larynx. The dyspnœa due to one or both of these may be augmented by hydrothorax; Cheyne-Stokes breathing may ensue, and in coma the patient may die. In other cases, the first suggestive symptom may be violent headache—due either to uræmia, cerebral congestion, or cerebral anæmia. The headaches are occasionally relieved by epistaxis, which is sometimes frequent and sometimes severe. Drowsiness and other of the nervous manifestations of chronic uræmia may be associated with the headaches, and frequently presage the onset of severe, acute, and even fatal uræmia. In some cases psychoses are prominent. In other cases neuralgias, or hemorrhages into the brain substance or into the meninges may occur. Hemorrhages into other structures, especially the stomach, intestine, skin, conjunctiva, lungs, etc., have been observed. Severe and uncontrollable vomiting and severe and exhausting diarrhœa are not uncommon. In other cases the disease is manifest by intolerable itching, formication, numbness of the extremities; in still other cases, by urticaria. In the late stages more or less widespread erythematous, or bullous, or desquamative skin lesions may occur. The most important symptom on the part of the special senses—neuro-retinitis—has already been mentioned. At times tinnitus aurium possibly associated with dizziness occurs; less frequently deafness or diplopia.

Diagnosis.—So long as cardiac compensation is maintained, the diagnosis of chronic indurative nephritis is evident from the results of an inquiry into the antecedents of the patient, the special etiological factors in the case, the course of the disease, the different clinical manifestations, and an examination of the urine and of the cardio-vascular apparatus. Indeed, a diagnosis based upon increased arterial tension, accentuated aortic second sound, and the physical signs of hypertrophy of the heart will rarely be wrong. The diagnostic importance of these cardio-vascular alterations cannot be overestimated. They are among the earliest clinical manifestations of the disease, and they may be detected even in the absence of positive signs of kidney disease on the part of the urine. Such is their double import, that, in the event of suspicion of chronic indurative nephritis, they should be diligently sought, and in the event of their accidental detection in the course of the routine examination of a patient, the presence of an unsuspected or latent chronic indurative nephritis must be persistently borne in mind until these cardio-vascular signs are otherwise adequately accounted for. It is usually a matter of extreme difficulty to distinguish clinically between primary chronic interstitial nephritis and the arteriosclerotic kidney; in some cases it is impossible; in most cases it is unnecessary. Usually the arteriosclerotic kidney exists for a long time without albuminuria, or with but the slightest trace of albumin in the urine. The disease may be suspected in elderly persons, with hypertrophy of the heart, increased arterial tension, and marked atheroma of the superficial arteries. Occurring as a manifestation of senility, it is usually of relative unimportance as contrasted

with the coexisting emphysema, dementia, marasmus, etc. When the heart begins to fail in chronic interstitial nephritis, diagnostic difficulties arise. The clinical picture in these cases much resembles that of a disorder primarily cardiac and secondarily renal. The presence of murmurs is not conclusive evidence, since they may be due to dilatation as well as to valvular disease. Diastolic murmurs, however, suggest a primary cardiac disorder, whereas albuminuric neuro-retinitis suggests a primary renal disorder. In some cases the diagnosis, for a time at least, is impossible; in other cases it may be made by a careful analysis and balancing of all the features of the case, by a knowledge of the past history of the patient, and by the results of administering digitalis and other heart tonics. Following the administration of digitalis, should the patient improve materially, should the urine increase in amount, and should albumin and casts finally disappear from the urine, the condition has been definitely determined to have been primarily cardiac with secondary renal congestion. If, however, with improvement in the condition of the heart, albumin and casts do not disappear from the urine, we may be confident of the existence of nephritis, a conviction that may be confirmed by examining the cardio-vascular apparatus now that compensation has been restored. It is scarcely likely that chronic indurative nephritis will be confounded with the so-called functional, physiological, cyclic, intermittent, paroxysmal, or adolescent albuminuria.

