CLINICAL EXAMINATION OF URINE

LINDLEY SCOTT

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THE

CLINICAL EXAMINATION OF URINE

COLOUR VARIATIONS OF THE URINE.

NORMAL PIGMENTS.

UROCHROME. UROERYTHRIN. UROBILIN. HÆMATOPORPHYRIN.

BLOOD PIGMENTS.

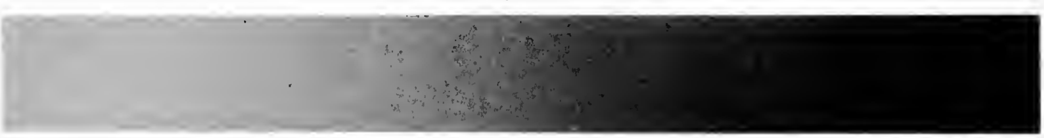
OXYHÆMOGLOBIN. METHÆMOGLOBIN.

BILE PIGMENTS.

BILIRUBIN. BILIVERDIN.

OXIDATION PRODUCTS.

CHARACTERISTIC OF CARBOLURIA, ALCAPTONURIA, AND MELANURIA.

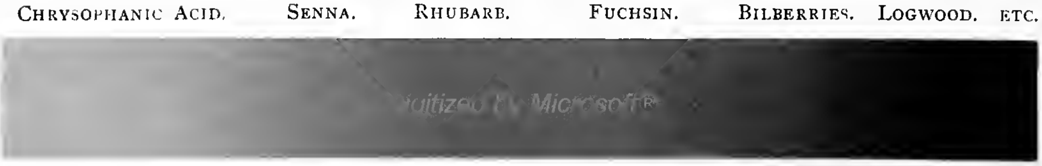


CHROMOGENIC PIGMENTS.

INDIGO-BLUE. INDIGO- AND SKATOXYL-RED AND UROROSEIN.

FOOD AND DRUG PIGMENTS.

CHRYSOPHANIC ACID. SENNA. RHUBARB. FUCHSIN. BILBERRIES. LOGWOOD. ETC.



THE CLINICAL EXAMINATION

OF

URINE

WITH

AN ATLAS OF URINARY DEPOSITS

INCLUDING FORTY-ONE ORIGINAL PLATES
MOSTLY COLOURED

BY

LINDLEY SCOTT, M.A., M.D.

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PREFACE

In the production of this book, with its forty-one plates, my endeavour has been to prepare a ready reference handbook to the Clinical Examination of Urine.

The various tests mentioned have been chosen from the experience of many observers, and I have found them in practice to be the most applicable and the most trustworthy.

The series of plates provide an Atlas of Urinary Deposits. With one exception, they have been drawn from specimens under my microscope, the aim being to arrange the objects in a microscope-field in such a way as to portray their essential and distinguishing characters. The exception is Plate XXX, which shows the Rare Animal Parasites; these have been copied from the original drawings, as specimens were not obtainable.

I have to thank Professor Halliburton for the benefit of his advice upon the plate of Colour Variations of the Urine. My gratitude is also due to Dr. PATRICK MANSON for help in connection with the Animal Parasites, and to Dr. JAMES GALLOWAY for valuable suggestions in the bacteriological and other parts of the book.

LINDLEY SCOTT.

LONDON: *January* 1900.

CONTENTS

GENERAL CONSIDERATIONS

THE urine is a watery solution of various organic and inorganic compounds. These compounds exist pre-formed in the blood, and are separated therefrom and excreted by the kidneys. They have their origin in the processes of digestion and absorption of the solid and liquid constituents of the food, and in the building-up and breaking-down of the tissues of the body. The composition of the urinary excretion thus depends upon these processes. Hence examination of the urine not only enables us to detect diseases of the urinary system, but also helps to indicate the manner in which the processes of metabolism are performed.

Physiologists have long debated as to the exact way in which the various constituents of the urine are eliminated by the kidneys. It is now generally believed that the water and part of the salts are excreted by the glomeruli, while most of the solids are eliminated by the renal epithelia, by means of their peculiar selective and secretory powers.

The collection of the Urine for examination

Collection of the urine

As a matter of routine the urine of every patient should be examined. This is often difficult or impossible to carry out in private practice. It is worthy of note, however, that such serious conditions as diabetes and chronic kidney disease are often unsuspected from the history of an illness, and may be otherwise overlooked. In some cases it is advisable to have the urine passed in one's presence: this ensures a fresh specimen, and avoids the possibility of substitution. When blood, pus, and other substances are found in the urine of females, it is sometimes necessary to draw off the excretion with the catheter, as vaginal discharges readily get into the urine.

For quantitative analyses it is usually essential to have a sample from the urine collected during twenty-four hours, the quantity passed being measured.

The urine passed early in the morning, owing to its high acidity and concentration, is best adapted for detecting any tendency to gravel. Sugar and albumin are most likely to be found in the urine passed two or three hours after the principal meal of the day. The first few drops of urine naturally wash out the urethra, and often show a few floating white particles or wavy threads, which may arise from the collection of the natural urethral secretions, or, if very abundant, from some abnormal state of the urethra or glands opening into it.

It is always wise to bear in mind the possibility of contamination by dirty bottles or utensils.

Composition of the Urine

Seventy grammes of solids and 1,500 c.c. of water are eliminated by the average adult daily. The following table (p. 3), constructed by Parkes, represents the amount of the various constituents of the urine passed *in twenty-four hours.*

—	Absolute weight of solids in grammes	Weight of solids per 1,000 grammes of body-weight
Urea	33·18	0·50
Uric acid	0·55	0·0084
Hippuric acid	0·40	0·0060
Creatinine	0·91	0·014
Pigment and other substances	10·0	0·151
Sulphuric acid	2·01	0·0305
Phosphoric acid	3·16	0·0480
Calcium	0·26	0·0004
Magnesium	0·21	0·0003
Potassium	2·50	0·0420
Sodium	11·09	0·1661
Chlorine	7·50	0·1260
Ammonia	0·77	0·0130
Solids	72·5	1·057 grammes
Water	1,500	23·00 c.c.

Urine also contains a small quantity of free nitrogen and carbonic acid gas. Xanthin, hypoxanthin, allantoin, lactic and glycero-phosphoric acids, ethereal substances, ferments and mucin, are also constituents of normal urine, but are present in small amount.

The *total solids* of the urine may for clinical purposes be estimated by multiplying the last two figures of the specific gravity by 2·33. This gives the amount of solids in grammes in every 1,000 c.c. of urine passed. Thus if a man passes 1,500 c.c. of urine in twenty-four hours with a specific gravity of 1020 Total solids

$$\text{the total solids} = \frac{20 \times 2{\cdot}33 \times 1500}{1000} = 69{\cdot}9 \text{ grammes.}$$

PHYSICAL PROPERTIES OF URINE

Odour

I. **Odour.**—Fresh urine has an odour peculiar to itself. The administration of turpentine gives to the urine a smell somewhat like violets. The partaking of asparagus, onions, sandal-wood, or cubebs causes the characteristic odour of these substances. In diabetes the urine has an aromatic or fruity smell. In septic urine, with much pus or bacteria, a heavy fœtid odour can often be recognised. Urine alkaline from decomposition smells of ammonia.

Colour

II. **Colour.**—Within physiological limits the urine varies from the palest yellow to a deep reddish-yellow colour, the tint depending upon its degree of concentration and the relative proportion of the different pigments. In normal urine the following pigments are found.

A. Normal Urinary Pigments.—1. *Urochrome* is present in considerable quantity; it gives the urine its yellow colour, and may be regarded as *the* pigment of normal urine.

2. *Uroerythrin* is a pigment of much intensity, and is present only in small amount. It has a strong affinity for uric acid compounds, and gives urates a pinkish colour.

3. *Urobilin*: only traces are found in normal urine. It is closely allied to bile pigment. In certain morbid states—such as febrile affections, pernicious anæmia, cirrhosis of the liver, severe internal hæmorrhage, and acute gout—it may be excreted in quantity, and gives a deep reddish-brown colour to the urine, so that it may look to contain blood or bile.

4. *Hæmatoporphyrin* gives a rich red or light port-wine colour. Traces occur normally in the urine, but it is excreted chiefly in

morbid conditions or after the administration of certain drugs, as sulphonal.

B. Chromogens.—A name given to certain colourless substances which develop into pigments on the addition of caustic alkalies or acids, or on undergoing oxidation by exposure to the air, as when the urine is kept standing for a considerable time. **Chromogens**

Urobilin and hæmatoporphyrin are often represented in the urine by their chromogens. The best-known chromogens are:

1. *Indoxyl*, readily oxidising into indigo-blue or indigo-red. The former may be found in cholera and typhus, and when the urine is putrefying. It gives rise to a bluish scum on the surface and a blue deposit, which may show under the microscope blue amorphous fragments or crystals.

2. *Skatoxyl-red* similarly arises from the chromogen of skatoxyl.

3. *Urorosein*, derived from its chromogen by the action of mineral acids, causes a red tint.

Allied to these is the smoky tint which occurs in the three following conditions:

4. *Carboluria*, a greenish-brown colour of the urine arising from the oxidation of aromatic substances, normally present, but excreted in excess after the administration of carbolic acid and other coal-tar products. They reduce Fehling's solution. **Carboluria**

5. *Alcaptonuria*: Bödeker in 1859 discovered in the urine a substance, which he called alcapton, having peculiar chemical reactions and causing a smoky discoloration. The condition is now generally believed to be due to the oxidation of such aromatic compounds as pyrocatechin, hydrochinon, uroleucic and homogentisinic acids. An alkaline urine containing these substances, when kept exposed to the air, develops a brownish or smoky tint, which gradually becomes more intense, and ultimately brownish-black. It is chiefly a congenital condition, and seems to run in families and to depend on **Alcaptonuria**

some peculiar faulty metabolism. It may be found in apparently perfect health or towards the end of toxic conditions like uræmia, and has then an unfavourable significance. The chief interest of alcaptonuria is attached to the strong reducing power of the urine on copper solutions, in some cases being equivalent to as much as two per cent. of grape-sugar.

Melanuria

6. *Melanuria*, a blackish colour of the urine occurring occasionally in the subjects of melanotic tumours. The black colour becomes intensified when the urine is kept exposed to the air; it arises from the oxidation of the chromogen of melanin.

Blood pigments

C. Blood Pigments give to the urine a reddish or smoky tint. In hæmaturia, oxyhæmoglobin and methæmoglobin are usually present together. The red colour will therefore predominate with the larger proportion of the unaltered hæmoglobin; the brownish-black shade when the methæmoglobin is in excess. (See also p. 34.)

Bile pigments

D. Bile Pigments.—Urine containing bile is of a yellowish-brown, greenish-brown, or dirty olive-green colour. The bilirubin gives the brown tint; the biliverdin the green colour.

Drug and food pigments

E. Drug and Food Pigments.—Certain drugs and articles of diet administered by the mouth discolour the urine.

Chrysophanic acid gives it an orange colour; santonin a greenish-yellow (acid) or reddish colour (alkaline); rhubarb and senna a brown or reddish tint; logwood, fuchsin, madder, and bilberries a red colour; and carbolic acid, resorcin, and some other coal-tar products a smoky tint.

In the frontispiece I have endeavoured to construct a table showing at a glance the possible variations in the colour of the urine, and the pigments which give rise to the particular colours and depths of tint. It must be remembered that both normal and abnormal urines contain several pigments, and that the proportions present may vary enormously, and occasion a corresponding variation in the tint of the urine.

It is hoped that reference to the table will afford an immediate indication as to the reasons for the colour of a urine under examination. Thus, a sample of a smoky or dirty yellow hue would suggest the presence of a small quantity of bile or blood, or possibly some oxidation product; while one of a deep red colour would indicate the presence of blood, or food and drug pigments. The various pigmentary substances would then be searched for by the usual tests.

III. **Transparency.**—Normal urine is clear and transparent when passed. In the course of half an hour the mucus tends to collect as a fine cloud towards the centre of the vessel, and ultimately deposits, forming a delicate wavy surface with the clear urine above. **Transparency**

Freshly voided urine may be opaque from—

1. Precipitation of the earthy phosphates, arising from an alkaline reaction; they disappear on the addition of an acid.

2. Organic sediments, such as pus and blood, kidney and bladder *débris*, urethral and vaginal discharges.

3. Bacteria located in the urinary passages and voided with the urine.

4. Chyluria, as seen in elephantiasis, where the urine has a milky appearance from the presence of chyle.

Urine which has been voided for some time may become opaque from—

1. Precipitation of the amorphous urates; they disappear on heating.

2. The growth of bacteria and fungi derived from the atmosphere.

3. Alkaline decomposition leading to the precipitation of phosphates, etc.

IV. **Reaction.**—The collected urine of twenty-four hours has normally an acid reaction, turning blue litmus-paper red. The acidity **Reaction** is not due to any free acid, as often supposed, but arises from the preponderance of certain acid salts, chiefly the acid phosphate of soda. (See also p. 19.)

The degree of acidity varies greatly from day to day and throughout the twenty-four hours.

The highest acidity is found in the early morning and midway between meals. After taking food the acid reaction gradually diminishes, till in three or four hours it may be replaced by an alkaline reaction. This is due to the conversion of some of the acid phosphates into alkaline phosphates by the salts of the food and is known as *fixed alkalinity*, in contradistinction to the *alkalinity arising from ammoniacal decomposition.* The latter can be distinguished by the litmus paper—turned blue by the alkaline urine—returning to its original red colour on gently heating ; in fixed alkalinity the blue colour remains on applying heat.

Ammoniacal decomposition of the urine depends on the fact that various bacteria have the power of decomposing urea to form ammonium carbonate:

$$CO(NH_2)_2 + 2H_2O = (NH_4)_2\ CO_3.$$

This decomposition tends to take place if the urine is kept exposed to the air ; or it may arise before the secretion is voided, and always denotes disease of some part of the urinary passages.

Some urines turn red litmus blue, and blue litmus red. This is known as amphoteric reaction, and denotes that the monohydrogen salts exceed the dihydrogen salts.

Estimation of acidity

The average acidity of the urine is estimated by Roberts as equivalent to 2 grammes of oxalic acid in twenty-four hours ; that is, to 1·33 gramme of oxalic acid in 1,000 c.c.

The acidity can be conveniently estimated by titrating with a decinormal solution of pure caustic soda (containing 4 grammes in

1,000 c.c. of water). Ten c.c. of the urine are put in a small glass flask or beaker, and two or three drops of phenol-phthalein solution added as an indicator. The soda solution is then dropped into the urine from a graduated pipette or burette, till a perceptible pink tinge is established. Each c.c. of soda solution is equivalent to 0·63 gramme of oxalic acid per 1,000 c.c. of urine. Thus, if 2 c.c. are used in neutralising the urine, its acidity is equivalent to $2 \times 0{\cdot}63 = 1{\cdot}26$ gramme of oxalic acid per 1,000 c.c.

V. **Density.**—The specific gravity of healthy urine varies between 1015 and 1025, the average being 1020. Departures from these limits are common even in health. As a rule the density is inversely proportional to the quantity excreted. Density

A urine habitually below 1012 would suggest the presence of granular kidney, and one persistently below 1007 the existence of diabetes insipidus. A specific gravity of 1025 to 1030 is not uncommon in highly concentrated urines or in the presence of much urea. Should the urine have a high specific gravity and a light colour, it probably contains sugar. The specific gravity may also be high in the presence of much albumin.

