rising in circles from the base of the thorax. The "diaphragmatic effusions" may give rise to pain in the pit of the stomach, and in the absence of all other symptoms, may very readily escape diagnosis. The temperature should be noted, and a careful physical examination made of the heart and lungs. The progress should be watched. Aspiration may be necessary before a positive diagnosis of pleuritic effusion can be made. Repeated rigors in a case of pleurisy indicate the formation of pus (empyema). If sudden pallor and syncope should occur, with first a fall of the temperature, followed soon by a rise, during the course of pleurisy—hæmorrhage has probably occurred. The disease then is probably tubercular in its origin. *Hydatid cysts* may give rise to signs like pleuritic effusion. There is no fever, and they develop slowly. Aspiration may remove all doubts. A *sub-phrenic abscess* is more frequently on the right side. There "may be a marked inspiratory descent of the lung above, and, in a right-sided case, of the liver below." When opened *inspiration* increases the outflow of pus—the converse occurs with fluid in the pleura (*Dickinson*). The pus is fœtid; and there may be a history which suggests a perforation of some part of the alimentary canal—the most frequent cause of sub-phrenic abscess.

*Pneumothorax* may be mistaken for very large vomicæ; but the latter are almost invariably at the apex. Cavities form slowly, the chest-wall is retracted, and the vocal fremitus is marked. The opposite conditions are present in pneumothorax. If fluid be present in the chest (hydro-pneumothorax), succussion is often heard; and this is rare in vomicæ, however large.

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## CHAPTER V.

## THE URINE.

**Contents.**—General remarks—Quantity, specific gravity, colour and transparency, odour and re-action—**Albuminuria**—**Peptonuria**—**Urea**, and its estimation—**Sugar**, and its estimation—Tests for bile, blood, pus, urobilin, indican, and acetone—Tests for chlorides, phosphates, and uric acid—**Examination of deposits**—*Naked-eye appearances*: mucus, pus, uric and oxalic acids, urates, oxalate of lime, triple phosphates, amorphous phosphates—Urinary concretions—*Microscopic examination of deposits—Inorganic deposits:* uric acid crystals, urates, oxalate of lime, triple phosphates, neutral phosphates of lime, cystin, leucin, and tyrosin—*Organic deposits:* tube casts, blood corpuscles, pus, epithelium, spermatozoa—Micro-organisms, and parasites. (See Appendix.)

As so large a number of diseases during some part of their course are associated with renal changes, and as so many apparently primary affections are really secondary to kidney disease—the examination of the urine should be more or less a matter of routine. Even when negative results are obtained, as is the case in a large proportion of the cases, the information gained is often of importance in relation



to the *prognosis* as well as the diagnosis. No case can, therefore, be said to be complete without an investigation of the urinary secretion. In Bright's disease, it is, of course, of special importance; and as the symptoms in all forms may begin very insidiously, it is often only by the examination of the urine that the disease is detected in the early stages. For these reasons, therefore, it will be well for the junior practitioner to continue to a great extent the routine practice of the hospital, until experience teach him how far such an examination may be unnecessary.

When the urine is to be examined the patient should be directed to empty the bladder at a certain time, and the quantity should be collected and measured for the next twenty-four hours. He should also be directed to micturate before going to stool; and in some cases of incontinence it may be necessary to use a catheter frequently. The whole of the urine need not be kept, but a small quantity, at each micturition, may be placed aside in a cylindrical glass. By this means a fair average sample is obtained—the urine varying in acidity and specific gravity in relation to the food and liquids taken during the day. A preliminary inquiry as to the act of micturition may elicit the fact that there is *pain* or *increased frequency*. Sugar in the urine, or great acidity, may give rise to the former—and an increased volume (as in diabetes mellitus and insipidus, and in chronic Bright's disease) to the latter symptom. There are many *surgical* affections in which micturition is painful and frequent. (See Caird and Cathcart's *Surgical Handbook*.)

The examination of the urine should first include the consideration of the *quantity, specific gravity, the colour and transparency, the odour and re-action.*

1. **The Quantity.**—In adults the normal amount of urine passed within the twenty-four hours should average about fifty ounces. Ten or fifteen ounces, *less* or *more*, may be allowed for the variations, which depend chiefly upon the character of the diet, the quantity of fluid imbibed, and the occasional withdrawal of the fluid through other channels.

The urine is *diminished* in heart disease; in acute Bright's disease, and in the latest stages of the chronic forms; in renal colic; in diarrhœic affections, and after effusions and hæmorrhages; in irritating nervous reflexes and shock; in severe anæmic conditions; in fevers; and from mechanical and surgical causes. Complete suppression may also result from the action of drugs (cantharides, turpentine, &c.). The urine is *increased* in diabetes mellitus and insipidus; in the chronic forms of Bright's disease, and in cystic disease of the kidneys; and by diuretics.