Course and Prognosis.—Chronic diffuse indurative nephritis may run a course entirely latent and lead to a sudden termination of life through the advent of acute uræmia, apoplexy, etc. In general the prognosis depends in large measure upon the condition of the cardio-vascular apparatus. In the early stages of the disease, in the stage when the disease may be discovered accidentally before there are any obtrusive evidences of it, the prognosis is fair with regard to life expectancy. The patient, however, should be warned of his affliction and advised to lead a hygienic, moral, and abstemious life. In these circumstances he may live ten, even twenty or more years; and it is not unlikely that the removal of the cause of the disease in the individual case may result in a standstill of the lesions in the kidneys and in the cardio-vascular apparatus. The dangers most to be apprehended are cardiac failure, rupture of the smaller arteries, especially those of the brain, and uræmia. In the later stages of the disease the factors that most influence prognosis are the condition of the cardio-vascular system, the quantity of urine excreted daily, the manifestations of uræmia, and the presence or absence of secondary infections, particularly those of the serous membranes, lungs, etc. With embarrassment of the circulation the prognosis becomes very bad. Compensation, when it fails, is sometimes never regained; with judicious treatment, however, the patient may be tided over the critical period and live, if careful, for a number of years. It is especially important to watch the quantity of urine excreted daily. Diminution in the quantity of urine often presages the onset of uræmia, which, however, may be averted by timely interference. The importance of the condition of the heart is here again brought into evidence, since failure of the cardiac power results in diminution in the amount of urine. This diminution in the amount of urine is especially a cause for concern if it be associated with headache, and both then demand immediate attention.

Treatment.—From the natural history of the disease we derive our indications for treatment, which are: (1) As far as possible, to remove the etiological factor in the individual case; (2) to prevent the accumulation of waste metabolic products in the system; (3) to maintain the integrity of the cardio-vascular apparatus; and (4) to meet the symptomatic indications as they arise. Thus the cardio-vascular manifestations of the disease assume a commanding position not only in the diagnosis and in the prognosis, but also in the treatment. In the early stages of the disease, when the patient may complain only of the passing of large quantities of urine, or of dyspnœa on exertion, etc., or when the physician may accidentally detect increased arterial tension, the nature of the disease should be recognized after a thorough investigation of the urine and of the heart, etc. Appreciating our inability definitely to cure our patient, we should be alert to the dangers that threaten him. He must be cautioned not to expose himself to the inclemencies of the weather; he must avoid all excessive muscular exercise, and if possible all mental worry; he must have his bowels open at least once daily, and in every way he must favor the action of the skin, so that as little work as possible may be thrown upon the diseased kidneys, heart, and blood-vessels. The skin, though a poor substitute for the kidneys, is still capable of performing considerable excretory work, and its action should be facilitated as much as possible—by wearing woollen underclothing and by a daily tepid bath. Hot baths may be given, but not indiscriminately; they are contraindicated if they increase the blood pressure to such a degree as to cause unpleasant throbbings. The action of the bowels should be promoted by a daily saline cathartic or by the use of any one of the well-known saline mineral waters. The diet is of the utmost importance, and probably the duration of life depends as much upon discretion in eating and drinking as upon any other factor. The matter, however, is largely individual. In many cases it is advisable to try an exclusive milk diet for a time. If the patient is obliged to take to bed this is all the more indicated. Milk, however, is most distasteful to many patients who will not submit to an exclusive milk dietary, even if assured that they will do better on it than on any other dietary—which is doubtful. In general it may be said that that diet should be allowed which best promotes a healthy state of body and mind; a diet free from irritative quantities and readily assimilable, a diet in which the nitrogenous elements are relatively reduced, and a diet sufficiently varied to be attractive. Whether or not albuminous food should be allowed must be decided in the individual case. In some cases it is well to withhold it for a time; in other cases moderate amounts may be permitted with good results. Large amounts appear to be injurious to most patients, though this is a subject that is still debated. However, it is quite certain that in many cases the exclusion of proteids from the dietary is not followed by good results. A wise rule is to permit some of the lean and easily digested meats to be partaken of once a day. Eggs are suitable and will be found of good service when taken raw—beaten up in milk. Tea and coffee in moderation may be permitted. In many cases alcoholic beverages must be prohibited; in other cases a little of the lighter wines may be allowed. In many cases the foregoing with an occasional mercurial purge, followed by a saline, for its depletive effect and its tendency to lower blood pressure, will suffice to produce amelioration of the symptoms, and possibly arrest of the lesions. If it accomplishes this, no other medicines should be given for the time being.