The specific gravity is taken by a urinometer, with an index reading from 1000 to 1060 and graduated to one-tenth of a degree. The mercury bulb and stem should be flat instead of the usual round pattern, as the index is thus readily brought into view on turning the urine-glass. For very accurate work the temperature of the fluid has also to be noted.

VI. **Quantity.**—The average amount of urine passed daily by an adult in health amounts to 1,500 c.c. (50 ounces), the normal range being 1,300 to 1,700 c.c. (45 to 55 ounces). Individual peculiarity and habit must be taken into account, and persistent departures from these figures are not infrequent. Much depends on the amount of liquid taken by the mouth and upon what escapes by other channels, such as the skin and bowels. Anything which increases the blood Quantity pressure within the glomeruli or the activity of the renal epithelium will increase the flow of urine; and upon this depends the diuretic action of certain drugs and articles of diet, such as tea, coffee, and alcohol.

In morbid states the quantity of urine is subject to immense variation. It is increased in diabetes mellitus, diabetes insipidus, granular kidney, waxy disease of the kidney., and in certain nervous affections like asthma and hysteria. It is diminished in febrile states; acute and subacute nephritis, and the earlier stages of chronic parenchymatous nephritis; in cardiac, hepatic, and pulmonary diseases causing obstruction to the renal circulation; and in dropsy from whatever cause.

Suppression of urine

Suppression of urine may be obstructive or non-obstructive. The former is mechanical, and arises from some interference with the passage of the urine about the pelvis of the kidney, ureters, or urethra, as by calculus, stricture, or pressure of some tumour on the urinary tract; the latter is a reflex phenomenon, and may occur after operations on the urinary system, such as the use of a catheter and operations for kidney stone, or during the course of acute nephritis, toxic febrile diseases, perforative peritonitis, cholera, and dysentery.

NORMAL CONSTITUENTS

I. Urea: CH_4N_2O

Urea

Urea is regarded by physiologists as the final product in the metamorphosis of the proteid components of the body and of the food. It is therefore an indication of the metabolism going on in the organism, and is the vehicle by which almost the entire nitrogen is eliminated. Its chief seat of formation is the liver.

Urea is a crystalline substance having the power of combining with acids to form salts. The nitrate of urea is frequently found deposited, as six- or eight-sided crystalline plates, in the test-tube which has been left standing after applying the cold nitric acid test for albumin. Urea itself is very soluble, and is never spontaneously deposited in urine.

In a healthy adult about 30 grammes (500 grains, or $3\frac{1}{2}$ grains per pound of body weight) are excreted in twenty-four hours, the normal range being 20 to 40 grammes. Children eliminate proportionately more than adults. Its excretion is increased after partaking of food, especially animal food, and during the period of rest following active exercise.

Pathological Relations

Pathological relations

During the acute stage of febrile affections the amount of urea is increased, owing to the rapid tissue metamorphosis going on. For the same reason it is increased in diabetes and in plethoric individuals accustomed to good living, a largely albuminous diet also playing a part in these conditions.

Estimation of urea

Its chief clinical significance is the diminished excretion in all forms of Bright's disease. A sudden diminution in diabetes is said by Ralfe to be a common prelude to the onset of coma. In acute yellow atrophy of the liver urea is much diminished or absent. In estimating the quantity of urea it is always necessary to have the urine collected for twenty-four hours and measured, a sample from the aggregate being taken for estimation.

Quantitative Tests

Hypobromite method

1. **Hypobromite Method.**—Urea is decomposed by a solution of sodium hypobromite; nitrogen and carbonic acid being set free:

$$CH_4N_2O + 3NaBrO = CO_2 + 2H_2O + 3NaBr + 2N.$$

The carbonic acid is absorbed by the caustic soda in the test solution, and the nitrogen thus isolated is measured; 370 c.c. of nitrogen at 0° C. and under 760 mm. pressure being equal to 1 gramme of urea.

The test solution is made by dissolving 100 grammes pure caustic soda in 250 c.c. of distilled water, and adding, when cool, 25 c.c. of bromine. This solution does not keep well, and for those who are not applying the test frequently it is safer to add the bromine as required. Thus to 23 c.c. of the caustic soda of above strength 2 c.c. of bromine are added immediately before use. Owing to the very pungent vapour of bromine it is more convenient to make use of the glass capsules of bromine, each of which contains sufficient for one estimation. The 23 c.c. of caustic soda solution are measured in a glass-stoppered jar or bottle, and the bromine capsule dropped in and broken by a smart shake.

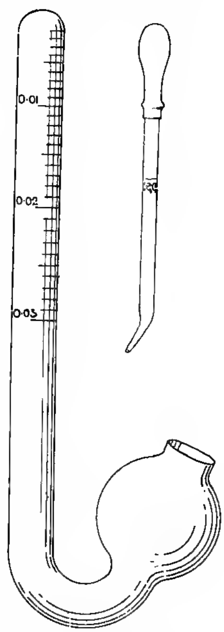


Fig. 1.—Doremus' Ureameter.

Doremus' ureameter

(1) *Doremus' instrument* is the simplest means of applying the hypobromite method, and is sufficiently accurate for all clinical purposes. It consists of a graduated bulb-tube closed at one end, and a bent pipette to measure 1 c.c. of urine. The test solution is poured into the bulb, and by tilting the tube the solution runs into the long arm, expelling all air. The urine to be tested is drawn into the nipple of the pipette up to the graduation; the pipette is then passed into the ureameter as far as the bend, and the rubber nipple slowly compressed. Bubbles of nitrogen are evolved, and collect at the upper part of the tube. In fifteen minutes the quantity of urea present is read off in percentage, or grains to the ounce, according to the graduations on the instrument.

Dupré's ureameter

(2) *Dupré's apparatus* is highly efficient, but is better suited for laboratory work. 'It consists of a bottle (*a*) fitted with an indiarubber stopper,

carrying a small test-tube attached to a glass rod, and a small piece of glass tube connected with the indiarubber tube (*b*), which conveys the nitrogen evolved to the graduated tube (*c*), in which it is measured. The upper limb of the graduated tube is fitted with a piece of rubber tube and closed by means of a pinch-cock (*d*). The cylindrical pneumatic trough (*e*) is fitted with a sliding rod and clip, which carries the graduated tube, and can be fixed in any position by means of the screw clamp (*g*). The operation is as follows: The pinch-cock is taken off and the graduated tube let down until the zero point corresponds with the level of the water in the pneumatic trough; 25 c.c. of hypobromite solution are put into the glass bottle; 5 c.c. of the urine are measured into the little test-tube by means of a pipette (*h*); the stopper is replaced, and the bottle held for a minute or so in cold water. The pinch-cock is now put on again and the graduated tube raised several inches, when, if all is tight, the column of water raised in the tube will remain stationary. The bottle is then tilted and all the urine allowed to mix well with the hypobromite solution, and the test-tube seen to be washed out by the hypobromite. In one or two minutes the reaction is almost over. The bottle is now shaken briskly several times; this causes an appreciable amount of effervescence, and brings the reaction sharply to the same point each time. In five minutes from the beginning of the experiment the bottle is put into cold water and the gas tube let down into the cylinder

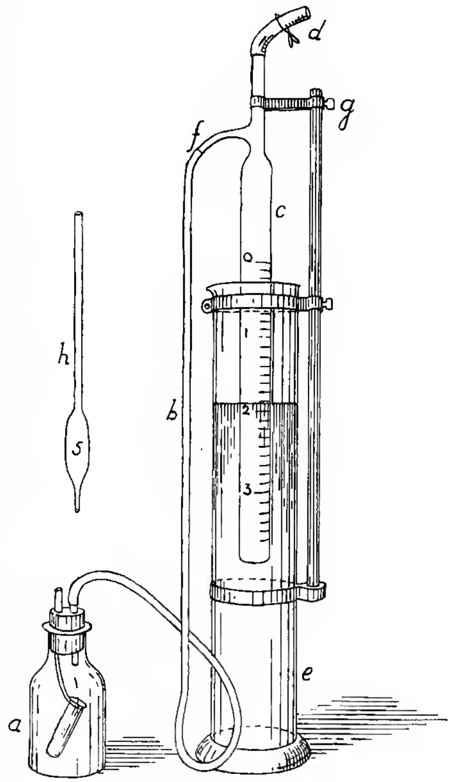


FIG. 2.—Dupré's Ureameter.

for a few moments, and then raised until the water inside and out is on the same level. The amount of gas collected is read off. Each graduation represents ·1 per cent. of urea.'

Liebig's method

2. **Liebig's** or **Mercuric Nitrate Method.**—A titration process dependent upon the fact that mercuric oxide forms an insoluble salt with urea in water and weak alkaline solutions. The solution of mercuric nitrate employed is prepared by a special process, and is of such a strength that 1 c.c. precipitates or is equivalent to ·01 gramme of urea. A saturated solution of sodium carbonate is used as an indicator.

As the phosphates and sulphates are also precipitated by the mercuric solution, they have first to be removed. This is done by mixing two volumes of the urine with one volume of baryta solution (prepared by adding two volumes of a saturated solution of barium hydrate to one volume of a saturated solution of barium nitrate), and filtering. Fifteen c.c. of the filtered solution—representing 10 c.c. of urine—are put in a beaker under a burette holding the mercuric nitrate solution, which is dropped in gradually till a precipitate ceases to form. This is determined by mixing a drop of the urine from the beaker with a drop of the sodium carbonate indicator (by means of a glass rod) on a white plate, when a distinct yellow colour will arise when the titration is complete.

As the chlorides are also precipitated, 2 c.c. have to be deducted from the number of c.c. of mercuric solution required.

Thus, 20 c.c. required—2 c.c. (for chlorides)=18 c.c.

$18 \times {\cdot}01 = {\cdot}18$ grammes of urea in 10 c.c. of urine=18 parts per 1,000.

This process is open to several errors, and should be repeated to accurately determine the point when titration is finished.

If albumin is present it must first be removed by precipitation with acetic acid and heat, and filtering.

II. Uric Acid: $C_5H_4N_4O_3$

From $\frac{1}{2}$ to 1 gramme of uric acid is excreted daily in the urine of an adult. The quantity seems to vary greatly according to the metabolic activity and body weight of the individual. Recent research points to its origin from the disintegration of the albuminous tissues, especially those rich in cells or nuclein; and its chief seat of formation is probably the liver. The proportion excreted is greatest during the early years of life, and decreases as age advances. Its formation is increased during leucocytoses—whether temporary leucocytosis, such as arises after the taking of food; or permanent, such as occurs in leucocythæmia. Its excretion rises a few hours after meals, and is *slightly* greater after animal than after vegetable food. The increase, however, is neither contemporaneous with nor proportional to that of urea, and the two substances bear no constant relationship to each other. An increased quantity of uric acid is eliminated in pernicious anæmia, leucæmia, leucocythæmia, febrile diseases like pneumonia, and after active exercise, alcohol and certain drugs. Later work seems to create a good deal of uncertainty as to its excretion in gout; it is most probable that there is an increase during the acute paroxysm and a decrease in the intervals of attack.

Roberts has shown that uric acid exists in the blood combined with potash and soda as a quadriurate, and as such is eliminated in the urine. The quadriurate readily undergoes disintegration, a portion of its uric acid being precipitated in crystalline form. This happens sooner or later in all urines, if kept from ammoniacal decomposition; but should it take place in a few hours after passage of the urine it indicates a tendency to the deposition of uric acid within the urinary system and to the formation of stone. It most readily occurs in urine rich in uric acid and with a strongly acid reaction, and is corrected by reducing the acidity of the excretion by the internal administration of alkalies.

Quantitative Estimation of Uric Acid

Gowland Hopkins has introduced the following simple and reliable method: 100 c.c. of urine are saturated with pure powdered ammonium chloride (30 to 35 grammes being necessary); let stand for a few hours, and **Estimation of uric acid** filter; wash precipitate on the filter paper two or three times with a saturated solution of ammonium sulphate. The precipitate, now consisting of pure ammonium urate, is washed into a beaker by a jet of hot distilled water, and dissolved by the addition of a few drops of carbonate of soda solution, and heating. The solution is cooled and made up to 100 c.c. Twenty c.c. of pure strong sulphuric acid are added, and titration immediately commenced with a one-twentieth normal solution of permanganate, made by dissolving 1·578 gramme of permanganate of potash in a litre of distilled water. As it is run in from the burette—at first quickly, and then drop by drop—the red colour disappears immediately it comes in contact with the urate solution. When the red tint remains *for a few seconds*, and is disseminated throughout the solution on shaking, titration is finished. Each c.c. of permanganate is equivalent to 0·0037 gramme of uric acid.

Sediments

The sediments of uric acid found in the urine are:

1. Amorphous Urate.—We are indebted to Roberts for determining its exact composition. He has shown it to consist of quadriurates of potash, soda, lime, and ammonia. It forms the common loose reddish sediment of the urine. It has a strong tendency to take up the urinary pigment, and varies in tint from fawn to brick-red, according to the colour of the urine. As a rule, it soon sinks to the bottom of the vessel; occasionally it forms an iridescent film on the surface.

The amorphous urates are readily dissolved by heat; on rapidly cooling the urine they are quickly precipitated.

Highly acid and concentrated urines most frequently deposit them. No conclusion as to excessive elimination of uric acid can be drawn from their deposition. Physiologically, they are apt to be found after profuse sweating, violent exercise, prolonged fasting, and in cold weather; pathologically, in all febrile states, derangements of the digestive organs, and in organic disease of the heart and liver.

Under the microscope the amorphous urates appear as somewhat opaque granules, scattered loosely or in masses.

2. Uric Acid.—See Plates I, II, and III.
3. Urate of Ammonia.—See Plate IV.
4. Acid Urate of Soda.—See Plate V.

III. Oxalic Acid: $C_2H_2O_4$

Oxalic acid is now known to be a normal constituent of the urine; from ·02 to ·07 gramme is excreted daily. Some assert it to be entirely derived from the soluble oxalates contained in the food; but the majority of writers believe it to have its origin also in the imperfect oxidation of proteids. The latter view is supported by the preparation in the laboratory of oxalic acid by the artificial oxidation of uric acid, and by the frequent association of oxalate of lime and uric acid crystals in urinary sediments. In the urine of a young woman affected with gastric ulcer I was able to repeatedly detect crystals of oxalate of lime, while she was being fed exclusively upon milk, a diet containing neither soluble nor insoluble oxalates.

Since Beneke and Neubauer's work, oxalic acid is believed to exist in the urine as the oxalate of lime, and to be held in solution by the acid phosphate of soda. This theory does not quite correspond with clinical experience, for most observers have noted the deposition of oxalate of lime in highly acid urines, that is to say, in urines rich in the acid phosphate of soda. Oxalate of lime is an extremely common urinary sediment; in fifty subjects, taken without selection, and many of them in the best of health, I found oxalate of lime deposited in about forty per cent. within twenty-four hours of the urine being passed; it was more often associated with uric acid than with any other crystal; a similar percentage is given by other writers (Baron, Gallois, etc.)

Clinical significance

Clinical Significance.—Oxalate of lime crystals are commonly found in the urine of dyspeptic, gouty, and diabetic subjects. At one time there was believed to be an 'oxalic acid diathesis,' showing itself by malaise, mental depression, indigestion, and oxalate crystals in the urine; it is much more probable, however, that these symptoms arise from imperfect digestive processes than from an excess of oxalic acid in the circulation, and the belief in a special diathesis is now almost entirely abandoned.