2. **The Specific Gravity.**—This varies in health and it should always be considered with the quantity of urine passed. Normally, the larger the quantity of fluid, the lower is the specific gravity. Taking fifty ounces as the average quantity passed in the twenty-four hours, the specific gravity should be about 1,020. It is estimated by the *urinometer*—care being taken that it do not touch the sides

of the glass vessel. All froth should be previously removed from the surface of the urine to allow of a clear reading. Any departure from these normal relations indicates an increase or a diminution of the solids excreted by the kidneys. When the specific gravity is *low* (in relation to the quantity of urine), it indicates either diminished tissue change throughout the body, or the failure of the kidneys to excrete the urea and uric acid products. A low specific gravity is often a precursor of uræmia (v. Jaksch). The specific gravity is *high* in diabetes (sugar), and in fevers (urea). It is *low* in chronic kidney affections.

3. **The Colour and Transparency.**—The colour of normal urine is characteristic, and is due to the pigments. The urine is pale in diabetes; in chronic Bright's disease; anæmia; and after "neurotic" seizures. It is dark when concentrated, as in fevers, diarrhœa, and dyspepsia, &c. In grave organic disease the deposits are sometimes much darker from adherent pigments (purpurine, melanin, &c.). Rhubarb and senna, when taken medicinally, colour the urine a light brown; santonin colours it yellow. When carbolic acid is absorbed, the urine is olive-green in colour. *Blood* in small quantity, imparts a "smoky" tinge to the urine; when in large quantity, the urine is bright red. *Bile pigments* in the urine give it a yellow-green appearance, well seen when the vessel is slightly agitated. The urine is normally *transparent*, but the presence of pus, mucus, or oil globules may render it opalescent; and after cooling, the cloud may be due simply to urates. Alkaline urines are generally clouded with pus, &c. Albuminous urines, and urine containing bile, urobilin or indican, are often *frothy*.

4. **The Odour.**—Normal urine has a characteristic odour. Decomposed urine smells of ammonia. Bloody urine is very offensive. Turpentine, when taken medicinally, imparts an odour of sweet violets to the urine; and copaiba, cubebs, sandal-wood oil, &c., may be detected by the smell, in the urine of patients taking these drugs.

5. **The Re-action.**—Healthy urine is slightly acid. It is tested by red and blue litmus papers. The urine normally becomes less acid after a meal, and its re-action is subject to variations during the day. It becomes alkaline on standing for some time. In fevers the acidity is increased; and in severe anæmic conditions the urine is often alkaline. The latter condition has been pointed out by von Jaksch, as suggesting a ready means of determining whether the morbid process in chlorosis is continuing or not. Ammoniacal urine is frequently induced by the use of a dirty catheter.

**Albuminuria.**—The presence of albumen in the urine must always be regarded as a serious morbid symptom, although it does not always indicate structural changes within the kidneys. It is believed by some to be occasionally present in healthy urine, but only in small quantity. In the latter case, it may only be temporary, and it is supposed to be due to disturbances of the circulation affecting particularly the renal organs. In estimating the value



of albuminuria as a symptom in medical cases, due regard must be given to these so-called *accidental* causes; and the surgical affections (inflammations of the urinary tract—*e.g.*, pyelitis, cystitis, urethritis, vaginal discharges, &c.), must be excluded. In such cases the albumen present is small in quantity, and the microscopic examination, with the absence of tube casts and the discovery of pus cells, &c., will enable the physician to discriminate these conditions.

*Serum-albumen* and *peptone* are the two varieties which are important—especially the former. Other albumens have been separated, but they need not be considered here as they do not as yet come within the range of practical medicine.

The most common cause of albuminuria is kidney disease. The amount of albumen present, however, is no guide to the severity of the lesion—at least in chronic cases. Albuminuria occurs also in acute fevers, and then it is due to secondary structural changes within the kidneys; in severe anæmic conditions—allowing of *exudation*; in heart disease, emphysema, &c.—from mechanical causes; and it is often present in epilepsy and other paroxysms. It may sometimes be present temporarily from some error in diet—a highly albuminous food (pastry, &c.) frequently producing it. A turbid urine in the morning with albumen in small quantity—*then*, but at no other time—is due generally to inflammation of the prostatic or other portion of the urethra. Sometimes in kidney disease—even in *acute* nephritis—the albumen may be absent in some of the urine passed.

