EXPLANATION OF PLATE XXXVIII.

FIG. 1.—Chronic Renal Tuberculosis. At the lower pole of the kidney is seen a calcareous concretion surrounded by a thick fibrous capsule. In the middle zone there is a caseous mass which is walled off from the surrounding tissues in a similar manner—nature's effort to effect a cure. At various places in the cortex are miliary and larger tubercles of later date. (Dr. Charles N. Dowd's case of nephrectomy.)

FIG. 2.—A Non-tuberculous Kidney which has Undergone Lipomatous Transformation, Secondary to Calculous Disease. The stone is seen in the pelvis. This metamorphosis is similar to that described by some writers as healed renal tuberculosis. (Dr. Robert F. Weir's case of nephrectomy.)

FIG. 3.—Pseudotuberculosis of the Kidney; "Pseudotuberculosis Cladothrichica" of Eppinger. (Dr. George A. Tuttle's case.)

FIG. 4.—Subacute Renal Tuberculosis. The kidney shows on its surface numerous cortical tubercles and one considerable cyst. (Dr. F. Tilden Brown's case of nephrectomy for disease involving one kidney.)

FIG. 1

FIG. 2

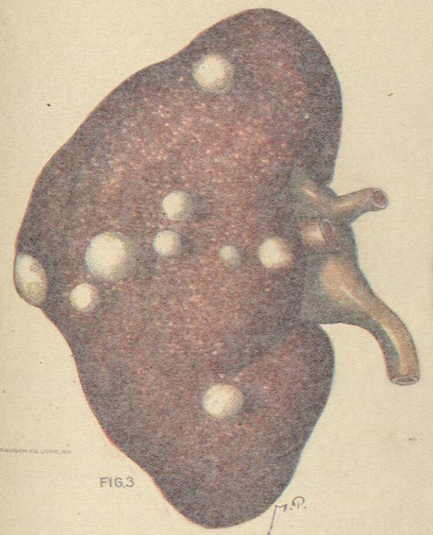


FIG. 3

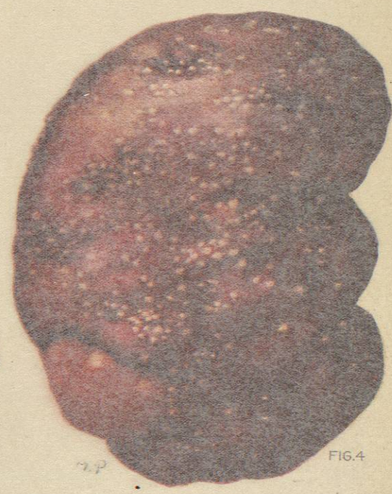


FIG. 4

POST-MORTEM APPEARANCES IN CHRONIC TUBERCULOSIS AND PSEUDO-TUBERCULOSIS OF THE KIDNEY.

(CASES OF DR. DOWD, DR. WEIR, DR. TUTTLE, AND DR. F. TILDEN BROWN.)

[Only slight reduction in size.]

