cases, the lesions are confined to the renal epithelium (that of the tubules as well as that of the Malpighian bodies), and they consist of: cloudy swelling (swelling and granulation of the cellular protoplasm), which is common in

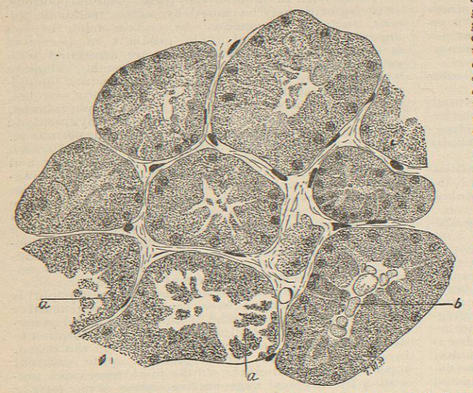

FIG. 3061.—Acute Parenchymatous Degeneration of the Kidney Epithelium from a Case of Yellow Fever. *a*, Swollen and granular epithelium desquamating and disintegrating; *b*, hyaline material in the lumen of the tubule. (Delafield and Prudden.)

most of the acute infective diseases (Fig. 3061); fatty degeneration (fat droplets in the cellular protoplasm and alterations in the nuclei), which is common in the severer infections, in certain intoxications, such as phosphorus poisoning and the like, and in severe anæmias (Fig. 3062); hydropsical degeneration (swelling of the cellular protoplasm and vacuole formation), and complete necrosis, which occur in the severest infections such as cholera, and in certain intoxications, such as, cantharidal poisoning, corrosive-sublimate poisoning, etc. These lesions are but different grades of the one process, and they may be found singly or in combination.

(*b*) ACUTE DIFFUSE NEPHRITIS.—In this form the lesions, though widespread, are frequently exaggerated in foci, and they involve especially the parenchyma—whence the common term, *parenchymatous nephritis*. In some cases the lesions are especially conspicuous in the glomeruli—*glomerular nephritis;* in other cases, in the tubules—*tubular nephritis*. The changes in the tubules are chiefly degenerative—the cloudy swelling, fatty degeneration, dropsical degeneration, and necrosis (karyolysis and karyorrhexis) already mentioned. These are usually of a high grade, though one or the other may predominate in different cases, and they involve especially the epithelium of the convoluted tubules—the epithelium of the straight and collecting tubules being for the most part normal or but slightly altered. Desquamation is common, sometimes excessive. In some cases evidences of regeneration of the epithelium in the form of mitotic figures are present to a slight extent. Here and there the lumen of the tubules is more or less obstructed, in some places completely occluded and in consequence dilated, by swollen and desquamated epithelium, coagulated and finely granular albumin, fat granules, erythrocytes, leucocytes (mostly mononuclear), hyaline and other tube casts, and detritus. The changes in the glomeruli vary much, depending upon the severity of the process. In some mild cases they reveal little or no deviation from the normal. Usually, however, the blood-vessels of the glomerulus are dilated and overfilled with blood, in some cases with a disproportionately large number of leucocytes that may present fatty degeneration and fragmentation of the nuclei. The endothelium of the vascular tuft is frequently swollen and proliferating (intracapillary glomerulitis), and this, together with the very common hyaline thrombosis, gives rise to considerable obstruction to the blood current, and seriously compromises the nutrition of the tubular structures. In some cases the cavity of the capsule of Bowman contains a small amount of an albuminoid or hyaline material that is usually crescent-shaped. In more severe cases, in addition to the foregoing, both degenerative and proliferative changes in the epithelium of the capsule occur (Fig. 3063). These implicate both the epithelium of the glomerulus as well as that lining the capsule. Usually both are implicated, though the changes in the capsular epithelium may be much the less marked. The degenerative changes are similar to those that affect the tubular epithelium. The proliferation of the epithelium is sometimes so marked as to warrant the term *proliferative* or *desquamative glomerulitis*. In severe cases the capsule is filled with, and the vascular tuft is compressed by, an exudate that consists of proliferated and desquamated epithelium, coagulated albuminoid or hyaline material, cell detritus, erythrocytes, leucocytes, fat droplets, etc. (Fig. 3064). In hemorrhagic cases intracapsular extravasation of blood may be excessive. In consequence of desquamation of the glomerular epithelium and of changes in the capillary loop itself the permeability of the vessels becomes increased and the albuminous constituents of the blood find their way into the urine. In other cases, in consequence of more destructive changes the glomerulus is rendered entirely functionless. The changes in the interstitial tissue vary much in different cases.