**Tests for Albumen.**—(A) *Nitric acid.*—Take about an *inch* of urine in the test-tube and allow the nitric acid to run slowly down the inside of the tube. It will form a layer at the bottom, and if albumen be present a white ring forms, more or less dense according to the quantity present, and extending gradually *upwards* through the urine. To add too small a quantity of nitric acid, or to allow the acid to mix with the urine, may spoil the test. A *brown ring* (urohæmatine) between the nitric acid and the urine is seen to form when only a trace of albumen is present, or when the urine contains none. In applying this test, very turbid urines require to be filtered. When the albumen is present in very small quantity, allow half an hour to elapse for the formation of a ring. There are three sources of fallacy in this test:—(1) Concentrated urine may precipitate urates, but *heat* clears these up, and, moreover, the precipitation begins at the surface of the urine and extends *downwards* (Roberts); (2) nitrate of urea may form, but this precipitate also clears up with gentle heat; and (3) resinous matters in the urine precipitate with nitric acid. (The *quantity* of albumen present is usually estimated by the careful application of this test.)

(B) *Boiling Test.*—The urine in this test must be slightly acid. It is well to add a drop of acetic acid in every case, and more must be used should the urine be alkaline. The upper part of the column of urine may be heated and the opalescence (when albumen is present) contrasted with the urine in the lower part of the tube. Should precipitation occur on boiling, acetic or nitric acid should be cautiously added to the boiled urine. If the precipitate be phos-

phates, they dissolve—if albumen, the coagulation remains. The chief source of fallacy arises in connection with the relative amount of acid used. If the quantity of albumen present be small in amount, the acid forms a soluble nitrate; and if phosphates be present as well as albumen, and the acid added be relatively small in amount, soluble albuminates may result. *Resins* are also a source of fallacy. These are soluble in alcohol—albumen is not.

(C) *Acetic Acid and Ferrocyanide of Potassium.*—After filtering the urine, if necessary, add a considerable quantity of acetic acid, and then a few drops of a 10 per cent. solution of ferrocyanide of potassium. If albumen be present in quantity a white precipitate forms—if only a trace, the urine becomes opalescent. This test may be applied by first preparing a fresh solution of the acid and ferrocyanide, and then adding it slowly (as in the cold nitric acid test). A ring forms between the urine and re-agent.

(D) *Biuret Test.*—Treat the urine with caustic potash, and then add cautiously drops of a solution of copper sulphate. The green precipitate which forms is dissolved if albumen be present, and the fluid assumes a red-violet colour.

It is unnecessary to allude to the other and numerous tests for albumen, some of which are, however, very delicate. The above will serve all practical purposes. The elaborate *quantitative* analysis of albumen is a matter for the laboratory. The cold nitric acid test gives it approximately, or the *picric acid* test with Esbach's albuminimeter may be used—directions for using being purchased with the tube.

**Peptonuria.**—The discovery of a simple test for peptone in the urine, by Hofmeister, and the subsequent investigations by von Jaksch, have brought this subject before the practical physician. According to von Jaksch—from whose work the following is taken—"the causes of peptonuria are quite different from those to which the other forms of albuminuria are due." It appears in the urine commonly, but not invariably, when suppurative processes (pneumonia, phthisis, purulent pleuritic effusion, purulent meningitis, &c.) are present in some part of the system, and when the products of such suppurations (peptone) pass into the blood, and are eliminated by the kidneys (*Pyogenic peptonuria*). Its detection is an indication of such suppurations, and, therefore, *peptonuria* has a high degree of significance in clinical medicine. Peptonuria, however, occurs also in severe cases of scurvy, and it may be present in ulceration of the intestine, in phosphorus poisoning, and in puerperal states. Excluding these, the discovery of peptonuria will indicate the stage of softening in pneumonia, or that purulent changes have occurred in pleuritic effusions, or in abdominal tumours, &c.

*Tests for Peptone.*—The urine should be tested for albumen by tests B, C, and D. If B and C give no results, add acetic acid alone to a fresh sample of the urine, and, if no precipitate form, apply the biuret test (D). The latter test will detect peptone if present in sufficient quantity, but the fluid becomes red instead of violet. A second test is to add first a concentrated solution of acetic acid, and then a mixture of acetic and phospho-tungstic acids. If peptone



be present, a clouding takes place either directly or shortly afterwards. Small quantities of peptone may also escape this test. Nitric acid (dropped) also precipitates albumoses which clear upon heating —re-appearing on cooling.

When serum albumen is present, it requires to be removed by precipitation with a metallic oxide, and then filtering.

*Hofmeister's test* is more accurate, but it is very elaborate (see v. Jaksch).