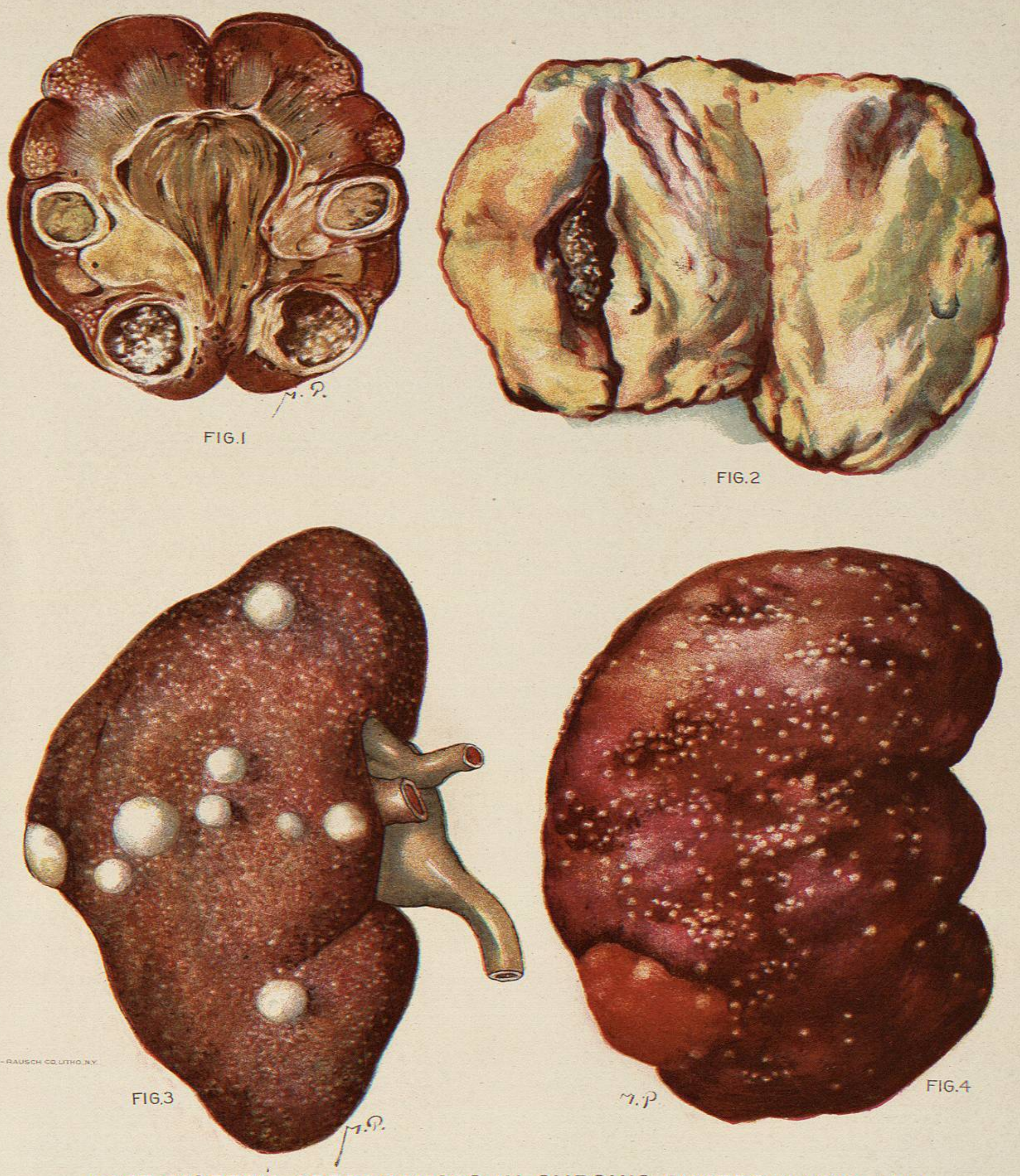
EXPLANATION OF PLATE XXXVIII.

FIG. 1.—Chronic Renal Tuberculosis. [illegible] of the kidney is seen a calcareous concretion surrounded by a thick fibrous capsule. [illegible] there is a caseous mass which is walled off from the surrounding [illegible]—nature's effort to effect a cure. At various places [illegible] of later date. (Dr. Charles N. Dowd's case of nephrectomy.)

FIG. 2.—[illegible] Transformation, Secondary [illegible]. This metamorphosis is similar to that [illegible]. (Dr. Robert F. Weir's case of nephrectomy.)

FIG. 3.—[illegible] "[illegible]" of Eppinger. [illegible]

FIG. 4.—[illegible] numerous cortical tubercles [illegible] for disease involving [illegible]



POST-MORTEM APPEARANCES IN CHRONIC TUBERCULOSIS AND PSEUDO-TUBERCULOSIS OF THE KIDNEY.

(CASES OF DR. DOWD, DR. WEIR, DR. TUTTLE, AND DR. F. TILDEN BROWN.)

[Only slight reduction in size.]

different times of various regional tuberculoses. Three months later his tuberculous right testicle was removed. Although he had never experienced any urinary symptoms, I examined his urine before the last operation, and found tubercle bacilli in each of four mountings; I then examined him more closely in reference to vesical or renal symptoms, but with negative result. Hence I was led to infer that the bacilli must have come from the urethra, prostate, or seminal vesicles. To test it, some two weeks later the urine was collected by a sterilized catheter, allowing an ounce, for cleansing the eye, to run off uncaught. As before, the urine by gross inspection was quite clear by centrifugal sedimentation; enough pus corpuscles were obtained to make six slim mountings. Tubercle bacilli were found in each; in nearly every instance the bacilli were in the leucocytes. The urine was sterile to culture on the ordinary media. It was acid, of 1.022 specific gravity. There was no albumin, and but an occasional blood cell was found microscopically. The patient did not have to urinate during the night, and he had no frequency of micturition during the day.