When the circulation becomes embarrassed medication is called for—the most serviceable remedies being nitroglycerin and caffeine. Nitroglycerin lowers the arterial tension and enables the heart still capable of performing its work to do so unhampered by excessive resistance in the arterial tree; it is said also to favor reduction in the amount of albumin excreted, but this action is doubtful and certainly immaterial. One minim of the one-per-cent. solution, or a tablet containing gr. $\frac{1}{100}$ of the nitroglycerin, should be given three or four times daily. This dose may be increased until the desired result is obtained. It is not necessary that the drug be given to the extent of producing unpleasant symptoms; sufficient reduction in the arterial tension is usually produced without this. Nor is great reduction in the arterial tension desirable; indeed, it is directly contraindicated, since a reduction of the tension even to that of the normal is fraught with danger of serous effusions, and of uræmia (on account of reduction in the amount of urine excreted). The nitroglycerin should be given for from four to six weeks and then discontinued for a week or ten days, unless the condition of the patient should render an earlier

return to its use necessary. In some cases the nitrite of potassium or of sodium may be substituted for the nitroglycerin, but they are less efficacious. During recent years erythrol tetranitrate has been employed with good results. Inasmuch as the nitrites interfere somewhat with the oxygenating powers of the blood their continuous administration should not be persisted in too long. The caffeine is best given in three-grain doses, and generally in conjunction with the nitroglycerin. The only contraindication to its use is an insomnia that it sometimes provokes. Iodide of potassium or of sodium is still advocated in the treatment of this disease, and in some cases good results appear to attend its prolonged use. It should be given in small doses, three to five grains three times daily, unless there be a clear history of syphilitic infection, or marked arterio-sclerosis. It is difficult, however, to estimate the amount of good that with justice may be attributed to its use. The same may be said of small doses of mercuric bichloride and of the chloride of gold and sodium. At all events their use is unattended by harm, and they may serve to retard the progress of the renal and cardio-vascular lesions. In the absence of serious cardiac debility, cardiac stimulants, such as digitalis and strophanthus, should not be given. Nor is the routine administration of iron to be countenanced. Anæmia is rarely marked, and any virtues that iron may possess are outweighed by certain ill effects, such as its tendency to constipate, to produce headaches, etc. However, should anæmia become marked, iron may be given in small doses. Quinine, strychnine, and arsenic are often of service in this stage of the disease, as well as earlier. Quite recently Edebohls and others have attempted the cure of chronic nephritis by stripping the capsule from the kidneys—with results as yet problematical.

When marked cardiac debility and manifest failure of compensation develop (generalized œdema, effusions in the several serous cavities, diminution in the amount of urine, increase in the amount of albumin, etc.), the time for administering digitalis and other cardiac stimulants has arrived. In general, the treatment of this stage of the disease does not differ from that already laid down in connection with acute and chronic non-indurative nephritis, and from that commonly practised in cases of lack of cardiac compensation. Impending uræmia, manifest by headache, restlessness, foul breath, coated tongue, vertigo, etc., may be warded off by nitroglycerin and caffeine, saline cathartics, alkaline diuretics, large amounts of water, hydrotherapeutic measures, and restriction of the diet to milk. In some cases digitalis is urgently called for. The treatment of acute uræmia developing in the course of chronic indurative nephritis does not differ from that already mentioned.

Aloysius O. J. Kelly.

KIDNEYS, DISEASES OF: PARASITES.—In the United States, renal parasites are very rare. Those which are most often observed are: 1, Echinococcus; 2, strongylus gigas; 3, pentastomum denticulatum; 4, distoma hæmatobium; 5, ascaris lumbricoides; 6, actinomycosis.