Oxalate of lime derives much importance from its tendency to form stone, especially renal stone. Fagge finds small oxalate calculi to be the most common cause of hæmaturia, pyelitis, and renal colic in middle-aged people. Beale says oxalate of lime forms the nucleus of most calculi; it is certain that it contributes largely towards the growth of mixed calculi.

It is now recognised that the precipitation of oxalate of lime in the kidney in the form of showers of crystals may give rise to attacks of hæmaturia, and renal pain, without the aggregation of the crystals in the form of a calculus.

The eating of such articles of diet as tomatoes, rhubarb, asparagus, and apples, rich in soluble oxalic acid salts, is frequently followed by oxalate crystals in the urine; a point worthy of attention in the subjects of gravel and calculus. For the characters of oxalate of lime crystals refer to plates VI, VII, and VIII.

The methods for the quantitative estimation of oxalic acid in the urine are all tedious and difficult.

IV. Phosphates

Phosphates

From 2 to 3 grammes of phosphoric acid are excreted by the kidneys daily. About two-thirds of it exist in combination with potash and soda, as highly soluble salts; the rest is united with lime and magnesia, forming far less soluble compounds, which are deposited when the urine becomes feebly acid or alkaline.

The phosphates are derived almost entirely from the phosphates in food; to a slight extent also from the metamorphosis of the tissues, chiefly nerve tissue. Their excretion is most plentiful after a meal, especially when composed largely of animal food. They are increased in acute cerebral inflammation, in certain functional nerve disorders, in convalescence from acute disease, and in some bone affections—osteomalacia and rickets. They are decreased in gout, kidney disease, most acute disorders, and in pregnancy.

When the urine is heated in a test-tube, it frequently becomes turbid from the precipitation of the earthy phosphates. This is probably due to the fact, as Roberts suggests, that the phosphates of lime are less soluble at high than at low temperatures, and are consequently precipitated on heating the urine. The point merits attention, for it may give rise to the belief that the precipitate is due to the presence of albumin; it will, however, be found to entirely clear up on the addition of any acid.

The phosphates exist as monobasic, dibasic and tribasic salts, varying in reaction according to the ratio of acid and base. The reaction of the urine depends upon the proportion of the various phosphates contained. Thus, its normal acid reaction is due to the predominance of the acid phosphates, chiefly the acid phosphate of soda. After meals or the administration of alkalies, and in certain dyspeptic states, the proportion of alkaline phosphates is increased, and the urine tends to become neutral or alkaline (fixed alkalinity). This naturally gives rise to a precipitation of the earthy phosphates, and in certain individuals, prone to such a reaction of the urine, the condition was at one time believed to indicate an increased excretion of phosphates and a 'phosphatic diathesis.' Precipitation of the phosphates does not, however, mean increased excretion, and the fallacy of the theory is readily apparent.

PHOSPHATIC SEDIMENTS

Phosphatic sediments

Consist of :—

1. *Amorphous phosphate of lime or bone earth*, $Ca_3(PO_4)_2$.

2. *Crystalline phosphate of lime or stellar phosphate*, $CaHPO_4$, $2H_2O$.

3. *Ammonio-magnesic or triple phosphate*, $MgNH_4PO_4$, $6H_2O$.

The first two are deposited when the urine becomes alkaline from any cause, the third only when ammoniacal decomposition has taken place.

They derive importance clinically from their tendency to form calculi: a pure phosphatic calculus is rare—more usually the phosphates combine with other calculi, chiefly by forming secondary incrustations upon them.

1. *Amorphous phosphate of lime*, $Ca_3(PO_4)_2$, occurs as a whitish, flocculent deposit, never taking up the urinary pigment. Occasionally it appears on the surface of the urine as a fine iridescent film. Under the microscope it is seen to be composed of minute, shapeless, unpigmented granules, somewhat like the amorphous urates: they are dissolved by an acid.

2. and 3. For the stellar and triple phosphates refer to plates IX, X, and XI.

Estimation of total phosphoric acid

Estimation of phosphoric acid

The necessary test solutions are :—

1. A standard solution of uranium nitrate, consisting of 20·3 grammes pure oxide of uranium dissolved in 1,000 c.c. distilled water.

2. Sodium acetate solution, prepared by dissolving 100 grammes of sodium acetate in 900 c.c. distilled water, and adding 100 c.c. acetic acid.

3. A saturated solution of potassium ferrocyanide.

Put 50 c.c. of urine in a flask, and add 5 c.c. sodium acetate solution; warm over water bath, and add the uranium solution slowly from a burette, till a precipitate ceases to form. This is recognised by mixing a drop of the urine with a drop of potassium cyanide solution on a white slab; a reddish brown colour forming at the point of contact when the titration is completed. Each c.c. of uranium solution used is equal to 5 milligrammes of phosphoric acid.

V. Sulphates

Sulphates

About two grammes of sulphuric acid are excreted daily in the urine. The greater portion is combined with potash and soda as simple sulphates; the rest is excreted as aromatic or ethereal sulphates; a small quantity of sulphur is also eliminated as sulphocyanides, hyposulphites, and sulphuretted hydrogen.

Simple sulphates

Ethereal sulphates

The sulphates are derived partly from the food and partly from the albuminous tissues of the body. Little interest is attached to the *simple sulphates*; the sulphate of calcium is the only one that has been found as a sediment, and that only very rarely (plate XII). The excretion of *ethereal sulphates* is, however, of importance: they are formed in the intestine from the decomposition of proteid matter, either during the processes of digestion, or through bacterial putrefaction. They are toxic products, and are absorbed from the bowel, and eliminated by the urine, and therefore serve as a means of estimating the amount of intestinal putrefaction. The ethereal sulphates are increased in constipation, intestinal obstruction, putrefactive diarrhœa, and peritonitis. By some they are considered to have an important bearing upon gout, on the theory of auto-intoxication. They include such substances as pyrocatechin, hydrochinon, and phenol, but the most important are skatol and indol (the antecedent of indican).

The estimation of the proportion of ethereal sulphates to the simple sulphates is often now carried out; the normal ratio is one of the ethereal sulphates to twelve of the simple sulphates ; the following is the method in vogue :—

Estimation of ethereal sulphates.

Quantitative estimation of sulphates

To 100 c.c. urine add 100 c.c. alkaline barium chloride solution (2 parts saturated solution of baryta to 1 part saturated solution of barium chloride) ; this precipitates the simple sulphates. Filter, and add 10 c.c. hydrochloric acid (sp. gr. 1·12) to the filtrate, boil for five minutes, and place in warm chamber for two hours. This converts the ethereal sulphates into simple sulphates, and precipitates them. Collect the entire precipitate on a weighed filter-paper, and wash it with boiling water, till the filtrate gives no precipitate with dilute sulphuric acid, showing the absence of free barium chloride. Next wash with boiling alcohol, and then with ether. Incinerate the filter-paper and contents in a platinum crucible, cool over sulphuric acid, and weigh. 233 parts of the barium sulphate correspond to 98 parts by weight of sulphuric acid ; from which the amount of sulphuric acid in the 100 c.c. of urine can be calculated.

To estimate the total sulphates repeat the above process with another 100 c.c. of urine, without previously adding the alkaline barium chloride solution to remove the simple sulphates.

VI. Chlorides.

Chlorides

Six to ten grammes of chlorine are excreted daily in the urine of an adult. The most of it is in the form of sodium chloride (10 to 15 grammes), the rest as chlorides of potash, ammonium, and magnesium. Their excretion depends largely upon the food, and to a certain extent on tissue metamorphosis. They are diminished during the acute stage of all febrile affections, particularly in pneumonia. At one time the quantity excreted in this disease was considered of importance, but is now believed to have no clinical significance. They never occur as urinary deposits, and their quantitative estimation is, in the present state of our knowledge, of little or no clinical value. The chief interest attached to them is, as

Roberts has shown, their tendency to prevent the deposition of uric acid, and, for this reason, a diet rich in saline material is recommended for the subjects of uric acid gravel and calculus.

ABNORMAL CONSTITUENTS.

I. Proteids.

Proteids

The term 'albuminuria' has generally been applied to the presence of proteid matter in the urine. The various proteids which may present themselves in urine are:—Serum-albumin and globulin, albumose, peptone, hæmoglobin, fibrin, and mucin. In the great majority of cases the predominating proteid is serum-albumin.

Albuminuria

A. Albuminuria.—When serum-albumin is present in the urine, a considerable proportion of globulin usually accompanies it, and occasionally also a trace of albumose. These three proteids give very similar reactions, and when present together their differentiation becomes complicated, and is *as a rule* of little practical value (see globulin, p. 26). It is to the presence of albumin in this fashion that the term 'albuminuria' should be restricted, in contradistinction to cases of albumosuria, mucinuria, etc., of rather different significance.

Functional albuminuria

No trace of albumin exists in the urine under normal conditions. A certain number of people, however, usually young adults from fifteen to twenty years of age, in apparent health or at least with healthy kidneys, show albumin from time to time in their urine. This condition is known as *functional, physiological, temporary*, or *cyclical* albuminuria. In some, the albumin appears only after taking food, especially food rich in albumin (dietetic albuminuria); in others, after exercise, as in soldiers on the march, or after cold to the skin, as in bathing; or, again, on rising in the morning (postural albuminuria), or at regular intervals in the day. As a rule the quantity of albumin is small, and though hyaline casts are occasionally present, the absence of other signs of kidney disease (see p. 38) will help to determine the class of case. Many such cases seem to be due to a functional aberration on the part of the kidneys or digestive system, and a good number of the subjects have impaired general health, or are scrofulous, nervous, and weakly: in the course of time a considerable proportion develop organic kidney disease.

Pathologically

Pathologically albuminuria arises from

1, *Organic diseases of the urinary tract*, as in the various forms of nephritis, pyelitis, cystitis, urethritis, etc.

2, *Changes in the circulation*, such as passive congestion of the kidneys in heart-disease, and pressure on the renal veins by tumours, or in pregnancy.

3, *Changes in the constitution of the blood* such as may arise in anæmia, toxic states, acute febrile diseases, lead poisoning, pregnancy, etc.

Two or more of these causes may act together. Urine containing spermatic and vaginal secretion may give the reaction of albumin. In pyuria and hæmaturia the urine contains more or less albumin.

Tests for albumin

Many tests have been advocated from time to time: we select those which are most reliable and best suited for clinical work; the danger lies, not so much in failing to detect albumin, as in mistaking for it substances which give somewhat similar reactions.

Before testing for albumin the urine should be rendered clear, preferably by filtering. Gentle heat will serve to clear up urates; when the opacity is due to bacteria, filtration will not give a transparent fluid, and one must compare any opacity present after applying the albumin test with the turbidity of the filtered urine.

Ferrocyanide of potassium and acetic acid

1. *Ferrocyanide of potassium and acetic acid.*—Put twenty to thirty drops of acetic acid into a test-tube; add about three times the amount of ferrocyanide of potassium solution (1 in 20), and mix by

shaking the tube; then pour in the urine till the test-tube is about two-thirds full, and mix the contents well by inverting the tube with the thumb over the end. An opalescence will form if albumin is present.

This is the best test for routine work, and is sufficiently delicate for practical purposes. It gives no reaction with peptone, mucin, the vegetable alkaloids, urates, or the pine acids (Purdy).

2. *Cold nitric acid.*—Cover the bottom of a test-tube to the depth of half an inch with nitric acid: inclining the tube towards the horizontal, let the urine run down the side of the tube from a nipple-pipette and float on the surface of the acid, till it forms a layer from half an inch to an inch in depth. If albumin is present, a ring of opacity will form at the junction of the two liquids. If only a minute quantity of albumin is in the specimen, the opacity may take fifteen to twenty minutes to appear. A ring of dark brown discoloration must not be mistaken for albumin. This test is ready of application, but is not highly sensitive, and is open to error, as follows:— **Heller's test**

Oleo-resins in the urine give with this test a ring like albumin, but the opacity is soluble in alcohol. An excess of *urates* may also give a precipitate, but the ring is less sharply defined than albumin, is a little distance above the junction of the urine and acid, and is removable by heating. *Mucin* sometimes gives a precipitate, but the opacity is more diffuse than that of albumin. I have found iodide of potassium, when administered in quantity, to give a reaction very like albumin in the urine with this test.

3. *Picric acid and heat.*—Fill about one-third of the tube with urine, and add another third of a saturated solution of picric acid; mix by inversion and heat the upper portion of the liquid—an opacity forms if albumin is present. This test is highly sensitive, and may be used to detect minute traces of albumin; it is not, however, sufficiently reliable for routine work, as it gives a precipitate with mucin and other substances.

4. *Nitric acid and heat.*—Half fill a test-tube with urine, add two or three drops of nitric acid, and boil. A precipitate forms with albumin present. If too much or too little acid is used, the albumin may fail to be precipitated; mucin and pine acids (excreted when copaiba, cubebs, etc., are administered) also react with it; and albumose separates when cool. Neither a very delicate nor trustworthy test.

Quantitative estimation of albumin

Even when the urine appears loaded with albumin the amount does not exceed five per cent. by weight. For clinical work Esbach's albuminometer (fig. 3) is a sufficient means of estimation. It consists of an elongated tube, graduated and marked as shown. The urine is filled up to the mark at U; a solution of picric and citric acids (10 grammes picric acid, 20 grammes citric acid, and 1,000 c.c. distilled water) added to the point R; the contents mixed by inversion, and the tube set aside in a vertical position. At the end of 24 hours the depth of the resulting precipitate is read off, the figures representing parts per 1,000 of albumin (by weight). Should the urine be rich in albumin, it must be diluted with several volumes of water, and the result multiplied by the degree of dilution.

Gravimetric method.—Add 5 c.c. of urine to 50 c.c. of boiling absolute alcohol; filter; wash with alcohol, ether, and water; dry and weigh.

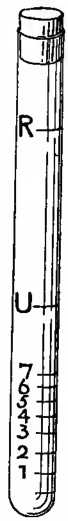


FIG. 3.—Esbach's Tube.

Globulin

B. GLOBULIN never occurs alone in the urine, but usually accompanies serum-albumin. The proportion is usually large in cystitis, acute nephritis, and waxy disease of the kidney.

Its presence may be detected by letting the urine fall, drop by drop, into a wineglass of water. The globulin is precipitated, giving rise to a milky opacity which clears up on the addition of acetic acid.

Those cases of puerperal albuminuria, in which the amount of serum-albumin exceeds the amount of globulin, are said by Herman to be the least favourable, and to terminate generally in chronic Bright's disease or in death. The globulin may be estimated by saturating the urine with magnesium sulphate, and filtering; this removes the globulin. The amount of serum-albumin in the filtrate can then be determined in the usual way. The difference, when compared with the total proteid present, will then give an indication as to the quantity of globulin in the specimen.

Albumose

C. Albumosuria.—The chemical significance of this condition is still a matter of much uncertainty. Albumose is a normal constituent of the blood, especially during digestion, and it is also present in pus. It has been detected in the urine in a variety of affections, but chiefly in osteomalacia, inflammation and tumours of bone medulla, syphilis, malignant disease, suppurative conditions, and also occasionally in febrile diseases.

Its chief interest is its excretion in the so-called myelopathic albumosuria. Bradshaw and others have found it eliminated in large quantity by the subjects of multiple myeloma. When present in the urine of people of mature age, the condition of the bones should be watched.

