

ent of the blood, and that not more of it is found in the blood of animals from which the kidneys have been removed than in the normal state. Treitz enlarged Frerichs's ammonia theory so that it included more facts, but it is not satisfactory. Treitz held that carbonate of ammonia is the toxic agent, but maintained that urea, excreted by the mucous membrane of the intestinal canal, is there decomposed, and diffused thence into the blood. Schottin next proposed that uræmia is due to the retained extractives, creatine, creatinine, etc., which are products of tissue metamorphosis, that later in the process of hystolytic transformations are converted into urea. Although these substances may be noxious when retained, the theory which restricts the phenomena of uræmia to their retention is insufficient in scope. Traube has also propounded a theory, in which doubtless there is a measure of truth, since it is supported by the only lesion which is really referable to the condition of uræmic intoxication, namely, œdema of the brain. By reason of the watery state of the blood, transudations of serum take place through the cerebral capillaries, producing œdema of the brain. With the increase of fluid in the perivascular lymph-spaces, more and more pressure is exerted on the intra-cranial vessels, so that ultimately sufficient anæmia is produced to cause convulsions. The existence of more or less œdema of the brain is doubtless true, and this facilitates the action of the toxic agents, while it is in part a result of their action. The last theory to be referred to is that of Feltz and Ritter, which ascribes uræmia to the toxic action of the potash salts of the urine, retained in the blood. Unquestionably the nervous and other symptoms of uræmia may be produced by injecting the potash salts into the veins. But, while this statement is admitted, it may also be affirmed that not to one, but to all of the retained constituents of the urine possessed of toxic activity, may be referred the phenomena of uræmia. When there are several capable of producing disturbance of this kind, it were wiser to include them all, and refer the condition of uræmia to the retention of the toxic urinary constituents.

**Symptoms.**—Uræmia may be *acute* or *chronic*: the former occurring in those renal affections characterized by a sudden great diminution or suppression of the urine; the latter belonging to the chronic affections, such as chronic interstitial nephritis, in which the quantity of urinary solids is slowly reduced. Acute uræmia consists in sudden violent headache, vertigo, disordered vision, twitchings of the facial and other muscles, followed by general convulsions of an epileptiform character. The convulsions may recur every few minutes or hours, and in the interval there may be coma, the condition of insensibility persisting until death, or until the renal functions or vicarious discharges are reëstablished. The convulsions may exist without loss of consciousness, and in some cases assume a tetanic rather

than an epileptic type. The acute form may also occur without convulsions, the patient passing from a state of somnolence into profound coma, in which the face is pallid; the pupils are dilated, but react to a strong light; the pulse is slow; the respirations are shallow, often irregular (Cheyne-Stokes), and sometimes stertorous; and there is general muscular resolution, but not localized paralysis. From this condition the patient may emerge, some hebetude of mind and diminution in general and special sensibility remaining for some minutes or hours, and then recurrences of the coma and periods of improvement until the end. In other cases this comatose form of acute uræmia is varied by attacks of a convulsive character or by a mild delirium.

Acute uræmia assumes still other forms, which, however, are extremely rare. There is a delirious form, in which the mental trouble assumes the shape of a tranquil delirium, less often of acute mania, and is preceded by headache, disorders of vision, by a listless apathy, dullness of apprehension, and weakness of will. There is a dyspnoic form, characterized by the sudden onset of intense dyspnoea without any change in the respiratory organs, but accompanied by a hoarse voice and sibilant inspiration, and terminating in a fatal coma.

The chronic form of uræmia is characterized at the outset by symptoms referable to the digestive organs—dyspepsia, nausea, and vomiting, without apparent cause. With these stomachal symptoms are associated headache, vertigo, and dimness of vision. The headache is very intense and persistent, and is often frontal, and accompanied by a band-like feeling. Early changes occur in the retina—a form of retinitis, known as retinitis albuminurica—which is often diagnosed by the ophthalmologist before the urine has been examined. Drowsiness is soon experienced. At first the patient falls asleep early in the evening, but after a time he dozes at all times in the day whenever his attention is not attracted by objects of interest. All of his special senses become dull, but vision suffers chiefly, hemipopia and diplopia being the most usual forms of derangement. Meanwhile the nausea and vomiting increase, the headache becomes more severe, vision grows more obscure, and presently muscular cramps occur at night, and twitchings of the facial muscles are observed at various times. Then develop the symptoms already described under the acute form.