**Urea.**—The significance of an increase or diminution of this body in the urine has been mentioned with the diseases in which such changes occur. The normal amount of urea excreted by healthy adults varies from about 300 to 500 grains, or more, in the 24 hours. An albuminous diet increases the quantity. It is estimated as follows:—

*Russell and West's Method.*—Take 25 c.c. of the hypobromite of soda solution in the conical flask; also 5 c.c. of urine into the small test-tube, taking care that they do not mix; attach to the graduated glass cylinder in the usual way; then allow urine and hypobromite sol. to mix, and in fifteen minutes read off the quantity of gas evolved within the graduated cylinder.

*Calculation*—

(1) 37·5 c.c. N. : c.c. in burette :: 1·55 gr. : grs. urea in 5 c.c.
(2) 5 c.c. : 28·4 (*i.e.*, ʒi) :: (grs. urea in 5 c.c.) : grs. urea in ʒi.

Instruments are now graduated so as to record the amount of urea in grains *per ounce* without the neccessity of making this calculation.

**Sugar.**—A small proportion of sugar is present in healthy urine,* but the usual tests do not discover it. Glycosuria is often a symptom in the gouty It sometimes occurs in fevers; in diseases of the heart, lungs, and liver; and in diseases of the brain—especially when the lesion affects the region of the fourth ventricle. The latter conditions are somewhat rare. Persistent glycosuria is the prominent symptom of diabetes mellitus. Other sugars besides grape-sugar are found in the urine, but the latter is the only important one clinically.

**Tests.**†—*Fehling's Solution.*—Take about an inch in depth of the test solution in a test-tube and heat, until it begins to boil; then add two drops of the urine. If sugar be abundant, a thick *yellow* ring of copper sub-oxide is formed which may quickly become *red.* If no change occur on the application of this test, go on adding urine and boiling until an equal bulk of urine has been added to the test solution. Boil, and if no clouding appears on cooling, the urine is free from sugar.

*Trommer's Test.*—Caustic potash solution is first added to the urine, and then a strong solution of copper sulphate is added drop by drop, until the cupric acid formed is no longer dissolved. Heat the mixture in a test-tube. If sugar be present the yellow-red oxide is formed before the boiling point is reached, and the solution loses colour. This test is not reliable for small quantities of sugar; and, besides, the re-action occurs with other bodies—*e.g.*, uric acid, bile pigments, mucin, &c.

*Phenyl-hydrazin Test.*—This test is believed by v. Jaksch to be

* Carbohydrate in the form of glycuronic acid: "dietetic" glycosuria.
† If albumen be present in quantity it should be removed in testing for sugar.

free from all fallacies. He takes two parts of hydrochlorate of phenyl-hydrazin, and three of acetate of soda, and places them together in a test-tube containing about two drachms of urine. A little water may be added if the salts do not dissolve on gently warming the solution. The test tube is now placed in boiling water for twenty to thirty minutes. Afterwards it is placed standing in cold water. A yellow crystalline deposit occurs when sugar is present; and this sediment when examined under the microscope is seen to be composed of long *yellow acicular crystals*—detached or in clusters (phenyl-glucosazon). The test is very delicate.

**Quantitative Estimation of Sugar.**—*Pavy's Method.*—Take 10 c.c. urine and dilute to 100 c.c. in the burette. Place 20 c.c. Pavy's sol. into the flask, cork, and attach to burette; boil one minute and keep simmering to expel air. Drop from burette, and shake flask, till Pavy's sol. = colourless. Read quantity of urine used.

*Calculation.*—(1) 20 c.c. Pavy's sol. = 0·01 gram: or 0·154 grains glucose ∴ $\frac{1}{10}$ of the quantity of urine used from the burette : 28·4 c.c. (*i.e.*, ℥j) : : 0·154 grains : grains glucose *per ounce*. Multiply by the total amount of urine passed in twenty-four hours.

*Fehling's Method.*—10 c.c. Fehling's sol. diluted, and placed in a porcelain dish. Urine placed in the burette, as in Pavy's method, and dropped into Fehling's sol. until colourless.

*Calculation.*—10 c.c. Fehling's sol. = ·05 gram: or ·77 grains glucose ∴ $\frac{1}{10}$ of quantity of urine used from the burette : 28·4 c.c. (*i.e.*, ℥j) : : ·77 grains : grains glucose *per ounce*. Multiply by the total amount of urine passed in the twenty-four hours.

**Tests** for *bile*, *blood*, and *pus*; *urobilin*, *indican*, and *acetone*; and for *chlorides*, *phosphates*, and *uric acid*.

*Bile.*—This re-action is really due to the pigments in the bile. A few drops of urine and of fuming nitric acid are placed *separately* upon a white porcelain slab, and then allowed to run into each other. If bile be present, there is a play of colours—violet, green, red—gradually fading away. The tests for the *bile-acids* are tedious, and only suitable for the laboratory.