Tests for albumose

Add a few drops of nitric acid to the urine in a test-tube; if albumose is present a precipitate forms and disappears on boiling; when the urine cools the opacity returns.

If albumin is also present, it should be removed by boiling the urine with acetic acid and sodium chloride, and the test applied to the filtrate.

Peptone

D. Peptonuria is considered by v. Jaksch to be a strong indication of suppuration, and to be a valuable aid in the differentiation of empyema, septicæmia, and abdominal tumours, from other conditions. Peptone has frequently been found in the urine where the breaking-down of pus occurs, as in the suppuration of phthisis, purulent meningitis, empyema, resolution stage of pneumonia, etc. It occurs, however, quite apart from septic affections, and its detection, though helpful, is by no means confirmatory. Later work by Kuhn and Chittenden has shown that many of the cases of peptonuria are in reality varieties of albumosuria.

Tests for peptone

Acidulate the urine with a few drops of acetic acid, and saturate, while boiling, with ammonium sulphate, and filter; this precipitates and removes all proteids except peptone. Test the filtrate with picric acid solution; any opacity resulting is due to peptone.

Hæmoglobin

E. Hæmoglobinuria is a condition in which the blood-colouring matter (hæmoglobin) finds its way into the urine without the red blood corpuscles. It occurs as an idiopathic affection, or in syphilis, malaria, and rheumatism. The blood-colouring matter is detected by the spectroscopic or tincture of guaiacum tests, and the absence of red blood-cells shown by the microscope.

Fibrin

F. Fibrin occurs in the urine in chyluria, hæmaturia, and catarrhal affections of the urinary passages. It tends to form coagula, which may be detected by the microscope.

Mucin

G. Mucin, or nucleo-albumin, occurs in the normal urine in small amount. When present in quantity it usually indicates a catarrhal condition of the urinary passages, especially the bladder. It is to be noted that its presence frequently gives rise to error in some of the tests for albumin.

II. Carbo-hydrates

Sugar

A. Glycosuria.—A minute quantity of glucose, or grape-sugar, is normally present in the blood, and also from time to time in the urine, but not in sufficient amount to be revealed by any clinical test. Its detection in the urine must always be regarded as pathological, although its importance, both as regards diagnosis and prognosis, varies greatly. Experimentally, glycosuria can be induced by interference with the nervous system, such as puncture of the floor of the fourth ventricle, section of the splanchnic nerves, irritation of the vagus, etc.

Temporary glycosuria

Alimentary glycosuria

Glycosuria may occur *temporarily* during the course of acute febrile diseases, cerebro-spinal meningitis, liver and lung affections, and after the administration of certain drugs. *Alimentary* glycosuria is applied to those cases which depend upon a partial mal-assimilation of the carbo-hydrate principles of food, as in a too liberal ingestion of sugar or starch, or in abnormal states of the digestive system (stomach, liver, pregnancy). It is common in gouty subjects and bon-vivants, usually assumes a mild form, and responds well to dietetic treatment. *Persistent* glycosuria, or diabetes mellitus proper, is a more severe condition, and indicates a serious affection of the central nervous system, liver, or pancreas. Large quantities of sugar may be excreted daily in this state; and, as a

Persistent glycosuria

rule, the younger the subject, the less favourable are the results of treatment and prognosis.

The urine passed two or three hours after a good meal is best suited for sugar testing. Tests for glucose

1. *Fehling's solution* is a standard solution of sulphate of copper, dissolved in liquor potassæ and sodium-potassium-tartrate: the copper solution and the alkaline solution should be put up in separate bottles to prevent decomposition, equal quantities being mixed shortly before applying the test. The reaction depends on the fact that grape sugar has the power of reducing the copper salts to the suboxide, in an alkaline solution, on the application of heat. A layer of Fehling's solution, about one inch in depth, is put in a test-tube and boiled; should it retain its deep-blue colour and transparency, it is in good condition; with a nipple pipette let two or three drops of urine fall into the tube, and bring to the boiling point, when if sugar is present in any quantity an opaque yellow or brick-red precipitate of the suboxide of copper will form; should no such precipitation take place, add the urine till about equal to, *but not more than*, the amount of Fehling's solution employed; bring to ebullition, and if no reaction takes place, set aside the tube to cool; a brick-red, yellow, or greenish-yellow sediment will be found if sugar is present. A dense greyish deposit of the earthy phosphates may form, and must not be mistaken for the suboxide precipitate. 1. Fehling's solution

Note.—More mistakes are made in the use of Fehling's test for sugar than in any other urine test. The points to bear in mind are:—

1. Boil test solution before use to see that it is good.

2. Avoid using more urine than Fehling's solution.

3. Avoid much boiling of the urine and Fehling's solution (Roberts).

4. Re-examine tube when contents are cold.

Substances in the urine other than sugar have the power of reducing Fehling's solution: among them are creatine, creatinine, and other xanthine bases, excess of uric acid, nucleo-albumin, pyrocatechin, hydrochinon, homogentisinic and glycuronic acids, and bile pigments; also certain drugs when given internally, such as chloroform, chloral, camphor, salicylic, benzoic, carbolic, and hydrocyanic acids, and glycerine. In the condition known as alcaptonuria (see p. 5) much reduction of Fehling's solution may take place. A confirmatory test for sugar has therefore often to be employed, unless the reaction is very distinct and the specific gravity of the urine high.

2. Phenyl hydrazine test

2. The best is the phenyl-hydrazine test, which should be carried out as follows:—Put 1 gramme of phenyl-hydrazine hydrochloride and about the same quantity (·75 gramme) acetate of soda in a capsule, with 25 c.c. of urine and 10 c.c. of distilled water. Heat over water-bath for one hour, and remove capsule; the characteristic yellow needle-like crystals of phenyl-glucosazone (see plate XVI) will be found in eight to twelve hours if sugar is present. These crystals melt at 204° C., which distinguishes them from a similar crystalline compound of glycuronic acid, which melts at 150° C.; the latter, however, will not form if the test is carried out as above (Purdy).

A much simpler way of applying this test is recommended by Williamson. I have found it give good results, and it will detect as little as 0·015 per cent. of sugar. Fill a test-tube of ordinary size for about half an inch with phenyl-hydrazine hydrochloride, and add acetate of soda for another half-inch; pour in urine till the tube is half full, and boil carefully for at least two minutes. Set aside tube and examine deposit in 6 or 8 hours.

Quantitative tests for sugar

Quantitative tests for sugar.—The collected urine of twenty-four hours should be measured, and a portion taken for the analysis.

1. Carwardine's method

1. *By Carwardine's saccharometer and Fehling's solution.*—This is a good clinical method, and can be performed in a few minutes. The apparatus (fig. 4) consists of a large test-tube, a measuring glass, and a graduated hand-burette, as shown. Pour urine into burette up to the mark U; dilute by adding water up to mark D U. Next pour Fehling's solution (of the standard strength) into the measuring-glass up to mark F, and dilute with water up to mark D F; transfer to the test-tube, and, while boiling, pour in the urine from the burette, drop by drop, till the blue colour disappears entirely. The loss of urine from the burette, as shown by the graduations, represents the percentage of sugar in the specimen.

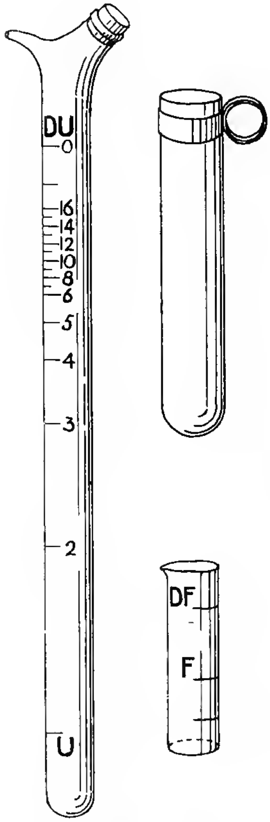


Fig. 4.—Carwardine's Saccharometer.

2. Fermentation method

2. *The fermentation method* is easy and accurate: fill two urine-glasses with urine; into one put a piece of German yeast about the size of a filbert, and let the specimens stand at an ordinary room-temperature (60° to 70° F.).

Fermentation takes place, the grape-sugar being converted into alcohol with the evolution of carbonic acid, and the specific gravity of the yeast specimen is thus lowered. At the end of twenty-four hours decant off the urines, and take their specific gravities. Each degree of difference is equivalent to one grain of sugar per ounce of urine. Thus:—

$$\text{Sp. Gr. } 1040 - \text{Sp. Gr. } 1024 = 16 \text{ grains to the ounce}$$
$$= 3{\cdot}68 \text{ per cent.}$$

(To convert into percentage multiply by 0·23)

3. Pavy's method

3. The apparatus required for *Pavy's method* (fig. 5) consists of a burette (graduated to $\frac{1}{10}$ c.c.) with stop-cock, arranged on stand as shown. The small glass flask (about 150 c.c. capacity) has an indiarubber stopper, through which pass two pieces of glass tubing, one admitting of the attachment of the flask to the point of the burette by an indiarubber tube, the other allowing of the escape of vapour on boiling. The urine, diluted 1 in 10 to 1 in 40 according to the probable amount of sugar, is placed in the burette. Ten c.c. of Pavy's modification of Fehling's solution, equivalent to 0·005 grammes of grape sugar, are put into the flask with twice the quantity of water (preferably distilled), and boiled. The urine from the burette is now allowed to drop into the boiling Pavy's solution, at first rapidly and then very carefully as its colour tends to disappear. When the blue colour has entirely gone, note the amount of liquid lost by the burette; this quantity contains 0·005 gramme of grape sugar. Thus if 4 c.c. of urine, diluted 1 in 10, are used, the amount of sugar per 1,000 parts is calculated as follows:—

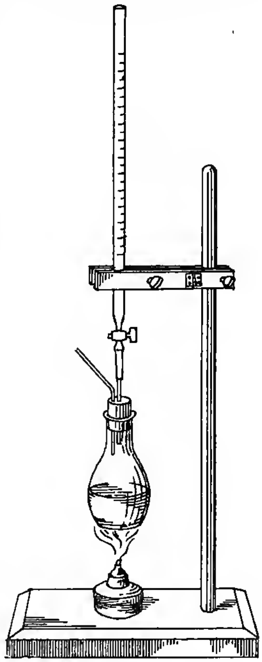


Fig. 5.

$$\frac{1000 \times 0{\cdot}005 \text{ (glucose) } \times 10 \text{ (degree of dilution)}}{4 \text{ (number of c.c. diluted urine required)}}$$
$$= 12{\cdot}5 \text{ parts of sugar per 1000.}$$

Multiply by ·4735 to convert to grains to the ounce.

Other carbo-hydrates

B. Other Carbo-hydrates.—Lævulose, lactose, dextrine, and pentose have occasionally been found in the urine in small quantities. They are difficult to identify, and their clinical significance is still very uncertain.

III. Acetone

Traces of acetone are said to exist in normal urine (v. Jaksch). It is believed to be formed chiefly from the disintegration of the albuminous constituents of the body and of the food. It occurs in considerable quantity in certain morbid conditions, such as diabetes, digestive disorders, febrile disease, cancer, starvation, and chloroform narcosis; also after over-indulgence in alcohol and nitrogenous food. Aceton-uria

Acetone is best detected by Ralfe's modification of Lieben's test. Boil 1 gramme potassium iodide with 4 c.c. liquor potassæ in a test-tube. On the surface float the urine: at the point of contact the phosphates are precipitated, and among them yellow crystals of iodoform will appear if acetone is present. The test is more delicate if applied to the distillate obtained from the urine. Test for acetone

IV. Bile

A. The Biliary Pigments, biliverdin and bilirubin, give to the urine a colour varying from yellowish-brown to dirty olive-green. They occur in the urine in jaundice (especially when due to obstruction of the bile ducts), in many liver affections with or without jaundice, and in certain abnormal blood states. Their presence is revealed by Gmelin's test:—Pour a little strong nitric acid, which should contain some nitrous acid, into a test-tube, and float on the top a layer of urine: when bile pigments are present a halo of colours (green, blue, violet, red, and yellow) forms where the urine and acid meet. The play of colours will also result on bringing a few drops of the urine and acid together on a white plate with a glass rod. Choluria A. Bile pigments Gmelin's test for bile pigments

B. Biliary Acids combine with alkaline bases to form biliary salts, a trace of which is said to be normally excreted in the urine. They are highly toxic bodies, and occur in appreciable amount in diseases of the liver (cirrhosis, congestion, carcinoma) and blood B. Bile acids (scurvy, hæmoglobinuria, anæmia, and leucocythæmia). There is no very simple and reliable method for their detection in the urine.

Pettenkofer's test for bile-acids

Pettenkofer's test is conducted as follows:—Mix in a porcelain capsule equal quantities of urine and solution of cane-sugar (10 per cent.), add drop by drop strong pure sulphuric acid while stirring with a glass rod. A beautiful red or violet colour supervenes in the presence of bile-acids.

V. Blood.

Hæmaturia Hæmoglobinuria

The appearance of blood in the urine depends chiefly upon the quantity and the length of time exuded. If small in amount and recently exuded, it will give the urine a pink tinge, or if still undiffused, as when derived from the vagina or urethra, it may appear as a reddish streak. Should the blood remain some time in contact with the unvoided urine, it will give rise to a smoky tint, and may show a fine brown deposit somewhat like coffee-grains. When the quantity of blood is large these naked-eye appearances are accentuated, the colour varying from bright red to dark brown, and the smoky or brownish-black hue, as a rule, being the more pronounced the longer the urine and blood have been together before elimination. The sediment may show blood-clots of variable size.

Blood may find its way into the urine from the urethra, vagina, prostate, seminal vesicles, bladder, ureter, pelvis of the kidney, or kidney substance. It is extremely difficult or impossible to locate the seat of hæmorrhage from the appearances of the blood in the urine. The only sure evidence of kidney hæmorrhage is the presence of blood-casts of the ureters or kidney tubes. Minute quantities of blood, though unable to be detected by the naked eye, will often be revealed by the pigmented deposit in the centrifuge tube. Urine with blood in it necessarily contains some albumin. (See also p. 6 and plate XVIII.)

Tests.

1. The *microscope* is the best means of detecting blood. It is however inapplicable as a test for blood in hæmoglobinuria, when the blood pigment escapes but rarely the red corpuscles. (See plate XVIII.) Tests for blood 1. Microscopic

2. *Tincture of guaiacum and ozonic ether.*—Half fill a test-tube with urine; add one or two drops of tincture of guaiacum, and mix well; on the surface pour a layer of ozonic ether (*fresh*) about half an inch deep; the ether will show a blue colour if blood is present, or if shaken up the froth will be tinted blue. 2. Tincture of guaiacum and ozonic ether

3. *The spectroscope.*—The freshly passed urine, if deeply coloured, should be diluted with water; it will show the two absorption bands of oxyhæmoglobin between D and E. If deoxidised, the oxyhæmoglobin is replaced by reduced hæmoglobin, the spectrum of which shows a simple broad band between D and E. 3. Spectroscopic

BACTERIOLOGY OF THE URINE

Much advancement has of late been made in the bacteriology of the urine: Alberran and Hallé in France, and Melchior and Rövsing in Denmark have largely contributed towards it.