In cases of toxic or degenerative nephritis they may be entirely or almost entirely absent. Usually, however, there are dilatation of the blood-vessels, swelling and œdema of the interstitial tissue (especially in septic cases)—whence the tubules appear more widely separated than in health—and cellular exudation (Fig. 3065). The cellular exudation consists of emigrated leucocytes (mostly mononuclear), erythrocytes, proliferated fixed connective-tissue cells, and plasma cells. In most cases this exudate is confined to the cortex through which it is distributed more or less uniformly, or, as is more frequently the case, it is collected in more or less circumscribed foci. Usually it is more marked about the interlobular veins and the stellate veins, at the inner boundary of the cortex, and about the Malpighian capsules. In severe cases, hemorrhages may occur, not only into the Malpighian capsules and the tubules, as already mentioned, but also into the interstitial tissues. These are especially common in the nephritis known as *acute hemorrhagic nephritis*.

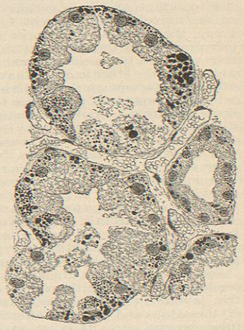

FIG. 3062.—Fatty Degeneration of the Epithelium of the Convoluted Tubules of the Kidney. The fat droplets are stained black with osmic acid. (Delafield and Prudden.)

(*c*) ACUTE INTERSTITIAL NON-SUPPURATIVE NEPHRITIS is "an acute inflammation of the kidney characterized by cellular and fluid exudation in the interstitial tissues, accompanied by, but not dependent on, degeneration of the epithelium; the exudation is not purulent in character, and the lesions may be both diffuse and focal" (Councilman). This form of nephritis, which occurs in the infective diseases, especially in scarlet fever and diphtheria, but also in typhoid fever, etc., was described first by Biermer, and later by Wagner, who gave it the name of acute lymphomatous nephritis. More recently it has been studied by Councilman, who states: "The disease is characterized by general and focal infiltration of the interstitial tissue of the kidney with cells which correspond to those which Unna has described under the name of plasma cells. The focal character of the infiltration is marked: even in cases in which all the parts of the kidney show some interstitial cellular infiltration, the cells are most abundant in certain foci. These foci are found in three places: in the boundary zone of the pyramids, in the subcapsular region of the cortex, and around the glomeruli. A considerable number of cases is found in which the blood-vessels of the boundary zone of the pyramids contain numbers of lymphoid and plasma cells without any infiltration of the interstitial tissue. The new cells in the interstitial tissues are due to emigration from the blood-vessels and multiplication by mitotic division of the cells which have emigrated. The cells can emigrate as plasma cells or as lymphoid cells, and the latter may change into plasma cells in the tissues. . . . No adequate explanation is found for the focal character of the lesions in the kidneys."

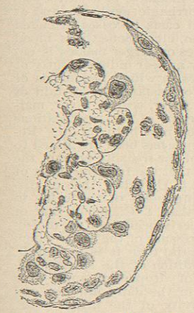

FIG. 3063.—Acute Diffuse Nephritis. Swelling of the cells covering the capillary tufts and lining Bowman's capsule. (Delafield and Prudden.)

(*d*) THE KIDNEY OF PREGNANCY is a peculiar condition of the kidneys occurring in pregnant women—a condition to be distinguished from a nephritis antecedent to the pregnancy, and a nephritis occurring in the course of pregnancy and attributable to the etiological factors already mentioned. It develops especially in the second half of pregnancy, in young primiparæ, and in twin pregnancies. Its nature is not understood. It has been ascribed to increased intra-abdominal and intrapelvic pressure exerted especially on the renal veins, on the ureters, and on the cœliac ganglion (occasioning renal anæmia in consequence of reflex contraction of the renal arteries); to bacterial infection and toxæmia; to autointoxication—the consequence of the inability of the kidneys to serve as emunctories for both the mother and the fœtus, etc. Writers are not agreed as to whether the lesions are purely degenerative or degenerative and inflammatory. The kidney varies in appearance in different cases and it is scarcely to be distinguished from some cases of acute diffuse nephritis. It is usually enlarged, its capsule strips readily, revealing a smooth, pale, usually yellowish surface. On section the cortex is somewhat swollen. Microscopically the most conspicuous lesions are retrograde changes similar to those already mentioned. Usually the lesions in the interstitial tissues are slight.

Symptoms.—Acute toxic or degenerative nephritis may run its usually short and benign course entirely devoid of symptoms and without noteworthy alterations in the urine. The general manifestations of the intoxication or infection that gives rise to the kidney lesions may be not in the least aggravated. Slight dull pain in the lumbar regions may be complained of, but the morbid condition

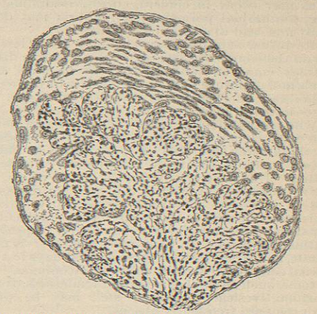

FIG. 3064.—Acute Diffuse Nephritis Following Scarlatina. Swollen cells are seen upon the capillary tuft and lining Bowman's capsule. Polyhedral and flattened cells lie in masses between the capsule and the tuft, the latter has been pressed upon by the cells and other exudate within the capsule. (Delafield and Prudden.)