*Blood.*—(1) A drop of tincture of guaiacum is added to a small quantity of urine in a test-tube, and about half an inch of ozonic ether is poured in. A *blue* colour forms. (2) The urine may be treated with caustic potash and boiled. The resulting precipitate of basic phosphates with hæmatin is coloured a bright-red. (3) The spectroscopic test may be applied (*Hæmoglobin* and *Methæmoglobin*).

*Pus.*—Add a solution of potash to the sediment, and if pus, it becomes *ropy*—*mucus* becomes thin and flocculent.

*Urobilin.*—The urine is dark and often frothy. The clinical interest of urobilinuria is associated with hæmorrhages and extravasations of blood into the tissues. The blood-colouring matter is re-absorbed and eliminated by the kidneys. Gerhardt suggests "that a chloroform extract of the urine should be treated with solution of iodine, and caustic potash added, when a beautiful green fluorescence develops." Urobilin can be tested spectroscopically.

*Indican.*—The urine has a rich dark-brown colour, and it is



generally frothy. Indicanuria, clinically, is a symptom of *albuminous putrefactive changes* within the system; but it occurs, sometimes, in simple constipation.

Jaffe's Test.—About a drachm of urine is mixed with an equal quantity of hydrochloric acid, and then a solution of a hypochlorite is added, drop by drop, and shaken up with the urine. An indigo-blue results if the hypochlorite is not used in excess.

*Acetone.*—Acetonuria occurs in fevers, diabetes, cancer, cerebral irritations, &c. A large nitrogenous diet tends to produce it. Acetonuria may sometimes render the prognosis of a case more grave (diabetes and cerebral affections). The most ready *test* consists of treating two drachms of the urine with freshly-made concentrated sodium nitro-prusside, and strong solution of caustic potash. The solution becomes red, but this soon disappears, and if acetone be present, it is replaced by a purple or violet colour on adding acetic acid. The latter re-action does not take place in the absence of acetone.

*Chlorides.*—Add nitrate of silver to the urine, and the result is a white precipitate which is insoluble in nitric acid.

*Phosphates.*—The same test as above, but the white precipitate formed *is* soluble in nitric acid.

*Uric Acid.*—Boil some suspected urine with a few drops of nitric acid in an evaporating dish. When dry, add a few drops of dilute ammonia. The result is a red-purple of *murexid*. To add solution of potash, further, produces a *blue-purple* colour.

(See Appendix for further tests for these substances.)

**Examination of Deposits.**—The urine should be allowed to stand for several hours in a cylindrical, or if the deposit be small in amount, in a conical glass; or the urine may be placed in a *centrifugaliser*, and deposits are thus got immediately for microscopic examination.

**Naked-eye Appearances of Deposits.**—*Mucus.*—A small quantity is present in health, which may be seen as a delicate cloud throughout the urine. In catarrhal conditions the quantity is increased, and after a few hours it forms a denser gelatinous-looking layer at the bottom of the glass.

*Pus.*—This deposit somewhat resembles mucus, but it is thicker and not so transparent. The potash test may be used to differentiate mucus from pus. Both mucus and pus may appear in the urine *before* it cools.

*Uric acid* and *oxalic acid* crystals may appear in the urine entangled in the mucus, shortly after the urine is passed. Uric acid crystals resemble grains of cayenne pepper. When in greater quantity they form a dense red deposit, which is soluble in alkaline solutions. They occur in gouty conditions. Oxalic acid crystals indicate imperfect oxidation, and they are found often in certain forms of dyspepsia.

*Urates.*—These deposits are by far the commonest. They consist of amorphous urates of ammonia, soda, lime, or magnesia, and they

are often associated with uric acid crystals and oxalate of lime. In urines which become quickly alkaline, the urates may be present along with phosphates of lime. The deposit of urates appears after the urine cools, and it varies in colour from a pink or pink-white to brick-red. It occurs in all feverish conditions; in renal congestion; in dyspepsia and liver complaints; and after active exercise with perspiration. Urates are entirely soluble on heating the urine.

*Oxalate of Lime.*—This deposit generally forms two layers—the upper being white in colour, and the lower grey and more gelatinous-looking. Urates are frequently present along with the oxalates. The latter are soluble in a strong solution of hydrochloric acid, but not in acetic acid. The presence of oxalate of lime is not significant unless in large quantity. Oxaluria is a form of dyspepsia associated with much depression; but oxalates in the urine may occur after the ingestion of rhubarb, tomatoes, beetroot, &c.

*In alkaline urines, triple phosphates* (ammonio-magnesium phosphates) form *white* deposits when seen alone. *Amorphous phosphates* form light flocculent deposits. Phosphates are generally present along with pus in ammoniacal urines—the result of inflammation of the bladder, or of the urinary passages. Phosphaturia occurs also in chronic dyspepsia. Phosphatic deposits are soluble in acetic acid.