Many micro-organisms are located in the urethra and vulva, and contaminate the urine in its passage, so that normal freshly voided urine is never sterile, but always contains a certain number of bacteria, some of them simple and harmless, like the smegma bacillus, others of them dangerous and pathogenic. The latter, on finding their way into the bladder or kidney, give rise to inflammatory conditions, such as pyelitis, cystitis, etc., but only when these organs are injured or their nutrition impaired by retention of urine, operative measures, calculi, tumours, and the like. The researches of Melchior also show the converse of this to be true—namely, that inflammatory conditions of the urinary system, such as cystitis and pyelitis, do not arise from interference with the bladder and kidney by retention of urine, calculus, operations, etc., without also the introduction of bacteria to these organs.

Pathogenic urinary bacteria are clinically divided into (1) *those which have the property of decomposing urea*, and give rise to cystitis, pyelitis, and pyelo-nephritis, with an *alkaline* (ammoniacal) urine depositing pus, epithelial cells, and debris—these include various staphylococci, streptococci, diplococcus urea liquefaciens, proteus Hauser, etc.; and (2) *those which do not possess the property of decomposing urea*, and cause either simple bacteruria, or cystitis, pyelitis, and pyo-nephritis, with a variable quantity of pus, epithelia, and debris in an *acid* urine—these include the tubercle bacillus, typhoid bacillus, gonococcus, and the bacillus coli communis.

These organisms find their way into the bladder from the urethra, usually by the passage of instruments, or by creeping along the short and wide urethra of the female; more rarely they are carried to the kidneys by the blood stream, and, if circumstances are favourable for their growth, they set up septic inflammations of the kidneys or bladder. Urinary infection may therefore ascend from the bladder to the kidney, or descend from the kidney to the bladder; so that cystitis, pyelitis, and pyo-nephritis may reasonably be considered various phases of one inflammation.

The bacillus coli communis is regarded by Melchior as the most common cause of cystitis and pyelitis, the urine in such cases being acid in reaction, containing more or less pus, and having a peculiar fœtid odour. Its point of entrance is usually the urethra, especially in females; but under certain conditions, such as diarrhœa, constipation, etc., it seems to be absorbed from its natural habitat the intestine, and being carried to the kidneys by the circulation, gives rise to an apparently spontaneous bacteruria, or to septic inflammation of the urinary system. Various bacteria may be simultaneously present, and thus it is not uncommon to have an alkaline urine in a naturally acid bacteruria, owing to the co-existence of urea-decomposing and non-urea-decomposing organisms.

Recent research work seems to make it very doubtful if there exists a special urea-decomposing organism, such as the micrococcus ureæ.

Toxic Properties of Urine

The urine of man possesses toxic properties, due to the presence of certain ptomaines and leucomaines. When healthy urine is injected into the blood of animals, it produces symptoms of poisoning, which increase in severity as the dose of injection increases, and ultimately leads to death. Toxins

The work of Bouchard, Lépine, Ruffer, Charrin, and others shows that at least six or seven different toxins are excreted in health, while in diseased conditions, such as pneumonia, cholera, and septicæmia, they are eliminated in excess, and are probably accompanied by many others. The urine of uræmic subjects is non-toxic, and may be injected into the blood without causing symptoms of poisoning, a circumstance which goes to confirm the view that uræmia arises from the retention in the system of various toxins, owing to the inability of the diseased kidneys to eliminate them from the blood.

Examination of the Urine for Life Assurance

In examining the urine for life assurance purposes, it is essential to have a fresh specimen of urine, and to know it to be passed by the subject under examination. For these reasons the water, where feasible, should be made in the presence of the examiner, or in an adjoining apartment, and its temperature noted. It is best to ask the patient to come for examination two or three hours after a meal, and to retain some water in the bladder.

After noting the specific gravity, reaction, and other physical characters of the specimen, proceed to look for *albumin* by the ferrocyanide of potash and acetic acid test. Its absence may be corroborated by the more sensitive picric acid test, should the specific gravity of the urine be low; and if, in addition, the subject is over forty years of age, casts must be looked for. Should albuminuria be found in a young subject, in apparent good health, the existence of a *functional albuminuria* may be considered probable from the following points—the absence of a low specific gravity and casts (see also p. 24) in the urine, the absence of cardiac hypertrophy, accentuated second aortic sound, arterial rigidity, fundus changes, and a family history of gout and Bright's disease. The acceptance of such a candidate should be deferred for three or four months; if on re-examination at the end of that time the albuminuria is still present, either a further postponement or non-acceptance is the safest course to adopt. Some recommend such cases for an endowment policy maturing at the age of forty-five to fifty-five.

Functional albuminuria

Sugar must next be looked for; this is best done by Fehling's solution, as recommended at p. 29. If only a slight reduction of the copper solution takes place, the phenyl-hydrazine test should also be employed, as a slight reduction may be produced by reducing agents other than grape-sugar, and which need not be a bar to acceptance of the life. The general principles that apply to functional albuminuria apply also to functional or temporary glycosuria; the candidates must either be postponed or refused, and in view of the still imperfect knowledge of the after-history of such cases, insurance companies do well to consider their acceptance risky and undesirable.

In all cases of suspected kidney disease the amount of urea should be estimated; and a microscopic examination for casts, pus, and blood corpuscles should be carried out, especially when one is apprehensive of such conditions as granular kidney, calculus, cystitis, or pyelitis.

Apparatus for Urine-testing

For clinical purposes the apparatus for examining the urine should include six or eight test-tubes, a urinometer, spirit lamp, two specimen-glasses, a small glass funnel, porcelain capsule, a small glass flask, one or two Esbach's albuminometers, Doremus' ureameter, Carwardine's saccharometer, a glass stirring-rod, a pipette with indiarubber nipple, several small pipettes, filter-papers, and a glass-stoppered jar or wide-mouthed bottle to hold two books of litmus paper, and several microscope slides and cover-slips; also glass-stoppered bottles for the following reagents:

Nitric acid	(size, two ounces, narrow mouth)
Acetic acid . . .	,, ,, ,,
Liquor potassæ	,, ,, ,,
Ferrocyanide of potassium solution (p. 24)	,, ,, ,,
Fehling's solution	,, ,, ,,
Acetate of soda in powder . . .	(size, two ounces, wide mouth)
Phenylhydrazine hydrochloride . .	,, ,, ,,
Esbach's picric acid solution (p. 6) .	(size, four ounces, narrow mouth)
Hypobromite of soda solution (p. 12) .	,, ,, ,,

All the above can be arranged in a double tier stand, about nine inches in diameter; it is best made of plain oak, and the addition of a glass shade saves dusting and ensures cleaner instruments (fig. 6).

Urinary Deposits

The centrifuge is now in common use in the collection of urinary sediments for microscopic examination. It possesses many advantages, for it admits of the deposit being examined within a few minutes of voiding the urine, and it will not fail to bring down, and concentrate into small bulk, whatever solid particles the excretion contains. For general use Gaertner's machine (fig. 7) answers every requirement. It consists of a covered hollow metal disc, into which fit four short glass tubes of about 20 c.c. capacity. By the sharp pull of a cord the disc is made to revolve rapidly (on the principle of a humming top) and any solid particles in the urine are thrown to the conical end of the tube. The clear urine is then removed, leaving the sediment behind, or the latter is lifted out with a pipette, for microscopic examination. Purdy has introduced the centrifuge for quantitative estimations; albumin or salts, after precipitation by suitable reagents, are thrown down by a definite number of revolutions, and the amount of precipitate measured in graduated tubes.

When a centrifugal machine is not at hand, the urine should be left in a conical urine-glass or large test-tube—an Esbach's tube usually answers the purpose—for eight to twelve hours, and at the end of that time the

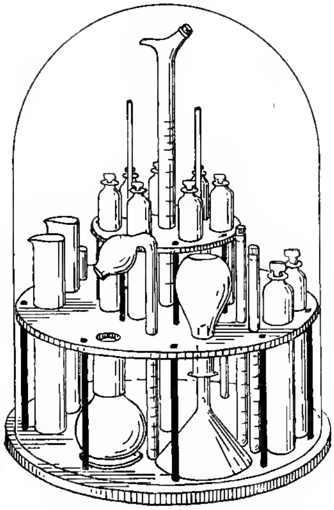


Fig. 6.

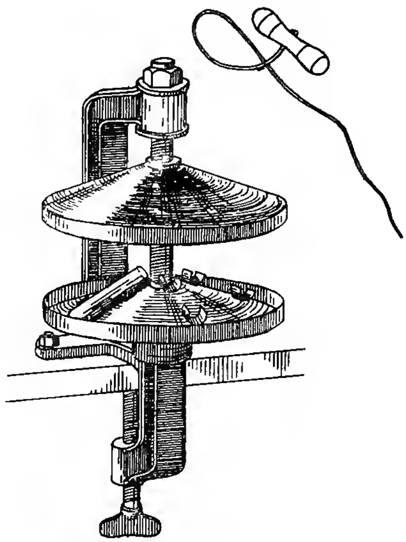


Fig. 7.—Gaertner's Centrifuge

deposit is reached with a pipette. It is convenient to have several such pipettes in use; they can be easily made by drawing out, in a gas-flame, a piece of glass tubing about a quarter of an inch in diameter. All pipettes, tubes, urine-glasses, etc., should be rinsed out with some commercial sulphuric or hydrochloric acid, and carefully cleaned.

A good microscope, supplied with 1-inch and $\frac{1}{6}$-inch objectives, is also essential. The addition of a $\frac{1}{12}$ inch oil immersion lens and polarising apparatus is an advantage.

LEADING CHARACTERS OF THE URINE IN DISEASE

Diabetes Mellitus

Quantity: largely increased (6 to 12 litres or more).

Sp. Gr.: raised, commonly from 1025 to 1045, occasionally below normal.

Colour: pale yellow, clear.

Odour: aromatic or sweet hay-like.

Reaction: acid.

Urea: increased.

Sugar: present, from 2 per cent. in an ordinary case to 12 per cent. in an extreme case.

Albumin: a trace is not uncommon.

Sediment: frequently shows uric acid and oxalate of lime crystals, yeast cells, and excess of epithelium.

Diabetes Insipidus

Quantity: enormously increased (15 to 30 litres).

Sp. Gr.: low, 1001 to 1006.

Sugar and Albumin: absent or extremely rare constituents.

Hæmoglobinuria

Quantity: usually normal.

Sp. Gr.: slightly raised, 1023.

Colour: from a bright crimson to brownish black, or smoky yellow.

Reaction: acid or slightly alkaline.

Urea: increased.

Albumin: abundant; some is frequently present in intervals of attack.

Blood-pigments: shown present by usual tests.

Sediment: heavy, granular, and pigmented; shows a few hyaline and granular casts, some epithelial cells, and much debris, frequently pigmented.

A few red blood-cells may be found.

Acute Parenchymatous Nephritis

Syn.: acute Bright's disease; acute diffuse nephritis; acute tubal or glomerular nephritis.

Quantity: scanty, may be reduced to a few ounces or even suppressed.

Sp. Gr.: raised, usually from 1022 to 1026.

Colour: from a smoky yellow to brownish black, less often bright red; turbid.

Urea: excretion in twenty-four hours always diminished.

Albumin: abundant, commonly from 5 to 10 parts per 1,000 (Esbach).

Sediment: copious, much renal epithelium, red and white blood-cells; casts, at first epithelial, blood, and hyaline, later also fatty and granular.

Chronic Parenchymatous Nephritis

Syn.: chronic Bright's disease; chronic diffuse nephritis; chronic tubal nephritis.

Quantity: diminished.

Sp. Gr.: raised, 1020–1025.

Colour: dirty yellow or smoky; turbid.

Urea: always diminished.

Albumin: abundant, fluctuates.

Sediment: copious; much debris, renal epithelium, fatty particles;

a few white and red blood-cells. Casts: most varieties to be found, chiefly fatty, granular, and large hyaline.

In the later stages the urine tends more and more to show the characters of chronic interstitial nephritis.

Chronic Interstitial Nephritis

Syn.: chronic Bright's disease; contracted kidney; cirrhosis of kidney; granular or gouty kidney.

Quantity: increased, occasionally largely.

Sp. Gr.: diminished, usually 1008 to 1012.

Colour: pale yellow, clear.

Urea: diminished.

Albumin: scanty, occasionally absent.

Deposit: scanty, showing granular and hyaline casts (chiefly small); occasionally a few epithelial cells, uric acid crystals, and blood-corpuscles.

Amyloid Disease of Kidney

Syn.: lardaceous or waxy disease of kidney.

Quantity: increased, commonly about 2,000 c.c.

Sp. Gr.: reduced, 1008–1014.

Colour: pale, clear.

Urea: daily excretion may be slightly diminished.

Albumin: scanty.

Sediment: hyaline and waxy casts (occasionally also fatty and granular).

Hyperæmia of Kidneys

A. **Active congestion**: such as may result from cold or irritants (turpentine, cantharides, cubebs, etc.), or in acute fevers, or in the very early stage of acute Bright's disease.

Quantity: usually diminished.

Sp. Gr.: little or no alteration.

Colour: high-coloured, occasionally blood-stained.

Albumin: present, usually small in amount.

Sediment: hyaline casts and a few renal epithelia; occasionally red blood-cells and leucocytes.

B. **Passive Congestion**: due to circulatory changes resulting from heart and lung disease or pressure on renal vein.

Quantity: diminished.

Sp. Gr.: raised, 1025.

Colour: high-coloured, or blood-stained.

Urea: unaltered.

Albumin: usually present in small quantity.

Sediment: hyaline casts, often long and ribbon-like; epithelium in excess; red blood-corpuscles common; occasionally blood casts.

Renal Infarction

Quantity: diminished.

Sp. Gr.: raised proportionately to quantity passed and amount of albumin.

Colour: high-coloured or blood-stained.

Albumin: appears suddenly, tendency to quickly diminish.

Sediment: casts, at first hyaline, later also epithelial. Red blood-corpuscles.

Hydronephrosis

Quantity: on the whole is diminished but is subject to great variation, at one time suppressed or greatly diminished, at another time superabundant.

Sp. Gr.: lowered.

Urea: reduced.

Albumin: occasionally a trace.

Sediment: excess of epithelial cells; a few red blood-corpuscles and leucocytes are not uncommon.

Pyonephrosis

The urine is similar to that of hydronephrosis, with the addition of pus (in variable quantity) and bacteria in the sediment; blood, epithelial cells, and mucus are more in evidence.

Cystic Disease of the Kidney

The urine is similar to that of chronic interstitial nephritis; hæmaturia is more pronounced.

Tubercular Disease of the Kidney

Quantity: slightly increased.

Sp. Gr.: normal.

Colour: pale, slightly turbid.

Reaction: usually acid, may become alkaline from mixed infection.

Albumin: a small quantity.

Sediment: pus-cells, tending to increase in quantity; a few red blood-cells are common, also caseous particles like crumbs of bread or cheese; the presence of the tubercle bacillus is alone conclusive; it can frequently be detected in the sediment by staining, cultivation, or inoculation (see plate XXXI).

Renal Calculus

The urine tends to show attacks of slight hæmaturia, brought on or aggravated by exercise; occasionally also some albumin (independent of blood).

Sediment: red blood-cells, and frequently an excess of epithelial cells, and crystals of uric acid and oxalate of lime. Later on the urine shows more the characters of pyelitis.

Renal Colic

Urine frequently normal: more often it shows blood, and has a noticeable tendency to deposit uric acid or oxalate of lime crystals; small calculi may be eliminated after attack; urine is occasionally suppressed.

Attacks of hæmaturia and pain, closely resembling renal colic, may be due to copious precipitation of crystals, especially oxalate of lime, in the renal tubules.