1. Echinococcus.—This is the most common of the renal parasites, but it is a rare affection. If we add together the figures of Davaine, Neisser, Finsen, and Thomas, we have 2,111 cases of hydatid disease of all parts and organs, in 115 of which the kidney was the seat of the disease—a percentage of 5.44. The actual frequency is probably even lower than this. Hydatids occur more frequently in the kidney than in any other organ of the body, except the liver (most frequent of all) and the lungs. The two sexes seem to be about equally affected. It occurs about twice as frequently in the left kidney as in the right, for which no satisfactory explanation has been offered. The cysts may occupy one or both kidneys. Any part of the kidney may be invaded by the cyst or cysts, which may lie just beneath the capsule or be buried deep in the substance of the organ. The remaining kidney tissue usually continues healthy until the pressure of the cyst becomes injurious. The large cysts often contain daughter cysts. The cysts may remain small in size, or they may attain immense proportions.

Symptoms.—These are very unsatisfactory, and are variously described. The trouble develops insidiously and slowly, is usually painless, and produces no changes in the urine until late in the disease. A palpable tumor takes months or years for its growth, and is usually the first symptom which is appreciable. The tumor is rounded, tense, elastic, not tender, and may have rounded nodules upon its surface. There may be fluctuation and "hydatid fremitus." The cysts may develop rapidly and produce painful sensations from pressure. Rarely hæmaturia may be produced. Some of the cysts may rupture into the pelvis of the kidney, causing the echinococci or their hooklets to appear in the urine, which will at once establish the diagnosis. The passage of the echinococci may produce miniature renal colic, similar to those symptoms caused by the passage of renal gravel or a small calculus. Very often the cysts set up a pyelitis, with pyuria and hæmaturia. The cysts may become infected and rupture externally, producing a perirenal abscess. The hydatid tumor may push upward, perforate the diaphragm, and open into the pleura or bronchi, in which case the echinococci will be coughed up and the expectoration will have a urinous odor. The cyst may rupture into the general peritoneal cavity, or into a hollow organ, *e.g.*, stomach, intestine, etc.

The *diagnosis* is made by considering (Morris):

1. The residence, habits, and calling of the individual in reference to association with dogs. 2. The slow, silent, insidious growth of a tumor. 3. The absence of pain, of fever, of general constitutional disturbances, and of any change in the composition, or any variation in the quantity of the urine. 4. The uniformly smooth, globular outline of the tumor, with fluctuation and hydatid fremitus. 5. The renal connection of the tumor and the lack of ordinary respiratory movements. 6. The possible discharge of hydatids per urethram, by the mouth, or by the anus. 7. Attacks of renal colic caused by, and associated with, the discharge of hydatids by urethra, producing diminution or subsidence of the flank tumor.

Treatment.—Aspiration is dangerous and should not be employed. The same remark applies to the injection of substances into the cysts. The only proper treatment is to cut down upon it by the lumbar route, tap it, removing the fluid by aspiration, and then to incise the cyst wall. As much as possible of the wall should be removed, and the edges of the cavity ought then to be sewn into the lumbar wound. The cavity is then packed and allowed to heal by granulation. Nephrectomy is allowable only when the whole kidney is involved in the cysts. In certain cases it may be possible to excise the whole cyst, in which case the raw surfaces of the kidney may be brought together by sutures. This expedites recovery.

2. Strongylus Gigas.—This parasite is very rare in man, but common in the lower animals. It bears a great resemblance to a large earth-worm but differs from the ascaris lumbricoides in its reddish color, and by its having six papillæ around the mouth, the ascaris having only three. It is found in the renal pelvis, producing great irritation, with pain, hæmaturia, pyrexia, and strangury. The pain is somewhat similar to, though milder than, renal colic. It is not known how the worm reaches the kidney. Incision into the pelvis of the kidney, and the removal of the worm is the only treatment.

3. Pentastomum Denticulatum is a pathological curiosity which has been found once or twice in man, but which has no clinical interest. It is a parasite belonging to the order of "mites," whose natural habitat is the nasal cavity of the dog.

4. Distoma Hæmatobium.—In temperate climates this is rather a pathological than a clinical verity, although it is very common in Egypt and Southern Africa. The embryos enter the alimentary canal through the medium of drinking-water, whence they make their way to the kidneys. They may block the ureter and cause hydronephrosis. Their entrance into the bladder may cause considerable irritation. The only certain means of diagnosis is the discharge of ova in the urine. No treatment can be adopted which will have any influence on the parasite.