**Course, Duration, and Termination.**—The progress made by a case of uræmia depends on the changes occurring in the kidneys. In a fatal case of acute Bright's disease, the duration of the uræmia in the severe form will not be greater than from three to five days. There are rapid cases, which terminate fatally in a few hours. The chronic form may occupy weeks or months. In the acute forms of Bright's disease opinions in regard to the termination of uræmia must be expressed with caution, since, under the proper treatment, very formida-

ble symptoms may disappear and health be restored. This statement is especially true of scarlatinal and pregnancy albuminuria. In chronic interstitial nephritis, in amyloid disease, and other fatal diseases, the appearance of uræmia is significant of a fatal termination. The chronic form of uræmia is, therefore, more serious than the acute.

**Diagnosis.**—The state of the urinary secretion is the first element in making a diagnosis. Is the secretion suppressed? Does the urine contain albumen or morphotic constituents, indicating structural changes in the kidneys? Albumen and casts wanting, does an analysis reveal a notable diminution in the solids of the urine? As regards the cerebral symptoms of uræmia, two points are especially significant: the absence of motor paralysis and of fever, which prove that meningitis, hæmorrhage, encephalitis, etc., are not the causes of the disturbance. That opium, belladonna, strychnine, and other narcotic poisons are not concerned in producing the phenomena of uræmia, is evident from the history of the case and the state of the urine. In every case of convulsions or sudden insensibility, not explainable by known conditions, if the urine has not been preserved, the catheter should be promptly used. If unconsciousness has been produced by a narcotic poison, the nature of it may sometimes be determined by injecting some urine under the skin of an animal.

**Treatment.**—As uræmia is due to the retention in the blood of excrementitious matters which ought to have been separated by the kidneys, obviously some mode of vicarious relief becomes necessary. By the skin and intestinal canal, excretion may be in considerable part effected. By the vapor-bath, and pilocarpine subcutaneously, the skin may be efficiently acted on. When the heart is weak, pilocarpine must be cautiously used; also, in case there be much bronchial mucus and weak respiratory muscles, this agent may not be safe. When no contraindication exists to its employment, there can be no question in regard to the good effects produced by it. As urea and the product of its decomposition—carbonate of ammonium—is eliminated by the gastro-intestinal mucous membrane, when excretion by the kidneys is stopped, we may imitate the method of nature, and free the blood of excrementitious matters by purging. For this purpose the compound jalap powder, podophyllin, calomel (cautiously), may be used. The kidneys may be stimulated to better action, when not too far gone, by the administration of saline diuretics, infusion of digitalis, etc. When convulsions occur, more prompt means of relief become necessary. The inhalation of chloroform is an excellent expedient, and is free from danger, if judiciously conducted. The hypodermatic injection of morphine, as proposed by Loomis, is a valuable expedient, if it may seem rather heroic. He advises the injection at once of a half-grain of morphine for an adult, and this is repeated two or three times if necessary. The author has seen cases yield to this which

continued under chloroform inhalations. This method is adapted to the cases of acute uræmia. Morphine thus administered seems to antagonize the condition of the cerebral vessels induced by uræmia, removes the œdema of the brain, and starts the kidneys secreting again.

#### CONGESTION OF THE KIDNEYS—ACTIVE.

**Definition.**—Hyperæmia of the kidneys signifies an increased amount of blood in the organs. The hyperæmia may be in the arterial supply—*active congestion*, or in the venous supply—*passive congestion*.