Renal Growths: malignant and benign

The most prominent feature is hæmaturia, often profuse, and occasionally containing blood-clots and moulds of the kidney-pelvis or ureter. A trace of albumin independent of blood may be present.

Casts, granular and hyaline, as well as many epithelial cells, are not uncommon in the sediment. It is impossible to diagnose malignancy from the character of the epithelium; and it is very doubtful if any fragments of tissue or collections of epithelial growth can be identified as malignant.

Pyelitis

Quantity: usually somewhat increased.

Sp. Gr.: correspondingly reduced.

Colour: pale yellow, with more or less turbidity.

Odour: heavy fœtid, or ammoniacal.

Reaction: acid or alkaline according to character of bacteria present.

Urea: daily excretion unaltered.

Albumin: a small quantity is commonly found after filtration.

Sediment: pus-cells and bacteria; epithelial cells increased (the presence of a large number of long-tailed, comma-shaped cells is considered by some writers as strongly suggestive); shreds of tissue and debris according to severity of case; red blood-cells are uncommon unless the condition arises from tubercle, calculus, or Bilharzia.

If the urine is ammoniacal, the earthy phosphates, crystals of stellar and triple phosphate, and ammonium urate are also found.

Pyelonephritis

Syn.: pyonephritis; suppurative or acute interstitial nephritis: surgical kidney.

Quantity: increased, 2,000 c.c. or more.

Sp. Gr.: diminished, 1005 to 1015.

Colour: pale yellow or blood-stained, turbid.

Odour: heavy fœtid, or ammoniacal.

Reaction: usually acid, may be alkaline.

Urea: diminished.

Albumin: present; independent of pus and more than amount of pus likely to account for.

Sediment: copious, forming a greyish-white layer of pus, debris, and bacteria; epithelial cells are more abundant than in pyelitis, and red blood-cells more common; hyaline and pus casts, occasionally casts of bacteria. If the urine is ammoniacal, the deposit also shows the crystalline substances characteristic of ammoniacal decomposition.

Cystitis

Quantity & Sp. Gr.: usually unaltered.

Colour: yellow or blood-stained; more or less turbid.

Odour: heavy fœtid, or ammoniacal.

Reaction: acid or alkaline, according to character of infection.

Urea: usually normal.

Albumin: some is usually found after filtering off pus and deposit.

Sediment: more or less pus, coagula of fibrin, epithelial cells, debris and bacteria; red blood-cells and blood coagula may be found, especially in advanced cases. In an acid cystitis the sediment is comparatively small and deposits quickly as a light-grey layer; in an alkaline cystitis it is usually more abundant, and forms in the course of time a viscid gelatinous deposit, with turbid urine above, and shows earthy phosphates, crystals of stellar and triple phosphate, and ammonium urate.

Vesical Calculus

The urine in the early stages may be perfectly normal; more often it shows the characters of cystitis (acid or alkaline) with the frequent appearance of red blood-cells, especially after movement. Fragments of calculus may be found; and not uncommonly deposits of uric acid or oxalate of lime.

Vesical Tuberculosis

The urine is that of cystitis, usually acid, and showing the tubercle bacillus in the sediment.

Vesical Growth: malignant and benign

The urine is that of cystitis, with marked hæmaturia. The sediment shows blood-cells, blood-clots, fibrin coagula, and epithelium. The character of the epithelial cells and fragments of epithelial tissue in the sediment cannot be relied upon in forming a diagnosis of malignancy.

Inflammation of Spermatic Vesicles

Syn.: spermato-cystitis.

The existence of rusty masses of semen, pus, and blood-corpuscles, eliminated in the early part of micturition, is strongly suggestive of this condition. The glairy masses when stained will often show the presence of gonococci.

Deep Urethra and Prostate

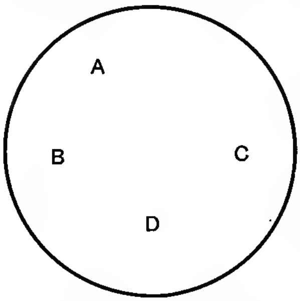
Chronic changes about the deep urethra and prostate—such as chronic prostatitis and urethritis—are apt to show a collection of glairy mucus and fibrinous threads, floating in an otherwise clear urine. These are evacuated in the first few ounces of urine, and appear under the microscope as fibrin coagula, studded over with pus and large round epithelial cells: when stained they frequently show gonococci.

LIST OF PLATES OF URINARY DEPOSITS

PLATE I.

COMMON FORM OF URIC ACID CRYSTAL.

Uric Acid ($C_5 H_4 N_4 O_3$) is deposited in the urine commonly as lozenge-shaped crystals with pointed ends. They are almost always coloured by the urinary pigment, and vary in shade from a faint yellow to a deep brown; in a pale urine they may be colourless.

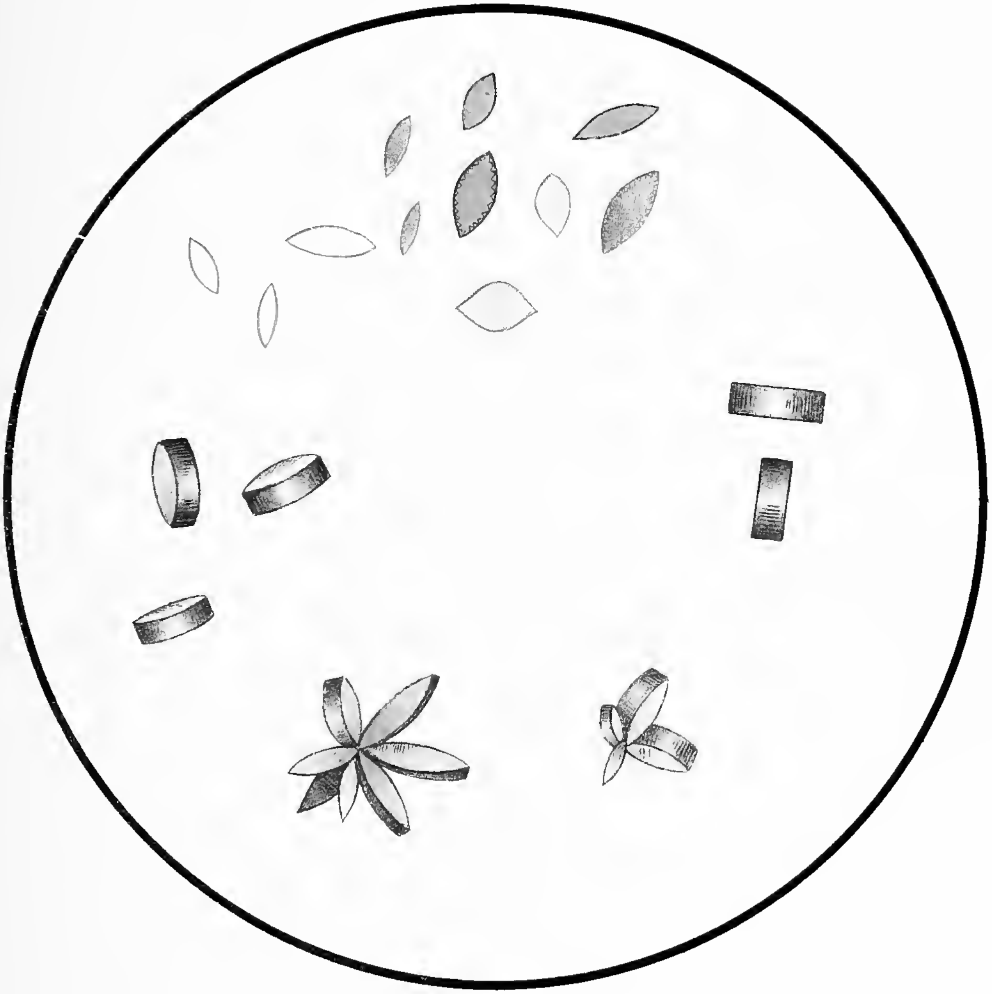


The crystals usually lie on the flat surface of the lozenge as at A, or may rest with their shorter diameter partially tilted as at B, or vertical as at C. They are frequently arranged in stellate groups or rosettes as at D.

Uric acid crystals are often visible to the naked eye as small brown grains, like cayenne pepper, at the bottom of the urine glass. The precipitation of uric acid in the urine is of much interest, owing to its tendency to form gravel and calculus.

All crystals of uric acid are insoluble in acetic and hydrochloric acids, but are dissolved by nitric acid.

PLATE I.



COMMON FORM OF URIC ACID CRYSTAL.

Magnification × 216.

PLATE II.

OTHER FORMS OF URIC ACID CRYSTALS.

Magnification × 216.

From 30 to 40 crystalline varieties of uric acid have been described, the majority being modifications of the primary rhombic form.

At the upper part of the plate are four crystals, marked with fine lines of crystallisation; when seen on their edge, they appear to be flattened whetstones. Most of the others are aggregations of rod-shaped crystals, some with sharply-pointed spicules attached. Below are two arranged in a bundle like a wheat-sheaf.

PLATE II.

OTHER FORMS OF URIC ACID CRYSTALS.

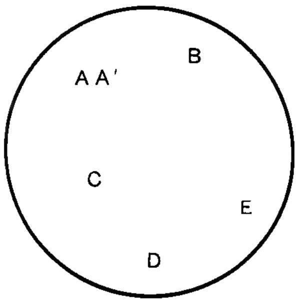
Magnification × 216.

PLATE III.

RARER FORMS OF URIC ACID.

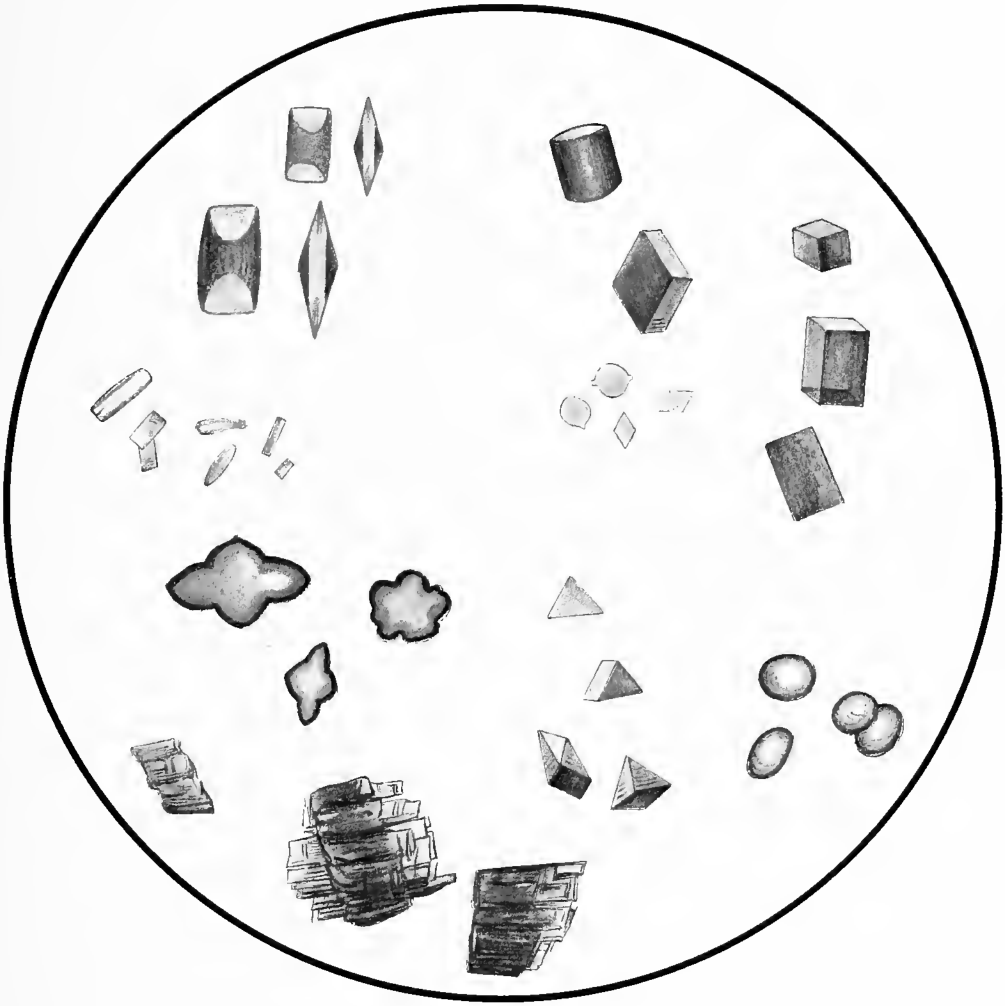
Magnification × 216.

THE crystals at A are rather uncommon modifications of the whetstone; they are represented resting on their broad surface. At A′ they are seen resting on their edge, and show deeply shaded angles.



At B is a barrel-shaped form; below and to the right are several cubical blocks of uric acid. At C the crystals are halbert-shaped. D represents irregular masses with secondary lines of crystallisation. At E are four spherical masses—colloidal form of uric acid.

PLATE III.



RARER FORMS OF URIC ACID.

Magnification × 216.

PLATE IV.

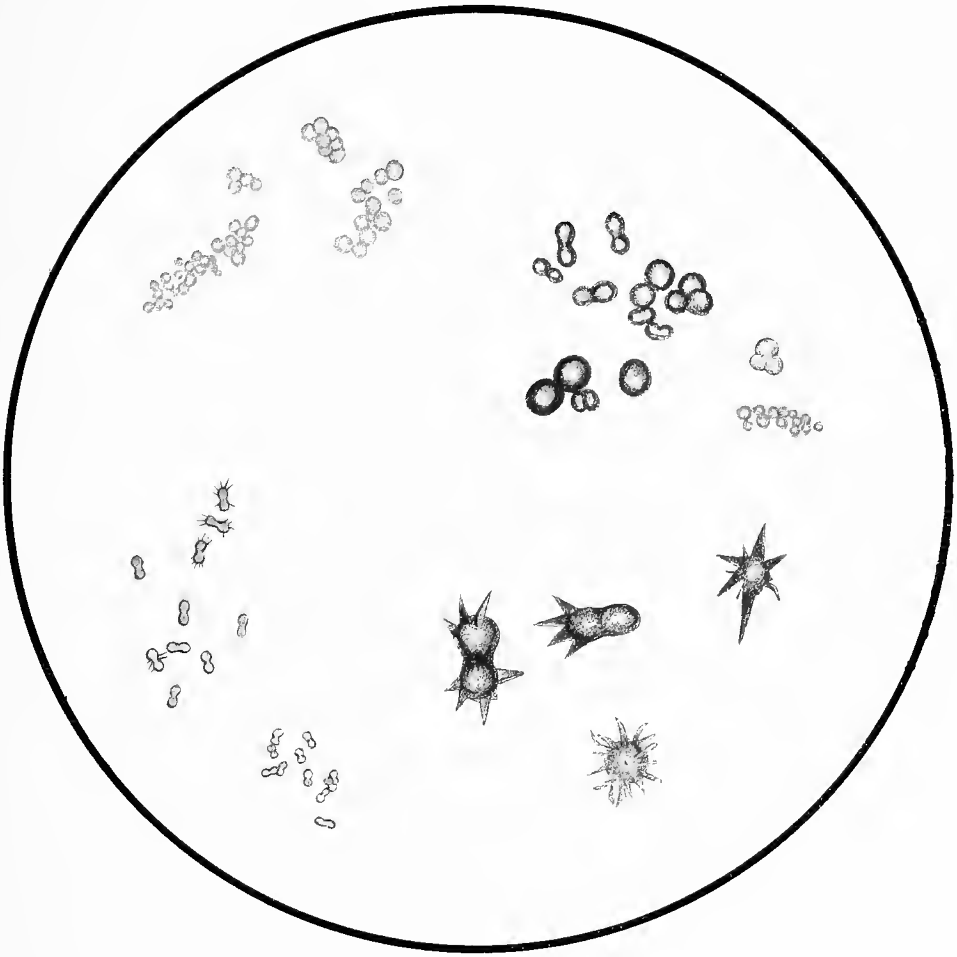
CRYSTALS OF AMMONIUM URATE.