5. Ascaris Lumbricoides (the common round-worm of the intestinal canal) has sometimes found its way into the renal pelvis—just how, it is difficult to explain. This worm has been mistaken for the strongylus gigas, but this latter has invariably a deep red color, which will differentiate it from the former.

6. Secondary actinomycosis of the kidney has been described several times. Israel has published the first case of primary actinomycosis, diagnosed in life, and successfully operated upon. For a further description of this case, I refer to Israel's work.

Clarence Arthur McWilliams.

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KIDNEYS, DISEASES OF: SYPHILIS. See *Syphilis*.

KIDNEYS, DISEASES OF: TUBERCULOSIS.—Renal tuberculosis occurs under two distinct forms, with one of which—the dead-room manifestation of a general disseminated disease—we have nothing to do except from a statistical standpoint. In these cases both kidneys are almost invariably affected and the diagnosis of renal involvement is not made during life, or, if it may be, it calls for no attention in the face of the more formidable menace to existence from the coexisting implication of the pulmonary, meningeal, and peritoneal structures.

If we permitted ourselves to be guided by such necropsy records it would be idle to expect to find among the living any cases of purely unilateral renal tuberculosis in which our therapeutic resources could be of benefit. The writer's morgue investigations in all forms of renal tuberculosis show a post-mortem percentage of 81, in which both kidneys were tuberculous, and 19 in which but one organ was affected; whereas his clinical material, in which urinary differentiations were made by ureteral catheterization, has given 33 per cent. of double and 66 per cent. of single renal involvement. It is needless to say that all of these cases belonged to the class in which more or less chronic and more or less localized manifestations of tuberculosis prevailed, and in which there were almost always symptoms referable to the urinary or genital organs.

From February 1st, 1893, to March 28th, 1902, 1,427 autopsies were made in the Presbyterian Hospital of New York City: 869, or 60 + per cent., were of males; 558, or 39 + per cent., were of females. Of this number 258 bodies, or 18 per cent., showed some tuberculous lesion somewhere in the body; 169, or 65 per cent., of these were in males, 89, or 35 per cent., in females. Of the 258 tuberculous bodies 48, or 18.5 per cent., showed renal involvement.

Age.	Kidney.	Spleen.	Liver.	Adrenals.	Total.
1 to 5 years	10	16	10	1	37
5 to 10 "	3	3	3	..	9
10 to 20 "	3	3	3	..	9
20 to 30 "	10	4	5	2	21
30 to 40 "	13	4	7	2	26
40 to 50 "	2	1	2	2	7
50 to 60 "	4	2	1	1	8
60 to 70 "	1	..	1	..	2
70 to 80 "	2	..	..	..	2
	48	33	32	8	121

Of the 48 renal cases 32, or 66 per cent., were in males, while 16, or 33 per cent., were in females. Of the 48 renal cases, 39, or 81 per cent., had double infection, while 9, or 19 per cent., had single infection. Of these 9 cases, the right kidney was involved in 5, the left in 4. None of the 48 renal cases failed to show some pulmonary tuberculous lesion. No instances of healed renal lesion were met with, while the records cite 5 cases of healed pulmonary processes.

Cases of acute miliary tuberculosis were much the most common in infancy, and in such the spleen and liver were affected at least as often as the kidneys. The periods between the third and fourth decades were the next most likely ones for tuberculous infection, although here many of the cases were of the acute general variety. The more chronic and more localized forms were, however, strikingly in evidence, and the prevalence of renal tuberculosis over that of the spleen and liver was noted. In respect to individuals at this age, both morgue and clinical records agree in showing that here are mainly to be found the victims of a chronic unilateral renal tuberculosis, which is amenable to surgery.

An attempt was made to collect the number of cases of renal tuberculosis received in the hospital during this same ten-year period, but many difficulties rendered it impossible to make more than an approximate estimate; doubtless quite a number must have eluded diagnosis, and, when no autopsy occurred, they escaped record. Most of the 48 autopsy cases had been unrecognized previously. If these are added to the 34 diagnosed as renal tuberculosis there were in all 81 cases, since one case is common to both counts.