*Urinary concretions* (chiefly urates and uric acid, and more rarely phosphates) are frequently found, and are of interest in relation to renal colic. Foreign bodies and impurities are discussed in surgical works.

**Microscopic Examination of Deposits**—Inorganic Deposits.—*Uric acid crystals* vary much in shape and size, but they are always brown-yellow in colour. The lozenge shape, rhombic tables, and spiculate forms are the commonest (see Fig. 17).

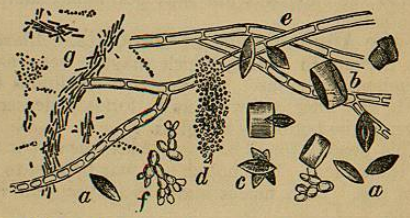


Fig. 17.—*a*, *b*, *c*, Uric acid; *d*, *g*, Micrococci and bacilli; *f*, Yeast fungi; *g*, *e*, Mould-fungi. (From *Landois and Stirling's Physiology.*)

*Urates.*—Amorphous urates appear as fine granules. Ammonium urates appear as round globules (see Fig. 18).

*Oxalate of lime* crystals appear as octahedra, dumb-bells, and in compound forms (Fig. 19).

*Triple phosphates* have chiefly the "knife-rest" shape (see Fig. 18).



*Neutral phosphates of lime* appear as wedge-shaped prisms, often forming rosettes; while *basic phosphates of magnesia* form elongated rhomboids.

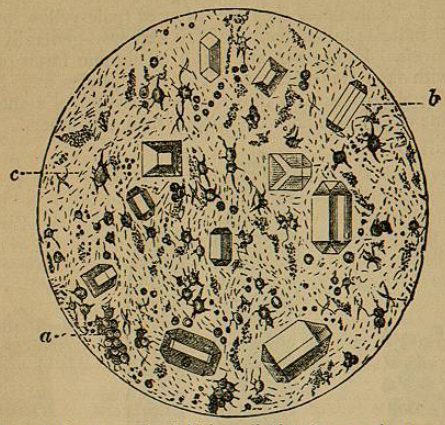


Fig 18.—Deposit in Ammoniacal Urine (alkaline fermentation).—*a*, Acid Ammonium urate; *b*, Ammonio-magnesium phosphate; *c*, Bacterium ureæ. (From *Landois and Stirling's Physiology.*)

*Cystin*, *Leucin*, and *Tyrosin.*—Cystin forms symmetrical hexagonal plates. Leucin and tyrosin are generally found together. The former are spherical in shape, and the latter are circular and in bundles (see Fig. 20). They are found in the urine in cases of acute yellow atrophy, phosphorus poisoning, &c.

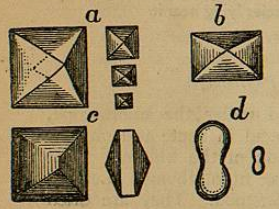


Fig 19.—Oxalate of Lime.—*a*, *b*, Octahedra; *c*, Compound forms; *d*, Dumb-bells. (From *Landois and Stirling's Physiology.*)

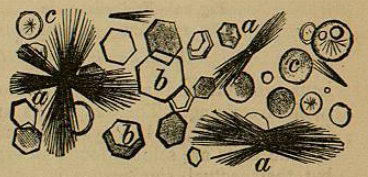


Fig. 20.—*a*, Tyrosin; *b*, Cystin; *c*, Leucin. (From *v. Jaksch and Cagney's Clinical Diagnosis.*)

**Organic Deposits—Tube Casts.**—As these have been found in non-albuminous urine, and in cases where no renal affection could be suspected, their presence (without other symptoms) does not always imply disease. The chief forms are the *epithelial*, *granular*, *fatty*,

*waxy*, *hyaline*, and *blood* casts. These forms are often *mixed*. When the casts are present in number, they are highly significant. The presence of epithelial and blood casts in albuminous urine always indicates acute renal disease. They are figured below. Granular casts vary in size. Sometimes they are coloured (pale-yellow to brown). They also indicate inflammation of the kidneys—generally the more chronic forms. Fatty casts are found in chronic forms of Bright's disease with fatty degeneration. The fat globules may be on the surface of granular casts, or they may form cylinders alone. Waxy and hyaline casts are somewhat longer than the others. They are

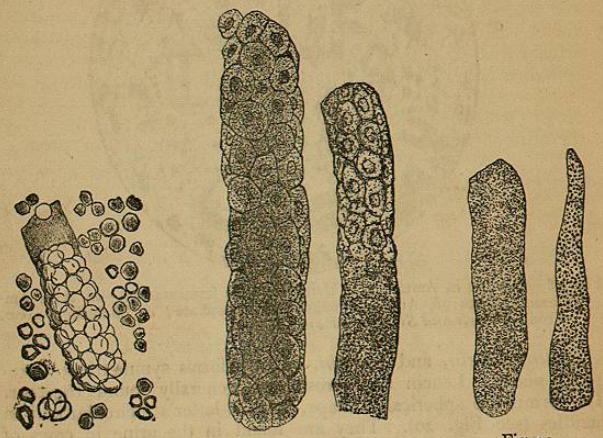

Fig. 21. Fig. 22. Fig. 23.