Magnification × 216.

THESE crystals are found in urines alkaline from ammoniacal decomposition, either as globular masses lying singly and in groups, or as small dumb-bells. They are pigmented, and vary in colour from a pale yellow to a deep brown. Some of them are covered with numerous spicules (hedge-hog or thorn-apple crystals).

Ammonium urate is deposited in the urine before emission in cases of alkaline cystitis and pyelitis, along with crystals of ammonio-magnesic phosphate.

PLATE IV.



CRYSTALS OF AMMONIUM URATE.

Magnification × 216.

PLATE V.

CRYSTALS OF ACID SODIUM URATE.

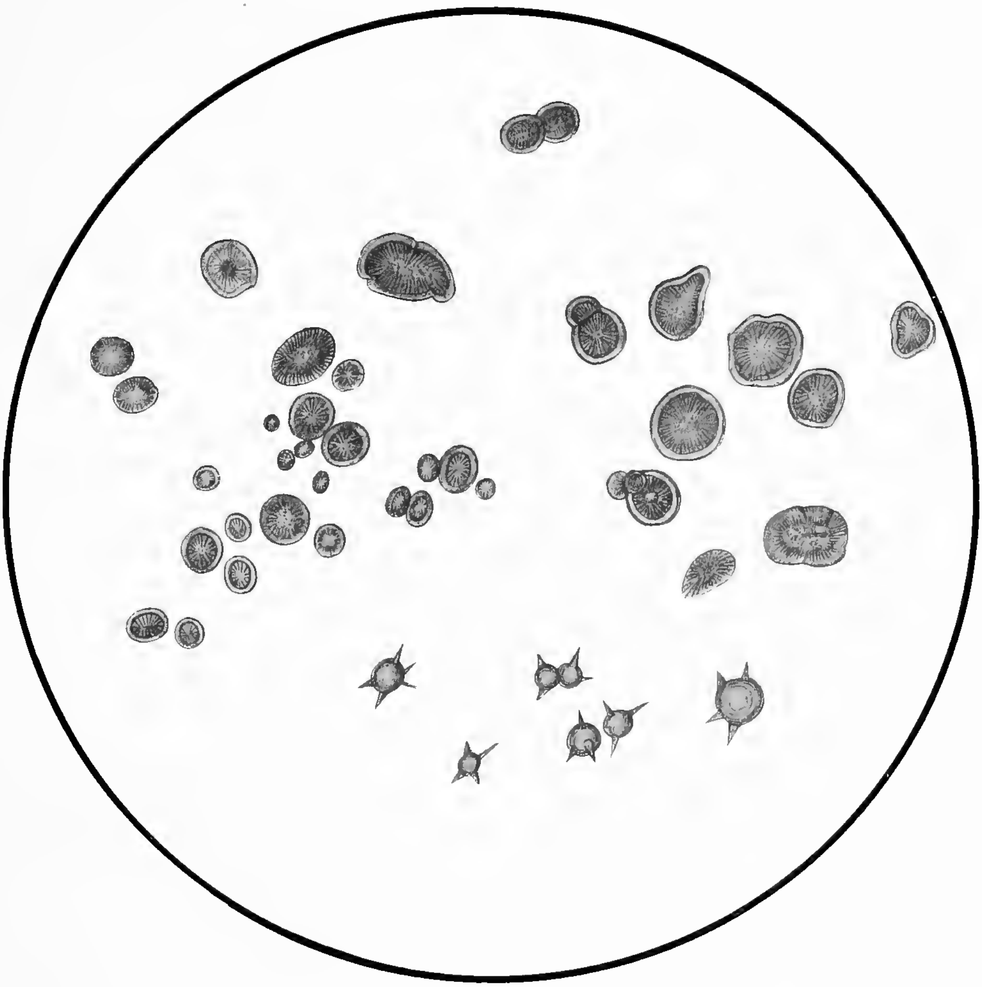
Magnification × 216.

ACID SODIUM URATE is a rare urinary deposit. It is found in acid urines, in cases of gout and in febrile conditions, particularly of children. Like all urates, it tends to take up the urinary pigment.

The crystals are spherical, many of the spheres having a radiating appearance, as if composed of close aggregations of the fine acicular crystals of sodium urate.

Others of the spheres are covered with fine spicules, like the hedge-hog crystals of ammonium urate.

PLATE V.



CRYSTALS OF ACID SODIUM URATE.

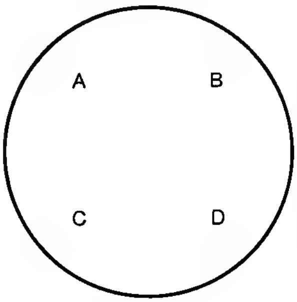
Magnification × 216.

PLATE VI.

OCTAHEDRA OF OXALATE OF LIME.

Magnification × 216.

Oxalate of Lime ($Ca\ C_2\ O_4,\ 2H_2\ O$) crystallises generally as octahedra, composed of two four-sided pyramids joined at their bases and flattened in one axis. According to the axis on which they lie, they appear as square (A) or diamond-shaped (B) crystals, marked by double diagonal lines. By pressure on the cover-glass they can be made to roll over in the microscope field, their appearance varying greatly with their position. At C the octahedra are shown resting obliquely on one of the triangular sides of a pyramid; at D the crystals are resting vertically upon one of the sides of the square base where the pyramids join. Octahedra of oxalate of lime are colourless, highly refractive crystals; they do not transmit light between crossed Nicol prisms.

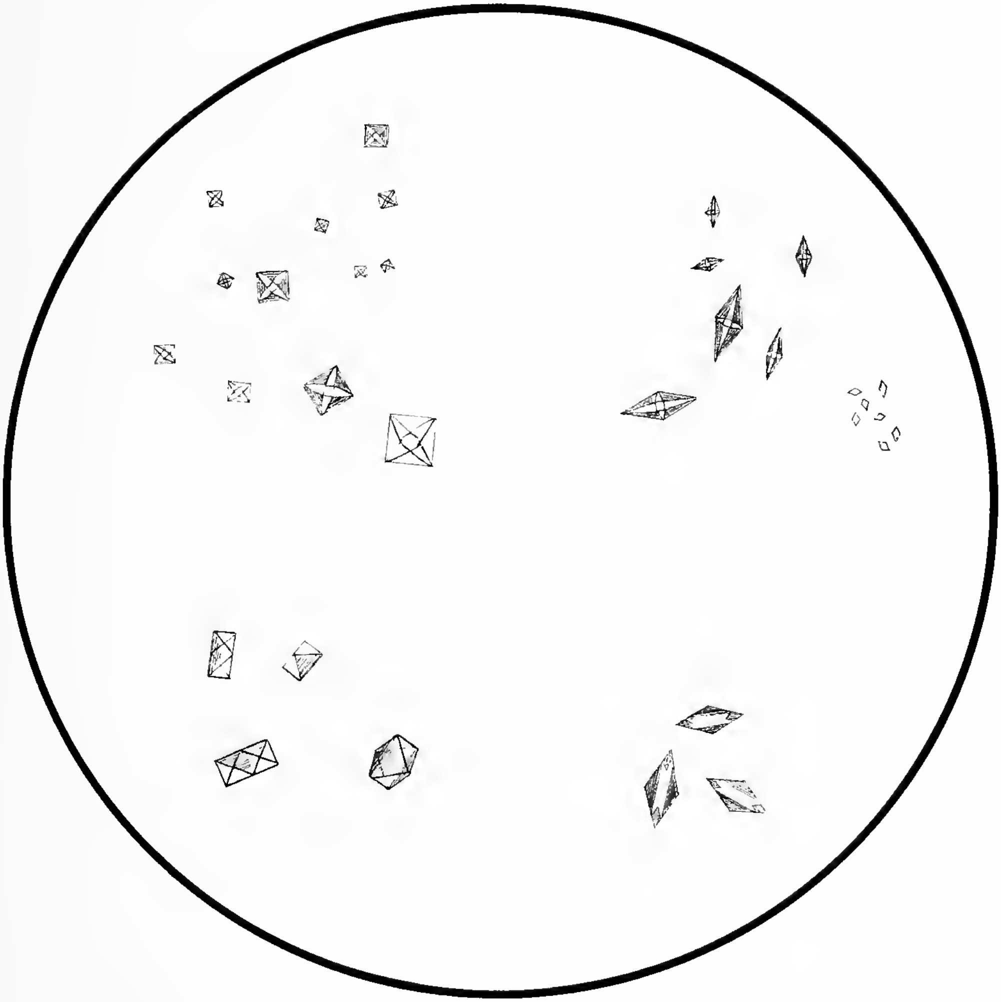


Oxalate of lime is, in my experience, the most common crystal in urine which has not undergone alkaline decomposition. If deposited while the urine has been left standing in the urine-glass, the crystals appear like a fine white dust on the surface of the mucus, already at the bottom of the vessel, forming, as it were, a delicate white fringe along its wavy outline. This appearance is sometimes so distinctive as to enable one to determine their presence by the naked eye alone

All crystals of oxalate of lime are insoluble in acetic acid, but are readily dissolved by hydrochloric and nitric acids.

Oxalate of lime contributes largely towards the formation of calculi, especially towards the smaller renal stones and gravel.

PLATE VI.



OCTAHEDRA OF OXALATE OF LIME.

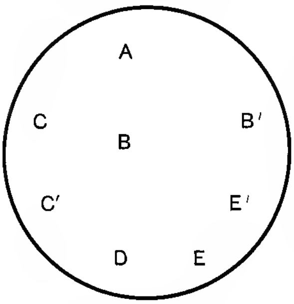
Magnification × 216.

PLATE VII.

RARE FORMS OF OXALATE OF LIME.

Magnification × 216.

OCCASIONALLY the pyramids of the octahedron remain unjoined as depicted at A, or have a cubical block of variable length interposed as at B; when resting vertically on the apex of a pyramid, these present the appearance shown at B′. Occasionally one meets with twin crystals—one octahedron, with its angles projecting through the sides of the other (C), or a small octahedron with a larger one crystallised round it (C′).



At D, peculiar lozenge-shaped tablets are shown; Neubauer and Vogel, and Beale have depicted similar crystals. I have found these deposited only in two cases—the sediment, from which they were drawn, showed also colloidal forms, namely, biconcave, biscuit-shaped crystals (F) and spheres (F′), the one form seeming to gradually merge into the other. They all gave the chemical reactions of oxalate of lime, and transmitted light between crossed Nicol prisms.

PLATE VII.

RARER FORMS OF OXALATE OF LIME.

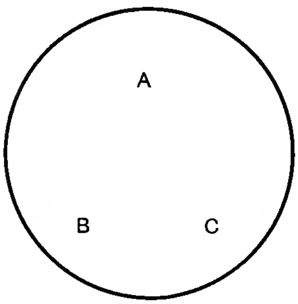
Magnification × 216.

PLATE VIII.

COLLOIDAL FORM OF OXALATE OF LIME.

Magnification × 300.

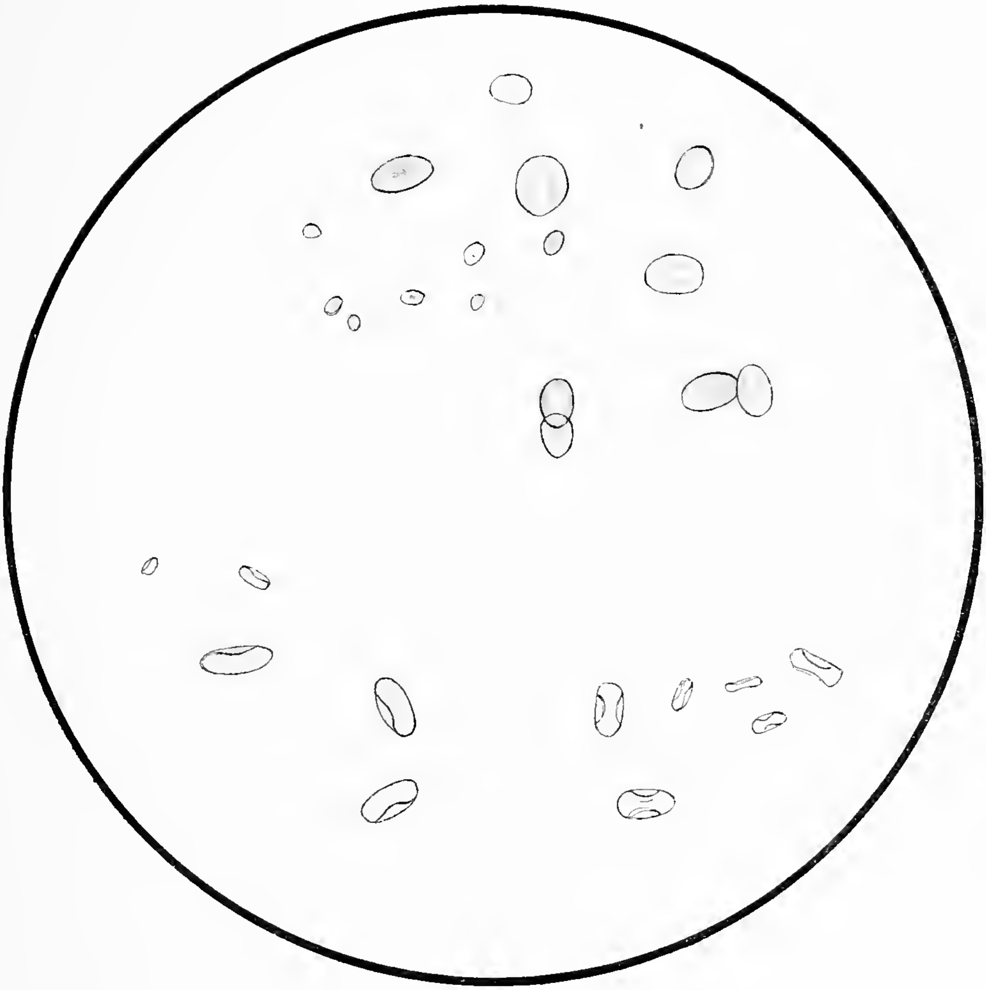
THESE crystals represent the so-called dumb-bells of oxalate of lime; they are in reality biconcave, oval bodies, shaped somewhat like a biscuit. At A they are shown resting on their flat surface. The smaller ones are difficult to distinguish from yeast-cells, unless highly magnified. At B they are shown with the shorter diameter of the biscuit partially tilted; at C they are resting vertically on their edges, and have the appearance of dumb-bells.



These crystals have a rather translucent appearance; they give the usual chemical reactions of oxalate of lime; but, unlike the octahedra, transmit light between crossed Nicol prisms, showing concentric bands of colour like small, bright mother-of-pearl buttons studded over the darkened field of the microscope.