**Causes.**—*Active congestion* is usually caused by some irritating substance which is eliminated by the urine. Various medicinal agents, containing an essential oil, or a camphor, as copaiba, cubeb, eucalyptol, etc., excite irritation in the kidneys, as these substances pass through in the process of elimination. Turpentine and cantharides are among the most active of these agents, and more frequently cause acute congestion than any other. A mustard-plaster may also have the same effect, due doubtless to the absorption and elimination of the oil of mustard. An extensive burn, a counter-irritant affecting a considerable extent of surface, and possibly other injuries or impressions on peripheral nerves, may induce a reflex paresis of the arterioles of the kidneys.

**Symptoms.**—More or less pain, sometimes very acute pain, is felt in the region of the kidneys, and extends downward along the course of the ureters, into the hips, through the bladder, which becomes very irritable, and into the testicles and penis. There is present an incessant and very pressing desire to pass water, which is high-colored, and rather scanty each emission, although in the aggregate up to the normal. The urine may contain blood, or but a few red globules, or simply fibrin and casts, some cells of renal epithelium and albumin. If the action of the cause continue, the state of hyperæmia will pass over into some of the forms of inflammation. The author is convinced that the persistent use of copaiba has kept up an hyperæmia, out of which has developed the chronic form of Bright's disease. If the agent producing the hyperæmia is withdrawn, irritation subsides in two or three days, and health is restored.

The only *treatment* required in the mildest cases is to withdraw the irritating agent, to dilute the urine by the free administration of lemonade, or Vichy water, or Bethesda water. If there are decided irritability of the bladder and much pain, relief is quickly afforded by the administration of two or three grains of camphor every four hours, or still more promptly and efficiently by the hypodermatic injection of one twelfth of a grain of morphine, or by the stomach administration of one sixth to one fourth of a grain.

**CONGESTION OF THE KIDNEYS—PASSIVE.**

**Causes.**—*Passive congestion* of the kidneys is caused by venous stasis. The chief lesions inducing venous stasis are obstruction and regurgitation of the mitral orifice, obstructive diseases of the lungs, obstruction and regurgitation at the tricuspid orifice, compression of the ascending vena cava above the renal veins, and thrombosis of the renal veins.

**Pathological Anatomy.**—The vessels are abnormally full, and hence the organ is larger, and more blood flows out on section. As there is a moister state of the organ, owing to mechanical effusion from the swollen veins, the capsule is easily detached. The parenchyma of the organ is darker, having a bluish aspect; it is moist and smooth; the glomeruli are not swollen and congested, but the vessels of the convoluted tubes are distended. The stellate vessels of the surface can be traced with the eye into the anastomoses of the interfascicular veins, and the vessels of the vasa recta are recognized as dark reddish striations (Rindfleisch). If hyperæmia becomes chronic, the over-supply of venous blood leads to important nutritional alterations—to hyperplasia of the connective tissue—and hence the whole organ increases in size, firmness, and weight.

**Symptoms.**—In cases of passive congestion of the kidneys, the central disorder quite masks the changes occurring in the kidneys. When dropsy occurs, attention is directed to the state of the urinary secretion, but previously no symptoms had arisen indicating that the kidney was suffering. Besides the venous stasis and increased pressure in the venous system, the disturbance in the urinary function is in part due to the diminished pressure in the arterial system. The urine is scanty, dark in color, and acid in reaction. On standing, a very abundant deposit of urates takes place, and the urine becomes thick. The specific gravity is increased in the ratio of the decrease in the urinary water, and is 1025 to 1035, but it is also high because of the quantity of solids, uric acid, notably of urea, which may rise to five per cent., or higher. An important change now is apparent in the composition of the urine—it contains more or less albumin, but not often any considerable amount. If such urine, thick and dark, is placed in a test-tube and gently heated, it will soon clear up, except some fine particles, but gradually, the heat continued, the clear urine will become milky, from the coagulation of albumin. The urates dissolve at the temperature below the coagulating point of albumin. On microscopic examination the morphotic elements present in the urine consist of a few red blood-globules, some tubular epithelium, and a few delicate, transparent casts. The amount of albumin present in such urine does not often exceed one per cent.