of the kidneys is detected only by examining the urine. The development of slight albuminuria during the course of any of the well-known infective diseases warrants the assumption of the existence of such toxic degeneration of the renal epithelium—a condition usually recognized clinically as *febrile albuminuria*. The albuminuria, however, is probably due to the action of a toxin rather than to the fever, though this may be a contributing factor. The urine in these cases reveals the characteristics of febrile urine generally: it is diminished in amount, turbid, of high color, increased specific gravity (1.022 or more), and markedly acid in reaction; it contains an excess of solids and a small amount or merely a trace of al-

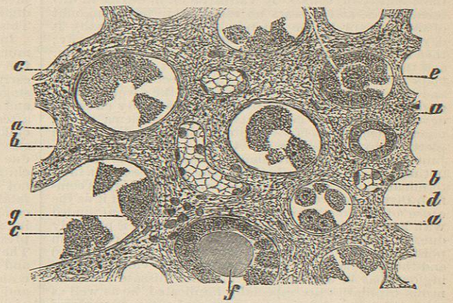

FIG. 3065.—Acute Diffuse Nephritis, with Sero-fibrinous Exudate and Catarrh of the Uriniferous Tubules (from a man who died of suppurative mediastinitis and pleuritis with nephritis on the tenth day after the beginning of the disease). *a*, Stroma distended by fluid and infiltrated with granules, filaments of fibrin, and several fat droplets; *b*, capillaries; *c*, epithelium of the convoluted tubules, in part fatty and desquamating; *d*, desquamated epithelial cells in a looped tubule; *e*, granular and fatty detritus in a looped tubule, the epithelium of which still remains but is the seat of cloudy swelling; *f*, hyaline tube cast in a convoluted tubule; *g*, round cells. ×350. (Ziegler.)

bumin; and it deposits a slight sediment that contains a few leucocytes and usually a few hyaline tube casts. With cessation of the toxic or infective process the urinary conditions revert to the normal. In severe cases, such as may occur in cholera, corrosive-sublimate poisoning, cantharidal poisoning, etc., anuria may develop speedily and lead to a fatal termination.

Acute diffuse nephritis may develop more or less insidiously in the course of some one of the infective diseases, or it may come on suddenly, as, for instance, after a debauch and exposure to the inclemencies of the weather. In either case its existence is revealed by œdema or by changes in the urine or by both. The general symptoms are by no means characteristic. If the disease develops after a debauch and exposure, there may be sudden and rather high fever (101°–103° F.), associated or not with chilliness, pain in the back, and possibly nausea and vomiting. If it develops during the course of one of the infective diseases, the fever of the infective disease may be not in the least disturbed. Frequently, however, there is some increase in the fever. In general, children, rather than adults, are likely to have fever, especially in scarlatinal nephritis. In these cases the fever is usually moderate, and it may last for from several days to a week. An initial convulsion in children is not uncommon. Local symptoms, such as lumbar pain and tenderness, frequent micturition, vesical tenesmus, etc., may be present, though they are frequently absent.

Œdema is one of the characteristic signs of acute nephritis. Though it may be detected by careful examination in some cases in which it is thought to be absent, it is not invariably present. Its absence in some of the cases of nephritis, in some of the cases of severe nephritis that occur during the course of infective diseases, such as diphtheria, typhoid fever, pneumonia, sepsis, etc., is noteworthy and suggests the wisdom of frequently examining the urine. It sometimes comes on slowly, the patient becoming gradually swollen and pale; in other cases it develops suddenly, coming on within twenty-four hours of the onset of the disease, and constituting the earliest and most obtrusive sign, especially after a debauch, in scarlet fever, etc. Usually it develops coincidentally with, rarely before, the lessening in the quantity of urine. It varies much in degree in different cases, and it bears no relationship to the amount of the urine or to the amount of albumin. Usually it develops first in the eyelids; then in the face generally; then about the ankles, in the hands, in the legs, scrotum, and the loose and areolar and dependent portions of the body generally. Finally the œdema becomes universal, and hydropsical effusions occur within the several serous cavities of the body. Rarely the serous cavity effusions develop before the cutaneous œdema. Œdema of the mucous membranes is not common, though œdema of the glottis is by no means rare. Œdema of the palate, conjunctiva, etc., is less frequent. Œdema of the lungs is not uncommon, especially if the case progresses to a fatal termination. Œdema of the meninges, which is less common than is maintained by those that look upon it as the cause of uræmia, does occur, and has been confounded with hemiplegia, etc. The œdema in general is probably due, as maintained by Senator, to the action of certain poisons on the blood-vessels (especially the capillaries) and the lymphatics. Senator supports his view by pointing out that in scarlet fever, in which disease the cutaneous capillaries are especially implicated, he as well as others has observed the occurrence of cutaneous œdema without nephritis.