This catheter test did not, of necessity, warrant the positive conclusion that the bacilli came from the kidney; but had they come from a vesical lesion, I think some epithelial cells would have been found with the pus cells. After he left the hospital I saw the patient several times, and always found him going about in what he called "good health," having gained about fifteen pounds since the operation. All attempts to discover some subjective evidence of urinary trouble were futile.

Four months later, in May, 1896, he was taken suddenly ill and brought to the medical service of the Presbyterian Hospital, where he died three days later of acute miliary tuberculosis of the lungs. It was of great interest to determine the source of the tubercle bacilli found in his urine two years before. Autopsy afforded this opportunity. In the left kidney were two moderate-sized chronic foci communicating by calyces with the pelvis. The left ureter was much dilated in two places, hyperæmic, and contained a few minute tubercles. There was a single lesion in the right kidney similar to those in the left, but it appeared to have no communication with the pelvis of the organ. The vesical mucous membrane about the left ureteral opening was conspicuous by the size of its blood-vessels only. Both seminal vesicles had old cheesy deposits. The size and shape of that on the right side caused a marked asymmetry of the base of the bladder and ureteral openings (see New York *Medical Journal*, April 3d, 1897). Every portion of both lungs was studded with minute tubercles. The patient's temperature had been almost constant between 104° and 105° F. during the three days that he was in the hospital. The urine appeared normal and showed no albumin. It was not then examined for tubercle bacilli.

Whether this fatal attack of pulmonary tuberculosis was instituted by a fresh infection of more virulent bacilli or by a great and sudden dissemination (by way of the blood channels), throughout the lungs, of the very parasites he had so long nourished elsewhere, must be left to conjecture.

A great proportion of the victims of chronic tuberculous disease of the kidney is to be found among those having a more or less marked family history of tuberculosis. For this reason trifling urinary symptoms should, in such cases, receive the most careful and persistent scrutiny.

The *pathology* of renal tuberculosis does not differ from that of the disease found elsewhere, and its morbid anatomy is mainly dependent upon the mode of invasion, the activity of the disease, the virulence of the bacillus, and the resisting powers of the individual. After deposition, by the blood, of tubercle bacilli in the cortical area of the kidney, the presence of epithelial and giant cells surrounded by and interspersed with lymphoid corpuscles is the typical picture of a commencing miliary granulation. The lesion, microscopic at first, progresses to a stage where it becomes a grayish-white or yellowish tubercle seen and felt on gross inspection. The further development of the single lesion or the coalition of several similar lesions leads by coagulation-necrosis to the cheesy nodule. Augmenting at the expense of the surrounding parenchyma, this finally ruptures into one of the calyces, its contents and subsequent débris then mingling with the excreted urine. More rarely the lesion works toward the capsule, lifting and possibly breaking through it, and instituting there a perinephritic tuberculosis. When a very low grade of activity pertains, the evidences of efforts to stay the progress of such a lesion may rarely be met with in the shape of a dense white-walled limiting membrane encasing either a putty-like substance or a still more advanced metamorphosis of the latter in the shape of a calcareous formation. A beautiful example of this is seen in parts of a tuberculous kidney recently removed by Dr. Dowd (Plate XXXVIII.).

Cystic degeneration of tuberculous origin, independent of ureteral occlusion, is occasionally met with. Tuberculous hydronephrosis is most commonly initiated by ureteral occlusion due to ascending disease, whereas tuberculous pyonephrosis is generally due to occlusion of the ureter by cheesy matter or tissue débris from the already diseased kidney.

Cases of complete arrest of renal tuberculosis are occasionally reported. Madelung describes a condition in which the renal pelvis is invaded by a sclerotic fatty tissue with closure of the ureter and apparent cessation of active inflammation; Michel reports a case in which extreme atrophy of the affected kidney was attended by the formation of an encasing mass of sclerotic fat. A very similar condition is shown by Morris to be at times initiated by a perinephritis, in which the cellular and adipose tissues surrounding the kidney take on this unusual change. And, again, some affection of the kidney itself appears responsible for a later invasion of its parenchyma by fat, probably starting along the vessels at the hilum. In extreme cases this will transform the whole organ into a fatty mass. A rare specimen, in which a calculus encased in the pelvis may have been the initial cause of this lipomatous metamorphosis, is seen in what was once a kidney, removed by Dr. Weir (Plate XXXVIII.); and, from the description given, we presume that it represents the same appearances as were observed in the cases of healed renal tuberculosis described by Madelung, Tuffier, Le Dentu, Newman, and Michel.