Of the 34 cases diagnosed as bilateral or unilateral disease, 14 of the patients in the latter class consented to the proposed nephrectomy, and of this number 13 recovered, an operative mortality of 7.1 per cent. The one death occurred in a man, twenty-five years of age, at the end of six months, while the patient was still in hospital, and was not due to the operation but to extension of previously existing chronic pulmonary processes and a final acute miliary distribution in these same organs. At autopsy a broken-down tuberculous nodule was also found in the remaining kidney.

A second death occurred in a lad of sixteen years, due to tuberculous meningitis, a little more than six months after operation and five after he had left the hospital. This patient was at the point of death when operated upon, and, although he gained thirty pounds shortly afterward, other foci of the disease had already been recognized before the emergency operation, and it was predicted that a return to his former unhygienic home conditions would result in an early and fatal recurrence. The remaining twelve patients are, so far as can be learned, in a fair state of health at this time. The fourteen patients were, with the exception just mentioned, between twenty and forty years of age.

Etiology.—Although instances of primary unilateral renal tuberculosis probably exist, we must not think that surgical measures of relief are to be reserved for these rare cases alone. Most of the patients supposed to belong in this category are certainly such as have had an insignificant dormant focus elsewhere, and from which bacilli have gained access to the blood and been planted successfully in one kidney. What the antecedent renal condition is which makes it a favorable soil for this bacillary growth and its subsequent necrotic processes, is not known. If irritation or faulty circulation were a predisposing cause the disease ought to be, more frequently than it is, found associated with cases of calculus or gravel, and of movable kidney. It is a striking exception to find a kidney which is at the same time the seat of lithiasis and tuberculosis, unless the concretion represents a terminal stage of the latter and possibly a natural and conservative effort at cure.

On the other hand, a history of some external lumbar or abdominal injury is not so infrequently to be had from these patients, but whether such was appreciated only because the organ was already the seat of disease and unduly sensitive it is difficult to determine, and this difficulty is increased by the fact that occasional cases of renal tuberculosis are far advanced when they first come to the

notice of even the patient. It is claimed by some recent writers that the parenchyma of the kidneys is insensitive, that pain is experienced only in connection with lesions of a distending nature such as lead to stretching of the capsule or distention of the pelvis. This statement would seem to be negatived by the fact that some considerably sacculated tuberculous kidneys are quite painless. It seems to be essential to the occurrence of a primary tuberculous renal lesion that a bacillus should gain admission to a blood-vessel by a prick or laceration, else that it should be taken up from a mucous or cutaneous surface by a lymph space, advanced until it enters a vein, thence carried to the heart and one of the renal arteries, an arteriole of which probably presents it to a glomerulus for elimination. Sherrington's experiments justify the assumption that the tubercle bacillus can pass this filter without any necessary lesion of the excretory membrane.

Durand-Fardel has demonstrated these bacilli within and just outside the vessels of a glomerulus, where they had not yet excited an initial tuberculous process, nor could the microscope detect the slightest evidence of their presence by tissue change or otherwise.

That more often than not the bacilli are carried by the blood to the kidney from some already existing but inappreciable pulmonary lesion is the writer's belief, so that although the renal focus becomes an ostensible and major object of offence it is not really the primary one. If the kidney is successfully removed the original focus elsewhere may long afterward again serve as a point of departure of infection for other organs, or may take on acutely destructive processes within itself. These possibilities should not, however, deter the surgeon from advising the earliest possible nephrectomy for purely unilateral disease.

Two other modes of renal infection are recognized. The so-called ascending, in which extension by continuity of the disease from bladder to kidney takes place (Plate XXXIX.). Here the lymphatic spaces and channels of the ureter are presumably the agents in conveying the bacilli. When this occurs the bladder may have had its original infection from a lesion of the prostate or seminal vesicles, from a descending tuberculosis from the opposite kidney, from an implantation made by an instrument passed through the urethra, or from a focus initiated by blood bacilli transported to the mucosa. This last cause may produce a lesion in the ureter from which the disease can be spread both upward and downward.

When tuberculosis reaches the kidney from the ureter it generally presents a picture of necrosis extending peripherally in contradistinction to the processes which characterize a blood-implanted tuberculosis in which the cortical zone of the kidney—that part containing the great majority of the glomeruli—is the principal part to suffer the early inroads of destruction; although the pelvis and parts of the ureter may be affected with tubercles planted from the cortical lesion.