The pathognomonic signs of acute nephritis are furnished by the urine, and the changes in the urine are an index of the seriousness of the lesions of the kidneys. Frequently the first sign to attract attention is diminution in the daily amount of urine. Commonly the daily amount is 300 c.c., or thereabout, though in the beginning of many cases it is less than 100 c.c. In some cases complete anuria supervenes and may lead rapidly to a fatal termination. The urine is acid in reaction, highly colored (smoky, rarely bright red—from the presence of considerable blood), turbid, and of increased specific gravity—1.024 to 1.030. In some cases, however, on account of great deficiency in the excretion of solids, the specific gravity may be rather low. Though the percentage of urea may be increased, the total quantity of urea as well of other nitrogenous substances is diminished; uric acid may be normal in amount, and the purin bases usually are increased. The urine always contains albumin, generally from 0.3 to one per cent., or 5 to 10 gm. daily. The albumin is the albumin of the blood serum—serum albumin and globulin; sometimes also nucleoalbumin (especially when a considerable number of cells are present), and rarely albumoses. Usually an abundant, sometimes hemorrhagic sediment is deposited. This consists of erythrocytes, leucocytes (mostly mononuclear), renal and bladder epithelium (single cells or cells in masses); crystals of uric acid and oxalates; hyaline, epithelial, granular, leucocyte, and blood casts; compound granule cells, cell detritus, bacteria (not always referable to extraneous contamination), etc. In different cases there is frequently a preponderance of one or another of these morphological elements, as, for instance, of erythrocytes and blood casts in acute hemorrhagic nephritis, and of renal epithelium and epithelial casts in cases attended with considerable desquamation, etc. Sometimes blood pigment in the form of granules or larger masses is encountered. This pigment may appear independently of erythrocytes, especially in cases in which hæmolysis or hæmoglobinæmia occurs, as, for instance, in certain of the infective diseases—typhoid fever, scarlet fever, malaria, yellow fever, etc.; in certain of the intoxications and following burns; and in the new-born infant. Recently some diagnostic importance has been attributed to cryoscopy, or the determining of the freezing point of the urine. Thus it has been found that the freezing point of normal urine is from 1.8° to 2.3° C. below that of distilled water, whereas the freezing point of the urine in nephritis is only 1° C., or less than 1° C., below that of distilled water. These differences depend upon the molecular concentration or osmotic pressure of the urine which is less in nephritis (hyposthenuria) than it is in health. With improvement in the condition of the kidneys, especially with the absorption of dropsy, if such were present, considerable increase in the daily amount and diminution in the specific gravity of the urine occur. This is frequently the first encouraging sign in the case.

In addition to the foregoing, few symptoms are of much diagnostic significance. The pulse, as a rule, is full and of increased tension, somewhat slowed in the early stages, and somewhat accelerated in the later stages. Hypertrophy of the left ventricle of the heart and accentuation of the aortic second sound may develop after the lapse of several (usually four to six) weeks. In children these sometimes develop unusually early. Epistaxis is a suggestive symptom in some cases. Dyspnœa, due to hydrothorax or hydropericardium, diffuse bronchitis, œdema of the lungs, or catarrhal pneumonia, is not uncommon, especially if the case progresses to a fatal termination. The appetite is poor; the bowels are confined, though sometimes there is diarrhœa; and vomiting is not rare. If vomiting persists, it is of bad augury, presaging the onset of more serious uræmic symptoms. The nephritic patient usually becomes rapidly anæmic and loses flesh, though the wasting is more or less concealed by the œdema. The blood usually is hydræmic and of lessened specific gravity; there is moderate oligocythæmia, and usually disproportionate oligochromæmia (moderate chlorotic anæmia). The leucocytes usually are normal in number, though a leucocytosis (16,000 to 22,000 per cubic millimetre), has been observed in a number of cases, especially severe cases of acute hemorrhagic nephritis, in uræmia, and in complicating infections. The blood becomes toxic, especially in uræmic conditions; increased viscosity has been observed, and according to some investigations the freezing point of the blood is higher than normally on account of the accumulation of waste products.

Uræmia is a term applied to a symptom complex, consisting largely of cerebral and gastro-intestinal symptoms, that occurs in the course of disease of the kidneys and in anuria, and that is referable to intoxication of the system with certain metabolic products that should be, and in health are, eliminated by the kidneys. The exact nature of the poison, whether there be one or several poisons, and whether the same poison or poisons are operative in all cases, have not yet been determined. The poison cannot be urea, uric acid, the potassium or sodium salts, creatinin, or certain other well-known constituents of the urine, since the injecting of each of these substances singly into the circulation does not give rise to uræmia. It has been attributed to a supposed internal secretion of the kidneys, in proof of which it has been pointed out that removal of a portion of one kidney results in considerable increase in the production of certain nitrogenous excretives. It has been attributed to œdema of the brain and meninges, but this view (Traube's) is no longer tenable. It has been attributed also to certain "urotoxins" (Bouchard), to acid intoxication (diminution in the alkalinity of the blood), to the toxæmia of the blood that has been found by Hughes and Carter, Herter, and others, to be present in uræmia, etc. All that can be said with certainty is that it is an intoxication of the system with certain metabolic products that in health are eliminated by the kidneys. These substances exert their deleterious action chiefly on the brain where they probably damage the nerve cells directly, though they may also influence the blood supply.