**Course, Duration, and Termination.**—The kidney complication in

cardiac and pulmonary obstructive disease follows the fortunes of the central lesion. When the cardiac lesion is compensated, and the pressure rises in the arterial and falls in the venous system, the congestion of the veins and the ischæmia of the arteries of the kidneys will cease—the urinary water will increase, and the albumin will disappear. If, however, the central lesions be permanent, the condition of the kidney will grow worse, the albumin increase, and, after a time, the specific gravity will fall. Cerebral symptoms do not arise from venous congestion of the kidney, because the tubular epithelium remains sound and whole, and therefore equal to its function of excreting excrementitious materials. Death may occur from some intercurrent malady, or the patient die exhausted from the persistent dropsical accumulation.

**Treatment.**—The management of passive congestion of the kidneys is that of the central lesion. It includes the use of digitalis, quinine, and iron, of hydragogue cathartics, of warm baths, vapor-baths, and pilocarpus, caffeine, convallaria, nitro-glycerine, of diuretics, etc. The condition of the kidneys is improved by those remedies which affect the heart trouble favorably. The account already given of the treatment of cardiac disease with dropsy is equally applicable here.

**ACUTE PARENCHYMATOUS OR CROUPOUS NEPHRITIS.**

**Definition.**—Under the head of "Bright's Disease" there are included several acute and chronic affections of the kidneys, which agree in the one important characteristic of the urine containing albumin. According to many authorities, acute parenchymatous nephritis is the first stage of Bright's disease: it is "the large, white kidney," "the large, smooth kidney" of English authors, and corresponds to Johnson's "acute desquamative nephritis." Although Charcot adopts the term "parenchymatous nephritis," he holds that we are not yet prepared to name it accurately.\* By Bartels it is designated "acute parenchymatous nephritis"; by Millard, "croupous nephritis"; the latter is the more happy term.

**Causes.**—To this form of nephritis youths are more liable than the aged. An exception to this exists in infants, and the liability continues till middle life, and, indeed, though greatly diminished, does not entirely cease after this period. Heredity appears to have an influence, although the facts are not numerous. Type of constitution seems very important among the causes. The pale, light-haired, full but flabby subjects of the albuminous type seem to have a special susceptibility to this form of nephritis. Those substances which cause active hyperæmia of the kidneys, as cantharides, turpentine, copaiba, etc., will induce inflammation of these organs, if they continue in action for a sufficient time. Scarlatina is probably the most common

\* On "Bright's Disease," translated by Millard. New York: William Wood & Co.

cause. It is not the character of the epidemic, nor the severity of the attack itself, which wholly determines the changes in the kidneys, for the mildest epidemics and the least pronounced cases may be remarkable for the extent of the renal complication; yet, if the epidemic have a malignant aspect, there will be more formidable cases of nephritis. As not all cases of scarlatina are accompanied by the renal disease, there must be some inherent bodily condition, or peculiarity in the structure of the kidneys, to account for the result. The same is true of diphtheria, in which an inflammation of the kidneys occurs in a proportion of the cases. But in diphtheria there seems to be a relation between the severity of the systemic poisoning and the occurrence of the renal complication. Oertel maintains that the disease of the kidneys is due to the transference to these organs of "bacterian colonies" and their subsequent multiplication. In diphtheria, more than in scarlet fever, there may be albumin in the urine, without recognizable changes in the structure of the kidneys. In analogous morbid states acute parenchymatous nephritis may be produced. These are typhoid, erysipelas, malignant pustule, etc.—diseases due to the reception and development of some specific infective material which, eliminated by the kidneys, excites inflammation in passing through these organs. The skin and kidneys stand in intimate functional relation to each other, and when one is inactive the other may act vicariously in its stead. This physiological fact has a corresponding pathological relation. Acute nephritis may be excited by exposure of the body to cold when the skin is warm and perspiring. The sudden arrest of the skin secretion throws a greatly increased labor on the kidneys; their vessels dilate, and an acute hyperæmia prepares the way for inflammation. Pregnancy is a cause of acute parenchymatous nephritis. Usually, but not invariably, it is the first pregnancy, and it is more common in twin pregnancies. It occurs in the thin, in the robust and plethoric, in those of low and high degree, and under the most varying conditions. Having occurred in one pregnancy it may happen again, and not unfrequently becomes a permanent malady pursuing a course independently of pregnancy. No satisfactory explanation has thus far been offered. That it occurs not more frequently than one time in one hundred and fifty pregnancies renders it probable that there must exist a renal or constitutional disposition which pregnancy excites into activity.