Diagnosis.—When a kidney is exposed at operation it is often impossible to say from gross inspection that it is affected with tuberculosis. But this is scarcely necessary, for the diagnosis as to both the nature and the localization of the disease should in every case be made before the patient is brought to the operating table; and consequently we shall not attempt to give a table of differential diagnoses to help distinguish between the appearances of a tuberculous kidney and those of the septic, syphilitic, malarial, carcinomatous, or leprous kidney. Because of its variety and novelty, it may, however, be of interest to give a brief description of a disease which presents cortical and disseminated lesions exactly like those of tuberculosis. Not alone macroscopically but histologically both diseases are characterized by infiltration of the framework surrounding the lesions, by degeneration of the inflammatory exudate going on to necrosis, and by solution of the tissue and exudate.

Pseudotuberculosis.—The illustration—Fig. 2, Plate XXXVIII.—is that of a kidney removed at autopsy from a patient of Dr. G. A. Tuttle, of the Presbyterian Hospital, by whom the specific micro-organism has been cultivated and extensively experimented with. Dr. Tuttle's studies are abbreviated in the following résumé.

A disease which closely resembles tuberculosis in the character and distribution of its lesions, and which, in certain stages at least, presents almost precisely the same appearances as those which are observed in miliary tuberculosis, so that it may easily be mistaken for that disease, was first described by Eppinger in 1891 as "pseudotuberculosis cladothrichica." The micro-organism proved by Eppinger to have caused the disease was reported by him as a cladothrix, although he described a real branching of the threads. It has been found to be a

species of streptothrix, a genus of the family to which belong the tubercle bacillus, the Klebs-Löffler bacillus, and the actinomyces. During the past six years seven similar cases have been reported by different observers, one of them having been diagnosticated during life and the diagnosis confirmed at autopsy. A few other cases have been reported as streptothrix disease, but the proofs have not been convincing.

A case of this disease in which were combined the lesions described by the different observers, occurred in the medical service of the Presbyterian Hospital in the summer of 1898. The patient, a woman thirty-three years of age, gave the history and presented the clinical symptoms of acute lobar pneumonia. The onset was sudden and was marked by a chill with fever, rapid pulse and respiration, pain in the left side and back, and cough. There was a small area of consolidation in the lower part of the upper lobe of the left lung, which increased to four or five inches in extent. Prostration was marked. Great pain in the lumbar region and thighs and intense vesical tenesmus were prominent symptoms in the later stages of the disease. Eight or ten small subcutaneous abscesses appeared on the forearms, abdomen, and thighs, and there were repeated attacks of profuse sweating. Death took place on the fifteenth day of the disease.

The autopsy, nine hours after death, showed a resolving area of pneumonia in the left upper lobe, the appearance of acute miliary tuberculosis in the right lower lobe, and small foci of suppuration scattered throughout both lungs. One small cheesy nodule was found in the right lower lobe. The heart muscle showed several small white infarctions which were becoming softened. Opposite one of these, in the wall of the left ventricle, was a thrombus of fibrin containing foci of pus. The pancreas contained many small abscesses. The liver and spleen were normal. The kidneys were thickly sown with minute white granules, evenly distributed through their substance and showing on the surface. In addition there were several prominent white nodules, one-quarter to one-half inch in diameter, which were found to contain thick, tenacious matter. The mesentery was densely studded with miliary tubercles and tubercles were scattered over the parietal and intestinal peritoneum.

The microscopical examination showed a typical picture of tuberculous inflammation in the lower lobe of the right lung. There were characteristic tubercles composed of epithelial cells and giant cells, some with cheesy degeneration, and some with fibrous tissue in the centre. There were also areas of cicatricial fibrous tissue such as are seen in chronic tuberculosis. No other characteristic tuberculous lesions were found. The kidneys showed little collections of small round cells, some of them with a central area of granular degeneration, but no giant cells were found. Careful searching failed to discover any tubercle bacilli in any of the lesions, even in those of the right lung which appeared most certainly to be tuberculous. Smears from the abscesses and sections of the lungs, heart, and kidneys, stained by Gram's method, showed an abundance of branching threads in the tubercle-like nodules. None, however, could be found in the tubercles with giant cells, these apparently being lesions of longer standing than the small round-celled tubercles.