Uræmia is an unwelcome event that may develop in any case of nephritis. As a rule it develops only when the daily amount of urine voided is very much diminished or when there is complete anuria. In some such cases, however, uræmia does not occur, a fact compatible only with the hypothesis that the other emunctories have taken on vicarious action, or that the amount of toxic materials accumulating in the system is very small. On the other hand, uræmia sometimes develops in cases in which large amounts of urine are being voided. These cases are sometimes difficult to explain, though doubtless there has been a gradual accumulation of small amounts of the uræmic poison, and then a sudden explosion alike to that which occurs in some cases of mineral poisoning, such as mercury, lead, etc. This is not uncommon in some cases of chronic diffuse indurative nephritis (primarily contracted, or red granular kidney). In some of these cases, however, the uræmia is referable to cardiac weakness with consequent lessening of the blood pressure and diminution in the amount of urine excreted. In other cases uræmia is coincident with the absorption of œdema.

Clinically uræmia may be acute or chronic. The acute form is especially common in acute and chronic diffuse non-indurative nephritis, whereas the chronic form is especially common in chronic diffuse indurative nephritis. Acute uræmia, usually preceded by certain subacute or chronic prodromes, such as headache, insomnia, breathlessness, restlessness, etc., comes on suddenly, lasts but a short time, and frequently terminates fatally. Chronic uræmia, on the other hand, is more insidious in its onset and more mild in its manifestations; it may last for a comparatively long time, disappear, and subsequently recur. The uræmia that occurs in obstruction of the ureters and that sometimes differs in its manifestations from the ordinary cases of uræmia, has been described as "latent uræmia."

The characteristic symptoms of acute uræmia are nervous in nature and consist of convulsions, coma, psychic derangements, etc. The convulsions resemble a true epileptic fit, and they are frequently exceedingly severe (uræmic eclampsia). They may or may not be preceded by prodromes; the initial cry of true epilepsy is usually absent. The convulsions may be local or unilateral, and finally general. Aside from the coma that attends the convulsions, there is a coma that is quite independent of convulsions. It usually comes on insidiously, being preceded by certain less serious uræmic manifestations, such as headache, apathy, dyspnœa, muscular twitchings, etc. Muscular twitchings are also common during the persistence of the coma, which may last for days, even weeks, and eventually disappear. Usually, however, the patient passes into a typhoid state and finally dies. The psychic derangements are melancholia, delusional insanity, mania which may develop suddenly, become exceedingly wild, and lead rapidly to a fatal termination, etc. In some cases the fatal termination is ushered in with a hemiplegia or a monoplegia that at the necropsy may be found to be due, not to intracerebral hemorrhage, but solely to œdema of the meninges and anæmia of the brain. The milder and chronic manifestations of uræmia, which frequently exist for a few days, in some cases even for years before the onset of the severer acute symptoms, consist of headache (often occipital), hemicrania, vertigo, tinnitus aurium, insomnia, a feeling of anxiety, muscular twitchings, languor, neurasthenia, pains in the joints or muscles, nocturnal cramps in the calves, etc. Amaurosis is not uncommon. It may or may not be associated with convulsions, though it usually follows convulsions. It is attributable to disorder of the cerebral visual centres. Usually it subsides after several days. Deafness and aphasia are rather rare. Uræmic dyspnœa or renal asthma is one of the most suggestive manifestations of chronic uræmia. It is frequently one of the earliest obtrusive symptoms of chronic nephritis, and it should always suggest the wisdom of examining the urine. It may be continuous, when it should always attract attention; it may be paroxysmal, coming on especially at night; and it may manifest itself by Cheyne-Stokes breathing. It is usually a manifestation of toxæmia, though it may be due to, or augmented by, hydrothorax or hydropericardium, diffuse bronchitis, œdema of the lungs, catarrhal pneumonia, etc. Persistent and uncontrollable vomiting, usually associated with nausea, is especially suggestive of chronic nephritis. It may be of centric origin the result of toxic irritation of the medullary centres, or it may be the result of gastric irritability produced by the excretion by the gastric mucosa of urea and its decomposition into ammonia. Profuse diarrhœa, associated with catarrhal and ulcerative enteritis and colitis, is rather common. Stomatitis, attributed to the elimination of urea by the saliva and the sputum, is observed rather frequently. In uræmia the breath frequently has a urinous or an ammoniacal odor, and crystals of urea eliminated by the sweat glands may be observed on the skin, especially about the normal folds where they sometimes lead to eczema. The pulse usually is slow and of increased tension, but with the supervention of convulsions it becomes small, rapid, and feeble. The temperature is usually subnormal, though a uræmic fever, occurring apart from the fever generally present during the convulsions, as well as uræmic chills, has been described.