**Pathological Anatomy.**—The changes in the structure of the kidney in acute parenchymatous nephritis are much disputed. To render clear the form of the disease under consideration, it may be repeated that it is the large, pale, and smooth kidney of the English writers. It is increased in size, so that it may reach twice its normal weight and volume; the cortex is pale, grayish-white, or a dull white; it is smooth, because when the capsule is stripped off there are no pits or

elevations as occur in the contracted kidney, and its texture is rather soft. There is but little hyperæmia of the cortex; here and there dark-red points are seen, or punctiform extravasations; but the pyramids are deeply congested, bluish red, or brighter red, and contrast strongly with the pale gray of the cortex. In other cases, according to Bartels, the cortex may not be so pale, may be reddish gray in consequence of a considerable hyperæmia, and there may be between this amount of congestion and the dead-white a great deal of variation.

The changes ascertained on microscopical examination are found "localized almost exclusively in the convoluted tubes" (Charcot), and consist in cloudy swelling of the epithelium, which remains *in situ*. The change in the appearance of the epithelium—the cloudiness—is due to the deposit of fine granulations, and in such large numbers that the lumen of the canal is almost closed by the distention of the epithelial cells. The ends of the tubules are also sometimes blocked by the deposit of fibrin-plugs. The convoluted tubes also become dilated and varicose by reason of changes taking place in the proper tunics of these tubes. The appearance of the kidney thus affected may be changed by localized or extensive fatty metamorphosis—by fatty change limited to a few tubes here and there, or by a general fatty change. When thus altered the color becomes yellowish, and, if localized, gives to the organ a granular appearance, and hence the name applied to it by Johnson as the *fatty granular kidney*; if general, it becomes the *large fatty kidney*. It has been much disputed whether the large, smooth kidney ever undergoes an atrophic change. It is held by Charcot that in very rare instances an atrophy may be effected by the liquefaction and disappearance of the fatty epithelium and the subsequent collapse of the tubules.

**Symptoms.**—When parenchymatous nephritis occurs during the course of scarlet fever, diphtheria, and other febrile diseases, the symptoms are modified in various respects. Two modes of onset are described when the disease occurs independently—one sudden, with high fever, aching pains in the lumbar region; the other slow, obscure, and with little disturbance. The first variety usually results from taking cold; the patient, while heated and perspiring, plunges into cold water or lies upon the damp ground, and in a short time—twelve to twenty-four hours—has some chilliness, even a rigor, followed by high fever, intense headache, pains in the lumbar region and through the limbs, nausea, vomiting, and anorexia. The symptoms which attract attention to the kidneys in either mode of onset are the changes in the character of the urine. In some cases the first symptom referable to the urinary organs is an extremely irritable state of the bladder, frequent desire to micturate; a few drops only, and these it may be bloody, can be passed. This symptom does not last long, and is not common. Usually there are observed changes in the quantity of the urine, the

amount passed in twenty-four hours being variously reduced from forty ounces, the normal quantity for an adult, to twenty, ten, even five ounces, and at the same time important new constituents appear in the secretion. There may occur entire suppression, when the most formidable symptoms will arise, and death result in a few days. The urine at the onset often contains blood, when it presents various appearances according to the quantity present: it may have a faint, smoky tinge, or with this there may be an admixture of a reddish hue, or it may be distinctly reddish without the smoky hue, or it may be dark, reddish-brown, almost black. When permitted to stand, a quantity of urates fall, and with them various morphotic constituents, chiefly blood-corpuscles, entire or disintegrated. The quantity of urea, as compared with the amount of urine, is much less than normal; uric acid is not less, but the saline constituents are reduced. The gross amount of solid constituents is, therefore, below the standard of health. The reaction of the urine is acid and the specific gravity is high, often reaching 1030, but this result is due to the diminished amount of

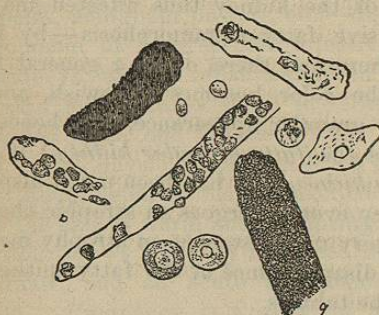


FIG. 37.—Casts of Acute Parenchymatous Nephritis. (Beale.)