Cultures from the kidneys showed a pure growth of slender branching threads in light yellow colonies. Growth occurred in the incubator upon all of the common culture media. Spores were formed upon potato. Subcutaneous abscesses developed in rabbits and guinea-pigs at the site of inoculation, but the animals recovered. When the bacilli were placed in the blood stream in rabbits, rapid emaciation and death resulted in from seven to sixteen days, with widespread tubercle-like lesions, always most numerous in the kidneys.

If we compare the appearance of the growths on different culture media and the results of animal experiments with the descriptions of Eppinger's streptothrix, we see that the micro-organisms found in the two cases are closely similar if not identical.

No motility of the bacilli was noted at any stage of growth in Tuttle's case, while Eppinger described a slow movement of the threads. Other observers were not able to confirm this.

The *symptomatology* of renal tuberculosis taken collectively gives a rather complex picture. The symptoms not only vary more or less at different stages, but it has to be remembered that, according as infection has occurred through either of the two main channels of access to the kidney—the arterial and the ureteral—the symptoms, during the early stages at least, will be dissimilar.

In cases of blood infection, when the newly planted tubercles act as excitants to the functional activity of the organ, polyuria is not infrequently noted; but with this exception the early stage may be symptomless until the lesions have advanced to the formation of a palpable tumor, or have reached the stage of eruption into one of the calyces, which often occurs before tumefaction. With this rupture may at once occur in a minor degree some of the symptoms which will have been present from the outset in a case of the ascending ureteral variety; for the victim of the latter will have been undergoing, for a long time prior to ascending access to the kidney, all those distressing vesical and ureteral symptoms to which for the first time a patient with the first form of infection is now exposed through the liberation of tuberculous material by rupture of the hitherto walled-in kidney lesion. Although the writer has never seen a specimen which demonstrated positively this invasion of the kidney by ureteral ascent—the kidney for instance being sound, while yet the ureter showed tubercles spreading upward from a similarly diseased bladder—he has no sufficient reason for denying the possibility of its existence. He has always regarded the right kidney and ureter shown in Plate XXXIX. as an example of this sort, while the totally destroyed organ of the opposite side must have been an antedating infection that originated by way of the blood, since the ureter shows nothing besides a stricture choked with débris.

Sometimes the subjective symptoms are pronounced even in the early stages, there being an acute pain referable to the kidney or upper part of the ureter. When such painful seizures occur as crises, accompanied by nausea and vomiting, the attendant is more apt to suspect renal calculus, gravel, or angled ureter with movable kidney than tuberculosis. In other cases, again, pain is little noticed in the advanced stages of the disease; all that we find is an irritable behavior of the bladder, and too often these vesical symptoms are cited by the physician as evidence that this viscus is the original and only seat of the trouble. A dull aching in the lumbar region is always significant, and when in addition to this a mass corresponding to the kidney can be palpated, the manipulation eliciting pain, the indication for further study is strong. Commonly in the early stage no kidney can be felt, but bimanual pressure will show that the organ is more sensitive than the opposite one.

In quite a proportion of the cases it will be found that the patients give a history of having had, at different stages of the disease, malaise with chilly and febrile manifestations resembling malaria, and of having been treated at various intervals for this disease. A mixture of blood with the urine in sufficient quantity to attract the attention of the patient and be called a hemorrhage is an unusual symptom, which has, however, at times been noted as one of the earliest; but it points with vastly greater probability to the presence of a new growth. On the other hand, a microscopic quantity of blood is among the commonest of the symptoms of renal tuberculosis from the time of rupture of the lesion until the end.

Pyuria, which in varying degrees is a constant symptom after the stage of rupture, is apt to be noted by intelligent individuals, and to the physician it should give immediate warning to look for tubercle bacilli. Especially is this the case when with an acid urine the microscopic mounting, dried and stained only with methylene blue, shows a field containing nothing but pus corpuscles and an occasional blood cell with very few or no epithe-

EXPLANATION OF
PLATE XXXIX.