Diagnosis.—The diagnosis of acute nephritis, frequently suggested by the history of the case and the occurrence of œdema, is based upon the results of an examination of the urine. Frequent examinations of the urine, therefore, should be made in all cases of disease, but especially in all cases likely to be attended by nephritis, as for instance, all the infective diseases, intoxications, and during pregnancy. The urine of all patients presenting pallor of the skin and even slight swelling of the eyelids should also be examined, and one should bear in mind that the first manifestation of nephritis may be a uræmic convulsion or coma. In all cases of convulsion and of coma, therefore, the urine should receive immediate attention, and catheterization, if necessary, should be performed at once. Whether the kidney lesion be an acute nephritis or an acute exacerbation of a chronic nephritis may be determined in part by the history of the case, and in part by examining the urinary sediment. From the sediment also one may glean an idea of the nature and severity of the renal lesions. Acute interstitial non-suppurative nephritis is not recognizable clinically; that is, the symptoms to which it gives rise do not differ from the symptoms of acute diffuse nephritis.

Course and Prognosis.—Acute toxic or degenerative nephritis occurring in the course of some infective disease usually runs a mild course and subsides with the in-

fective disease in question. In some cases, however, especially in cholera, yellow fever, some cases of typhoid fever, etc., as well as in certain intoxications, such as corrosive-sublimate poisoning, phosphorus poisoning, etc., it either leads directly to the death of the patient (possibly by producing anuria), or it contributes to the fatal termination. Acute diffuse nephritis of mild or moderate severity usually runs its course in from several days to four or six weeks. Probably more than one-half of the patients recover; the remainder die or the kidney lesions progress to chronic diffuse non-indurative nephritis. The termination depends in great measure upon the cause of the nephritis and upon the severity of the lesions. In favorable cases improvement is usually manifest at the end of ten days. In all cases the prognosis is anxious, and it is materially influenced by the occurrence of complications, such as secondary infections, internal dropsies, inflammations of the serous membranes, catarrhal pneumonia, œdema of the lungs, etc. The prognosis is very bad in cases with extreme anasarca or marked uræmia that does not respond promptly to treatment—though both of these conditions may be followed by recovery. There is always danger of a fatal termination in the kidney of pregnancy, but recovery ensues in the majority of cases when delivery has been effected.

Treatment.—The natural history of acute nephritis furnishes clear indications for treatment, the principal of which are: (1) To secure temporary physiological rest for the kidneys, and to guard against the dangers that threaten from accumulation of waste products in the system, by promoting the activity of the other emunctories; (2) to lessen the inflammatory phenomena in the kidneys; and (3) to meet the symptomatic indications, such as uræmia, pulmonary œdema, hydrothorax, etc., as they arise.

The prophylaxis deserves some consideration. It is said that an attack of acute nephritis, such, for instance, as follows scarlet fever and other infective diseases, and the imminence of which is manifest by hæmaturia and increased arterial tension, may be averted by a brisk purgative—a full dose of a saline cathartic or of compound jalap powder. In other cases, cases in which it is known that acute nephritis may develop, such as the acute infective diseases, etc., measures should be adopted early to guard against the occurrence of the renal complication. These comprise the avoiding of exposure to weather inclemencies, the using of suitable personal and bed clothing, the prohibiting of irritating food and drink, the promoting of the functional activity of the other emunctories (the skin and the intestines), the diluting of the toxic material in the body by the use of large quantities of water, etc.

In most cases, however, we have to deal with the developed disease. Our first endeavor should be to secure for the patient rest, warmth, and a suitable diet, under the beneficent influence of which many of the milder cases subside. In all cases, even the very mild cases, absolute rest in bed is imperative. The rest must be prolonged until all indications of the disease have disappeared. It is advisable that the patient wear light flannels and that he rest between blankets: the flannels promote the functional activity of the skin and the absence of sheets lessens the risk of chill. The diet should be as non-nitrogenous as possible and as small in amount as is compatible with preserving the strength of the patient. Milk is the most suitable diet and should constitute the sole diet for some days at least; in case it be well borne and the patient do not object too strenuously no other diet may be given for some weeks. The milk may be given warm, if relished so. Whole milk may be given, or it may be diluted one-third with lime water (which serves to decrease the acidity of the urine), or with hot water, or some carbonated water, or some thin oatmeal gruel. Its (to some people unpleasant) taste may be disguised by the addition of a small amount of strong black coffee, a little salt, or a little extract of vanilla. As occasion requires, buttermilk or skimmed milk may be substituted in whole or in part. The milk should be given in small amounts at frequent intervals rather than in large amounts at longer intervals. The free use of bland fluids, such as ordinary water, is extremely serviceable; they serve to allay thirst and to flush the kidneys. Sodium bicarbonate, potassium citrate, or sodium benzoate, in the proportion of five to ten grains to the ounce, may be added to the water. A pleasant drink consists of a drachm of cream of tartar and the juice of half a lemon added to a pint of boiling water, and used when it has cooled. Weak lemonade and thin arrowroot also are permissible.