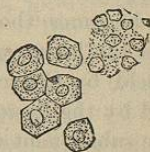


FIG. 38.—Epithelium from Convoluted Tubes. (Beale.)

water, since the solids in the aggregate are below normal. In the further progress of the case, as the amount of water increases, the specific gravity falls; but there is an increase in the solids and in the urea in the aggregate, although the quantity of each is small in any single specimen of the urine. The decline in specific gravity may be from 1030 to 1005. With the diminution of specific gravity or increase of water the acid diminishes, the urine becoming very faintly acid or neutral. The most characteristic condition as regards the urine is the presence of albumin, in this affection ranging from distinct traces to three per cent. The albumin may be absent at the initial period, but only for a brief period, the aggregate amount of the urine being very small. Besides albumin and blood-globules, perfect and disintegrated, there are present casts of the tubules, of coagulated blood, and pale, transparent, hyaline casts, with an occasional epithelial cell adherent. The pale casts are usually few in number, but in the progress of the case they are supplanted by large hyaline casts and numerous large

granular casts. Usually, also, the sediment contains epithelial cells cast off from the tubes and granules in great numbers. Very often it is not until œdema of the ankles and feet appears that attention is called to the state of the urine, when it is found to be scanty. In consequence of the diminution in the amount of water separated by the kidneys, the condition of the blood and the rate of absorption, especially, the cellular tissue becomes œdematous; if the patient is up, the water settles in the feet and legs; if recumbent, it accumulates in the lumbar region and hips, and may first, or coincidentally with its appearance elsewhere, manifest itself in the eyelids. Puffiness of the face, with a peculiar pallor of the skin, and broadening of the bridge of the nose, while the eyelids are swollen, present a striking appearance which can hardly fail to be observed, and may be the first indication of the œdema. The effusion extends, the subcutaneous areolar tissue becomes universally filled, and the great serous cavities are ultimately distended to their utmost.

The retention in the blood of the excrementitious substances in health discharged by the kidneys has a disastrous effect. The nervous system is poisoned, convulsions (eclampsia) occur and vary in severity, from twitching of the muscles of the face and of the extensors of the forearms to general convulsions involving loss of consciousness and clonic spasms of all the voluntary muscular system. The appetite is lost, and there are usually nausea and protracted vomiting, and sometimes there is very troublesome diarrhœa. The loss of albumin and of blood and the poisoning of the blood by retained excrementitious matters soon lower very seriously the nutrition of the body. Vision is impaired, both in consequence of simple anæmia of the retina and of the changes of albuminuric retinitis.

**Course, Duration, and Termination.**—Those cases occurring spontaneously are more acute in character, accompanied by fever and disorders of micturition, which attract attention to the kidneys. The fever does not continue longer than a few days. If there is complete suppression, the case may terminate fatally within a week. If, as is usual, the development is slower and the urine is greatly diminished in quantity, the amount of the dropsy will depend on the reduction of urine for a lengthened period. The promptness with which œdema appears is determined by the scantiness of the urine, so that well-developed dropsy may be produced in a week. When the cellular tissue and the cavities are filled with fluid, the duration of the case depends on the degree in which the kidneys can be made to functionate, for, although temporary improvement and alleviations may result from vicarious discharge of the urinary functions, results obtained in this way are not permanent. This form of nephritis is not nearly so fatal as the other forms; indeed, the percentage of recoveries is quite large. When this disease occurs in scarlatina, it modifies the course of the latter materially, and prolongs its duration. Death may ensue in convulsions, or