The third mode of infection is by indirect extension through the renal capsule from a tuberculous disease of some contiguous or remote organ. Such occurrences are very rare. Although it is not infrequent to find advanced tuberculosis of the adrenals at autopsy, the writer has seen no cases cited of a contact infection of the kidney, whereas, in cases of new growth of the adrenal body, invasion of the kidney is not rare.

The renal capsule seems to exert a sufficient protection against tuberculous contagion of its parenchyma, but through the pelvis a burrowing vertebral abscess occasionally makes a successful breach, and very rarely a tuberculosis may by invasion of the ureter extend from the peritoneum to the kidney. It is probable that eighty per cent. of the cases of renal tuberculosis are of hæmatogenous origin.

The susceptibility of the kidney to infection by pathogenic germs carried to it by the blood has been clearly demonstrated, whereas the potency of the lymphatic channels for rapid and wide germ dissemination is not so well established; yet in the distribution of bacteria by the continuity of their pathological processes for limited distances and by comparatively slower progress, the efficacy of the lymphatics is perfectly appreciated. That this is the mode of dissemination in an ascending renal tuberculosis I am inclined to believe, and I have also no doubt that it represents the mode of extension of the disease to the other parts of any already infected organ or group of organs.

It was a good while ago ascertained that the secretions and tissues of the healthy body do not contain bacteria, and it is now equally well recognized that when bacteria have gained an entrance to the body they may appear in the secreta. Particularly is this true when the germs are such as are known to be capable of exciting lesions in the organs affording the secretion. Thus, in Wyssowkowitsch's experiments in seventeen animals subjected to intravenous injection of different micro-organisms pathogenic to the kidneys, the germs were found in the urine thirteen times.

The non-pathogenic germs, on the other hand, wholly disappear from the circulation after a while without having appeared in the secreta.

As long ago as 1882 Cohnheim maintained that by renal excretion the human organism possessed the means of ridding itself of both dissolved and organized poisons. Among the latter he included tubercle bacilli, and he held that they could be transferred from a pulmonary source by the blood to the urine and so enter the bladder, there to establish a tuberculosis without injuring or infecting the kidney during their passage.

Von Kahlden professes to have observed kidneys through which the bacilli have filtered, and which organs did not present the slightest alteration caused by the transit of the bacilli. The impossibility, however, of a thorough examination of the entire glandular tissue of a kidney must be apparent.

The investigations of Cavazzani and Wyssowkowitsch led them to conclude that this passage of micro-organisms through the renal membrane could not occur prior to some lesion of the renal epithelium, and that such a lesion was associated with the escape of blood.

From his own experiments Sherrington decided that because proteids are not always discoverable in the urine, it is not necessary to insist upon the conclusions of Wyssowkowitsch and others, according to which noxious bacteria escape in the secreta only when the blood itself containing them escapes. And yet he admits that his deductions are equally opposed to the extreme views of Cohnheim and others, who assert that the transit of bacteria across the renal membrane occurs while it is still normal in condition.

"This membrane," Sherrington says, "is rather to be regarded as then exhibiting in a minor degree the pathological change which, when increased, will render it pervious to the same extent that the capillary wall becomes pervious in an area of inflammation." This opinion he believes to receive support from the fact that the injected germs are not immediately found in the secreta, but only subsequently, after the poisons produced by the infection have had time to act upon the membrane and render it pervious.

Sherrington adds: "Among the species observed to escape through the membrane, even in the absence of escape of blood, are some that are not motile; this suggests that in their transit across the secreting membrane the bacteria themselves are passively conveyed, that the transit is less an active migration than a passive transference."

It is, then, a pertinent question whether the victims of more or less general tuberculosis can present tubercle bacilli in their urine and yet not have tuberculosis of the urinary tract. One of Sherrington's experiments, already referred to, would lead us to believe that this is possible.

I saw some years ago a case in which this hypothesis could not but be considered. I had resected the tuberculous left knee of a man, A. O., twenty-eight years old, who since the age of ten years had given evidence at

EXPLANATION OF
PLATE XXXVIII.