Local depletive measures are of some service, tending as they do to relieve the engorgement of the kidneys. They should not be neglected in cases that begin with much local distress, hæmaturia, or marked suppression of urine. Probably the most serviceable measure is free dry cupping (a dozen or more cups). They should not be allowed to remain until stagnation of the blood in the capillaries has occurred, as the object of their use may be thus defeated. They may be followed by hot linseed poultices which should be frequently renewed before they have cooled. The poultices may be used instead of the cups as occasion seems to warrant. In case the nurse is not trustworthy it is well to substitute a hot woollen jacket for the poultices, as the risk of chilling is thus reduced. In some cases the Paquelin cautery will be found of service.

Temporary and partial physiological rest for the kidneys is secured, and the dangers that threaten from the accumulation of waste products in the blood are counteracted, in part at least, by promoting the functional activity of the skin and the intestinal tract, by the use of diaphoretics and of purgatives. The activity of the skin is best promoted by hydrotherapy—by the hot-water bath, the hot-air bath, the hot-vapor bath, and the hot wet pack. In adults any one of these forms of hydrotherapy may be employed with confidence, though probably the hot-water bath or the hot wet pack are the easier of application. In children the hot wet pack is usually serviceable. The sweating induced by these measures is usually profuse and non-exhausting. In cases in which the first use of these hydrotherapeutic measures is unattended by the desired sweating, the procedure should be repeated in a short time. In many cases the sweating is induced or augmented by the concurrent administration to the patient of a hot drink or of a diaphoretic mixture, such, for instance, as contains spirits of nitrous ether, solution of the acetate of ammonium, etc. A Dover's powder may answer the same purpose. In urgent cases, pilocarpine, alone or in conjunction with hydrotherapeutic measures, should be used. The pilocarpine may be given in doses of gr. $\frac{1}{8}$ or gr. $\frac{1}{6}$ hypodermically, and repeated in half an hour, if necessary. Caution, however, is necessary in its employment; no more should be given than is required, and it should be avoided in children. Its depressing effect, as well as the depression that sometimes attends the use of hydrotherapeutic measures, calls for the administration of stimulants. In serious cases the bath should be repeated daily; and less frequently in less serious cases, and as convalescence becomes established. In some cases gentle and continuous sweating is effected by the administration of fluid extract of jaborandi, ℥x. to xv. to an adult, every two to four hours.

Free purgation is called for in all cases—whether or not constipation exists. At the outset of the treatment, it is generally well to administer sufficient of a concentrated solution of some saline cathartic to secure several watery evacuations. In cases with gastric irritability, the effervescing preparations will be found of service. Compound jalap powder (gr. xxx.) may be substituted for the saline. In many cases calomel is found to be a very efficient cathartic, but it is well to avoid it in the early stages of severe attacks since, on account of its diuretic action, it tends to increase the already existing congestion of the kidneys.

The question of the use of diuretics is still debated. However, stimulating diuretics, since they act by producing congestion of the kidneys, are assuredly contraindicated in the early stages of a severe attack of acute nephritis. The very best diuretic is water, which, merely flushing the kidneys, facilitates the removal of the products of inflammation and desquamation from the kidneys, reduces the acidity of the highly acid and irritating urine, and dilutes the toxins present in the blood. The alkaline diuretics already mentioned may often be employed with hope of good result. The more stimulating diuretics, such as digitalis, strophanthus, caffeine, cocaine, etc., which increase the blood pressure, should be reserved for a later date when they may render very valuable service. In many cases when the blood pressure is low and the heart action is weak, the happiest results are obtained by the combination of digitalis (preferably the infusion) with the alkaline diuretics. Diuretin and theobromine also may be found of service. The iodide of potassium has been recommended.

With the onset of uræmia treatment must be energetic. Inasmuch as in many cases of uræmia the blood pressure is increased the first indication is to bleed the patient. The extraction of from ten to twenty ounces of blood from the arm of the patient removes a considerable quantity of the poison, and the subsequent injection of a pint or two of decinormal saline solution serves to dilute the poison that remains. Serviceable in all cases, these two procedures should not be delayed in the event of convulsions or coma. If the convulsions are violent they should be controlled by the inhalation of chloroform. The convulsions that attend *acute* nephritis may often be satisfactorily controlled by the hypodermic injection of a quarter of a grain of morphine—a method of treatment successfully employed for many years by the late Professor Loomis. Free and immediate evacuation of the bowels must be secured, preferably by the use of one or two drops of croton oil, diluted or not with a little olive oil or glycerin, placed on the back of the tongue, or by the administration of a quarter of a grain of elaterium in solution. At the same time a stimulating enema should be given. Profuse perspiration should be induced as soon as possible, preferably by the use of a hot wet pack or a hot vapor bath and by the hypodermic injection of pilocarpine. After the thorough evacuation of the bowels, if the convulsions have not ceased, they may be controlled by the rectal administration of chloral (gr. xxx.–xl.) and potassium bromide (ʒ i.). In some cases imminent uræmia, due to cardiac insufficiency, may be warded off by the free use of cardiac stimulants, such as digitalis and strychnine, diffusible stimulants, such as camphor (hypodermically), hypodermoclysis, and a calomel or saline purge.