Fig. 21.—Blood cast; altered corpuscles lying near it.
Fig. 22.—Epithelial casts.
Fig. 23.—Finely granular casts.
(From *Landois and Stirling's Physiology.*)

homogeneous, and more difficult to find under the microscope, without staining. The waxy casts are found in acute and chronic Bright's disease, and in waxy degeneration of the kidneys. The hyaline casts are seldom found in urine free from albumen (Leube), but their actual significance is not understood. They are often present in disease of the kidneys, but it is the coating upon these casts (epithelial, fat, leucocytes or blood corpuscles, &c.), which is of clinical importance.

*Cylindroids* are the long ribbon-like casts found by Thomas in the urine of scarlet-fever patients. They have been observed in renal diseases and in healthy urine. The hyaline casts and cylindroids are believed to be products of epithelial secretion, probably connected with the exudation of albumen within the tubules. A



dilute solution of iodine and iodide of potassium may be used to detect the hyaline casts.

*Red blood corpuscles* in the urine may retain their proper form, or may appear as attenuated and shrunken yellow rings—the biconcave character being often lost. The latter is generally the case when the blood is intimately mixed with the urine and does not form a sediment. This occurs when the hæmorrhage is from the kidneys, or from the pelvis or ureters. When the blood is not diffused the origin is probably the bladder.

*Pus.*—The cells are readily detected under the microscope. Sometimes they are unaltered; but in alkaline urines they swell up and the nuclei disappear. The addition of acetic acid renders the nuclei visible. They are found in inflammations of any part of the urinary tract, and they are sometimes derived from abscesses in the neighbourhood. In the latter cases they are usually fatty. The addition of the iodo-potassic iodide solution stains them mahogany-brown, while epithelial cells become a light-yellow colour. Pus, in the urine of women, may be derived from the vaginal secretions.

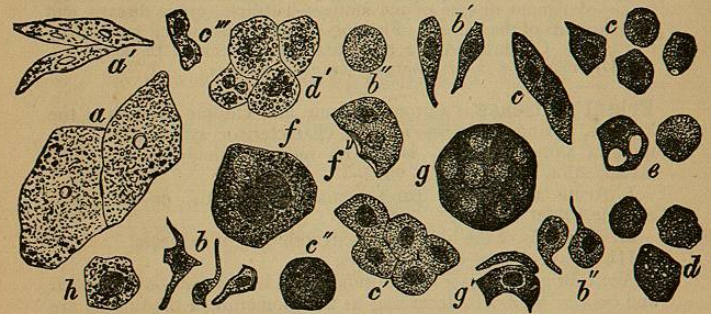


Fig. 24.—Epithelium from the Urinary Sediment.—*a*, *a'*, Squamous epithelium from the urinary sediment: *b*, *b'*, *b''*, Epithelium from the bladder; *c*, *c'*, *c''*, *c'''*, Epithelium from the kidneys; *d*, *d'*, Fatty epithelium from the kidneys; *e-h*, Epithelium from the bladder. (From *v. Jaksch and Cagney's Clinical Diagnosis.*)

*Epithelium.*—It is only when epithelial cells are present in quantity in the urine, that they are of diagnostic importance. They indicate a catarrh of some part of the urinary tract. They differ in form, and sometimes it is possible to locate the seat of origin. (See Fig. 24).

Cancer cells cannot be clinically differentiated from the normal cells with any certainty. Cells from pigmented tumours may sometimes be detected.

*Spermatozoa* are readily recognised by their tadpole appearance.

*Micro-organisms and Parasites* (see Fig. 17).—Moulds, yeasts, and fission-fungi are often found in the urine. Moulds are common

in diabetes, while fission-fungi and yeasts appear in ammoniacal urines. Bacteriuria is observed after the use of unclean catheters. The tubercle bacilli may be found in phthisical cases with renal complications. The presence of infusoria has no pathological significance. The parasites which affect man and which may be found in the urine, are as follows, viz., *Distoma hæmatobium*, *Filaria sanguinis hominis*, *Echinococci*, *Eustrongylus gigas*, and sometimes *Ascarides*.