Certain special symptoms sometimes demand attention. Thus vomiting and diarrhœa may be marked. Generally manifestations of uræmia, they often subserve a useful purpose, and should not always be unceremoniously stopped. When vomiting gives rise to marked weakness, small bits of cracked ice or sips of very hot water may be given. In addition counter-irritation to the epigastrium and the internal administration of bismuth, creosote, and lime water, or dilute hydrocyanic acid, or carbolic acid, etc., may be tried. Extreme dropsy that does not yield to the measures already detailed may necessitate small incisions in the skin or the use of Southey's tubes. Dropsy of the serous cavities may require tappings. Headache not relieved by general depletive measures may demand the use of bromides, etc. Pulmonary œdema, catarrhal pneumonia, and other complications are to be treated on general principles.

During convalescence extreme care must be exercised to avoid chill and a possible relapse. The increase of diet must be gradual—first farinaceous foods may be permitted, then light vegetables, non-acid fruits, and finally light meats, etc. Flannels should be worn next the skin, and the resumption of exercise must be gradual. In combating the anæmia and weakness so common during convalescence iron is useful, but it should not be employed until all acute manifestations have subsided. The liquor ferri et ammonii acetatis (Basham's mixture) enjoys a well-deserved popularity, and may be combined with strychnine. The limitations of our therapeutic resources are sometimes only too apparent from our inability to control the single obtrusive symptom of the disease that may remain—the albuminuria. Lactate of strontium, which has been recommended for this, continues to merit trial. Residence in a warm, somewhat humid, equable climate is to be recommended to those able to avail themselves of its advantages.

II. Chronic Nephritis.

(*a*) Chronic Diffuse Non-Indurative Nephritis.—*Etiology and Pathogenesis.*—In many respects no sharp line of demarcation can be drawn between acute nephritis and chronic diffuse non-indurative nephritis, on the one hand, and between chronic diffuse non-indurative nephritis and chronic diffuse indurative nephritis, on the other hand. In many cases they doubtless represent different stages of the same process. However, chronic diffuse non-indurative nephritis is most common in young male adults between the ages of twenty and forty years; but it is not rare in women, and in children, especially after scarlet fever. In many instances it follows acute nephritis, so that its etiology and pathogenesis are not to be divorced from the etiology and pathogenesis of acute nephritis. Probably, however, in the majority of cases it develops insidiously, subacutely or chronically, being the expression of the action of toxic or infective agencies insufficient in amount or virulence or both to give rise to acute nephritis. Malaria, for instance, is more likely to result in this chronic non-indurative nephritis than it is in acute nephritis. The disease is also attributable to continued exposure to the inclemencies of the weather, to insanitary surroundings, to over-indulgence in alcoholic beverages, etc. It is common in tuberculosis, syphilis, and prolonged suppuration, in which cases it is frequently associated with amyloid disease of the kidneys and of other organs. It occurs also in chronic heart disease apart from the cases of cyanotic induration. Though it runs an essentially chronic course, acute exacerbations are common.

Pathological Anatomy.—In nature the anatomical lesions in acute and in chronic diffuse non-indurative nephritis are alike; they differ only in intensity and in the presence, in the chronic nephritis, of certain secondary lesions the development of which requires time. As in the acute process, so also in the chronic process, the kidneys present quite different appearances, depending upon the conspicuousness or subordinateness of certain lesions. Usually two kinds of kidneys are described—the large white (more properly yellow) kidney and the large red, mottled, or variegated kidney. The *large white kidney*, as the name implies, is at least as large as a normal kidney; frequently it is considerably enlarged. The capsule is thinned and non-adherent. The surface is smooth, pale, yellowish or mottled grayish-yellow or reddish-yellow in color. The stellate veins are usually more or less distended and some foci of hemorrhage may be apparent. On section the cortex is swollen, pale, yellowish or reddish-yellow in color, somewhat opaque, sometimes distinctly fatty, and diminished in consistency. Usually the pyramids are slightly congested, or at least darker in color than the cortex. The *large red, mottled, or variegated kidney*, the kidney of *chronic hemorrhagic nephritis*, though enlarged, scarcely attains the extremes of size common to the large white kidney. In many respects the kidney resembles the kidney of acute nephritis, being, however, much firmer. The capsule is thinned and in general non-adherent. The surface is smooth, grayish-red or very dark reddish-brown or hemorrhagic in color. Frequently it is mottled—irregularly shaped, pale, yellowish, anæmic, and fatty areas commingling with darker reddish-brown and hemorrhagic areas. On section the cortex is swollen, grayish-red in color, or mottled and striated, and darker in color. The pyramids usually are congested. Sometimes the organ is more or less translucent on account of associated amyloid disease.