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## CHAPTER VI.

## DISEASES OF THE URINARY SYSTEM.

**Contents.**—Acute and chronic parenchymatous nephritis—Cirrhotic Bright's disease—Waxy disease of the kidneys—*Differential diagnosis of the three forms of Bright's disease*—Diabetes mellitus—Diabetes insipidus—Renal calculi—Hydronephrosis—Pyelitis, and suppurative nephritis—Perinephritis, and perinephric abscess—Malignant disease of the kidney—Tubercle, cystic disease, and hydatids of the kidneys—Floating kidney—Paroxysmal hæmaglobinuria—Chyluria—Active and passive congestion of the kidneys—Uræmia, *and the diagnosis of kidney affections.*

**Bright's disease** is a group of acute and chronic diseases of the kidneys, having *albuminuria* as a characteristic symptom. Albuminuria alone, however, is by no means pathognomonic. The classification is as follows, viz. :—

I. Acute and chronic parenchymatous nephritis, or acute and chronic Bright's disease.

II. Cirrhosis of the kidneys, or cirrhotic Bright's disease.

III. Waxy disease of the kidneys.

Acute Bright's disease may be subdivided into *glomerular nephritis* and *tubular nephritis*, according as the glomeruli or the tubules are most affected—the former being the case in scarlet fever. Glomerulo-nephritis is known as the scarlatina kidney.

Sir T. Grainger Stewart tabulates the different forms of Bright's disease thus, viz. :—

I. Inflammatory—
  (A) Glomerular nephritis.
  (B) Tubular nephritis, { Simple. / Fatty kidney. / Atrophied kidney. } As stages.
  (C) Both forms together.

II. Cirrhotic Bright's disease.

III. Waxy disease of the kidneys, { Simple. / Enlarged. / Atrophied. } As stages.

IV. Combined forms.

Clinically, the atrophied stages may be regarded as cirrhotic Bright's disease.



**Acute Parenchymatous Nephritis—Tubular Nephritis—Acute Desquamative Nephritis—Acute Bright's Disease.—Pathology.**—The kidney is increased in size and weight. The capsule strips off easily and reveals a smooth surface beneath it. The cortex is pale, with small extravasations throughout its textures; and the pyramids are deeply congested. The microscopic examination reveals changes in the convoluted tubules, almost exclusively. Fine deposits—cloudy granulations—are seen within the epithelial cells, and these, producing distention, almost occlude the tubules which frequently become dilated and tortuous.* In the later stages, fatty degeneration of these products of inflammation give the organ a yellow mottled appearance (large fatty kidney). In the extremely chronic stage, the organ becomes atrophied. According to Grainger Stewart, *all* forms of Bright's disease end in atrophy, if the patient live long enough, and the disease do not terminate in recovery.

The **causes** of acute Bright's disease are, sudden exposure to cold when the skin is warm and perspiring; scarlatina, diphtheria, typhus, diabetes, and erysipelas; pregnancy; substances which irritate the kidneys, as cantharides, turpentine, copaiba, &c.; and hereditary influences. Acute Bright's disease is commoner in youth than in the aged; and a constitutional type is described—pale, light-haired, and flabby individuals—who are predisposed to this form of the disease.

Most frequently the early **symptoms** of acute Bright's disease are obscure, and there is little constitutional disturbance. Œdema of the ankles, or a puffiness of the eyelids, is often the first symptom which attracts attention, and calls for examination of the urine. At other times, there is a history of a chill followed by fever and lumbar pains, with painful, and frequent, attempts at micturition, resulting in the passage of a few drops of urine, often bloody.

The urine is diminished in quantity, and sometimes there may be entire suppression, which may lead to very urgent symptoms, and death within a few days (*uræmia*). The specific gravity is increased to 1,025 or 1,030 in the early stages, and this is due to the diminished amount of water secreted; but soon, if the disease proceed, it is as low as 1,010 or 1,005. The urine is acid in re-action, and it is often loaded with urates; and if it contain only a small proportion of blood, it has a smoky appearance. When the quantity of blood in the urine is greater, the colour is from a red hue to a dark-brown, according to the amount. Albumen is always present in this form of Bright's disease, and it may be roughly estimated by the quantity which appears with an inch of urine in the test-tube, when the cold nitric acid test is applied. In severe cases, the amount of albumen may show from a half to three-fourths of the bulk of urine within the test-tube. The maximum is about 35 grammes in twenty-four hours.

* In the scarlet fever kidney, these changes affecting the glomeruli more than the tubules, the Malpighian bodies are more prominent on section of the kidney. The microscopic changes are also observed in this neighbourhood (glomerulo-nephritis). In acute nephritis the inflammation frequently extends to the intertubular substance.