Such a form of crystal and others allied to it depends, as Ord has shown, upon the slow crystallisation of salts from the urine in the presence of colloidal substances.

PLATE VIII.



COLLOIDAL FORM OF OXALATE OF LIME.

Magnification × 300.

PLATE IX.

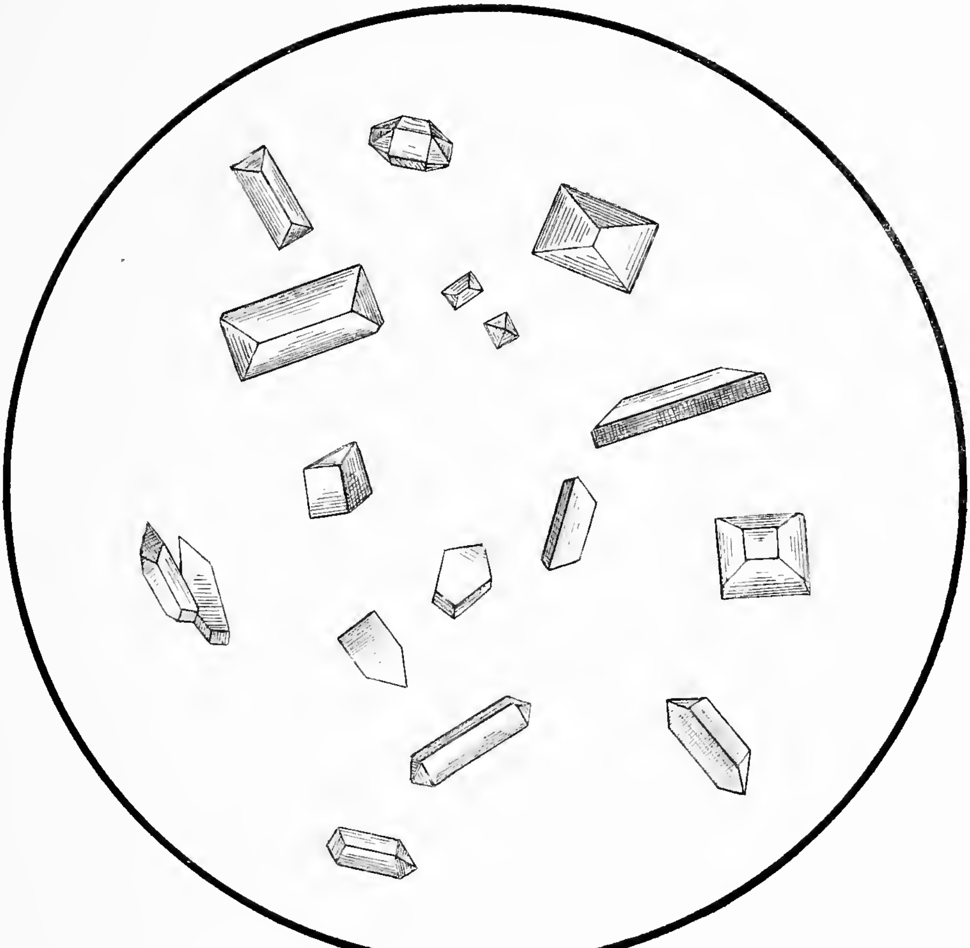
CRYSTALS OF AMMONIO-MAGNESIC PHOSPHATE, OR TRIPLE PHOSPHATE. $MgNH_4\ PO_4, 6H_2O$.

Magnification × 216.

Colourless Crystals found in urines in which ammoniacal decomposition has taken place, and frequently deposited before emission in cystitis and pyelitis. They are met with in an endless variety of shape, the prevailing form being a triangular prism with bevelled ends (coffin-lid crystals).

They are dissolved by acetic, hydrochloric, and nitric acids, and transmit light between crossed Nicol prisms: the smaller bevelled crystals can thus be distinguished from octahedra of oxalate of lime, which they often resemble. They contribute towards the formation of secondary calculi.

PLATE IX.



CRYSTALS OF AMMONIO-MAGNESIC PHOSPHATE OR TRIPLE PHOSPHATE $Mg\,NH_4\,PO_4$.

Magnification × 216.

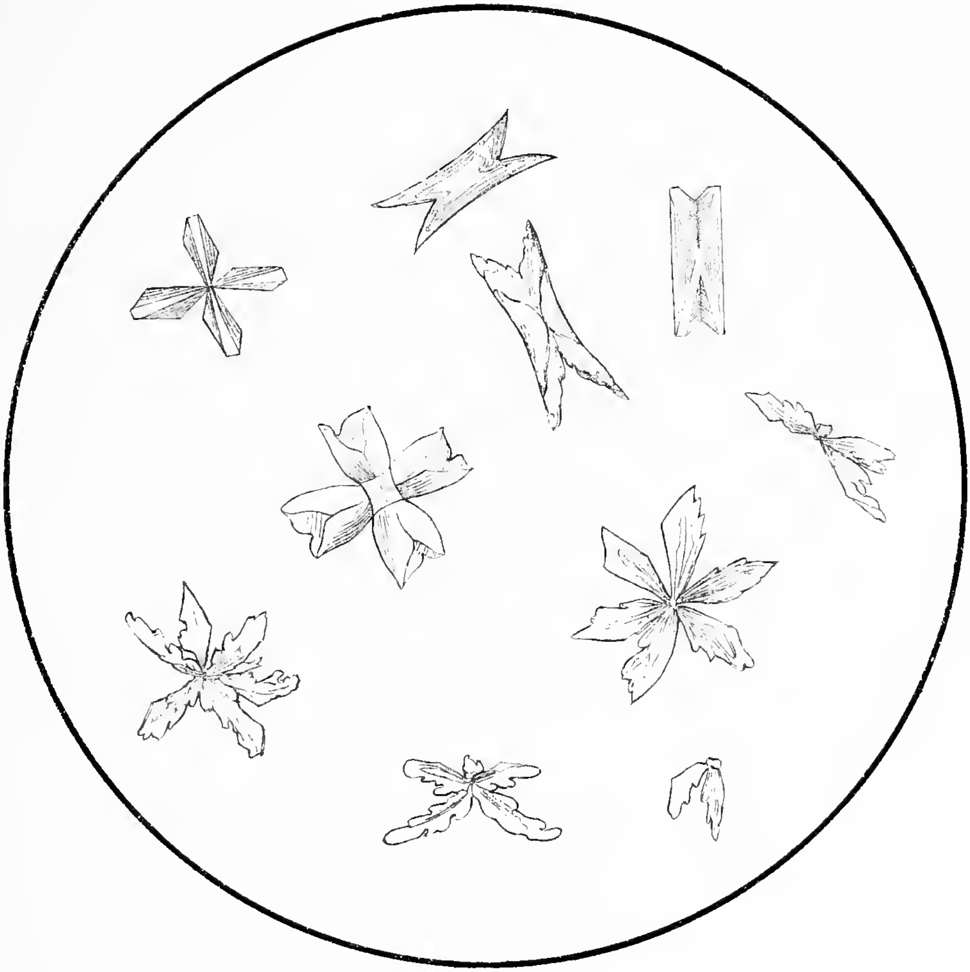
PLATE X.

RARER FORMS OF AMMONIO-MAGNESIC PHOSPHATE.

Magnification × 216.

SUCH crystals are most commonly deposited in highly albuminous urines, and seem to be colloidal forms of the triple phosphate. The variety and beauty of their formation are remarkable. The fine, feathery crystals (feathery phosphates) can readily be prepared by the addition of a little gelatine and ammonia to the urine. They present the chemical and polariscopic properties of other triple phosphates.

PLATE X.



RARER FORMS OF AMMONIO MAGNESIC PHOSPHATE.

Magnification × 216.

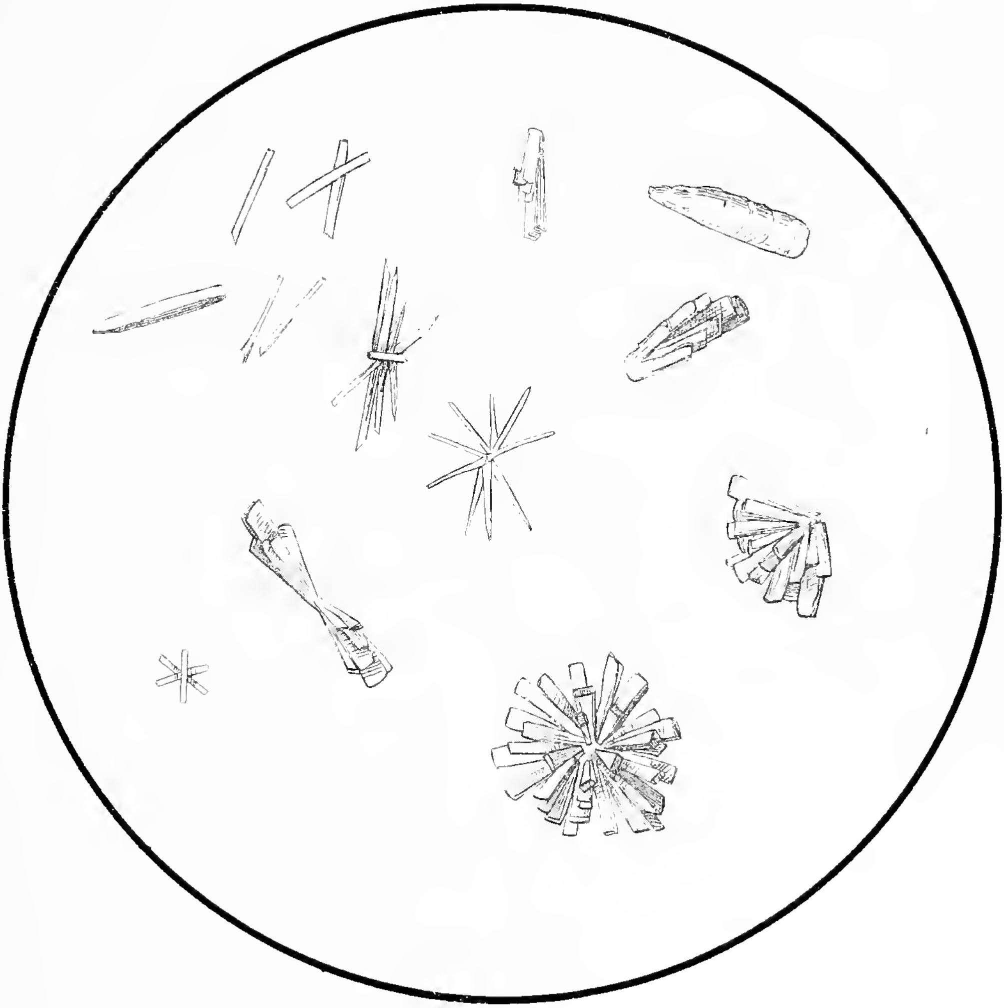
PLATE XI.

CRYSTALLINE PHOSPHATE OF LIME, OR STELLAR PHOSPHATE. $CaHPO_4, 2H_2O$.

Magnification × 216.

THIS salt is deposited in feebly acid, neutral, and alkaline urines as rod-like or club-shaped crystals, commonly arranged as rosettes and fan or sheaf-shaped bundles. They are frequently marked with lines of secondary crystallisation. Acetic, nitric, and hydrochloric acids dissolve them. They enter into the formation of phosphatic calculi.

PLATE XI.



CRYSTALLINE PHOSPHATE OF LIME OR STELLAR PHOSPHATE.

Ca HPO_4, $2H_2O$.

Magnification × 216.

PLATE XII.

CARBONATE AND SULPHATE OF LIME.

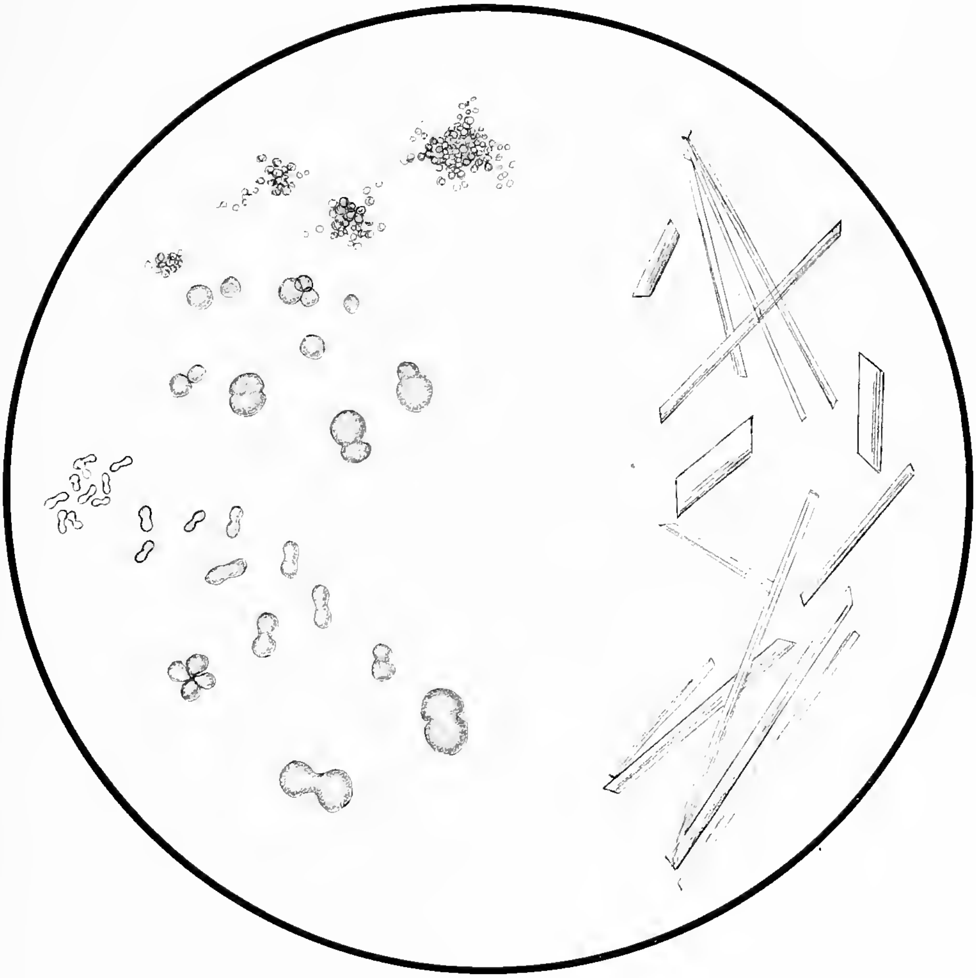
Magnification × 216.

Crystals of carbonate of lime are shown on the left-hand side of the plate. They are very rarely found in the urine of man. When alkaline decomposition is far advanced, colourless spheres of this salt may be deposited; and are usually minute and aggregated into clumps, somewhat like an amorphous sediment. More rarely they are found as dumb-bells. Large spheres and dumb-bells are readily obtained from the urine of the horse.

Crystals of carbonate of lime are dissolved by acetic acid with effervescence, and show a dark cross between crossed Nicol prisms. They derive some interest in that they contribute occasionally towards the formation of calculi.

Crystals of sulphate of lime are drawn at the right-hand side of the plate. They are extremely rare in the urine, and appear as long colourless needles and tablets, insoluble in ammonia and acids.

PLATE XII.



CARBONATE AND SULPHATE OF LIME.

Magnification × 216.

PLATE XIII.

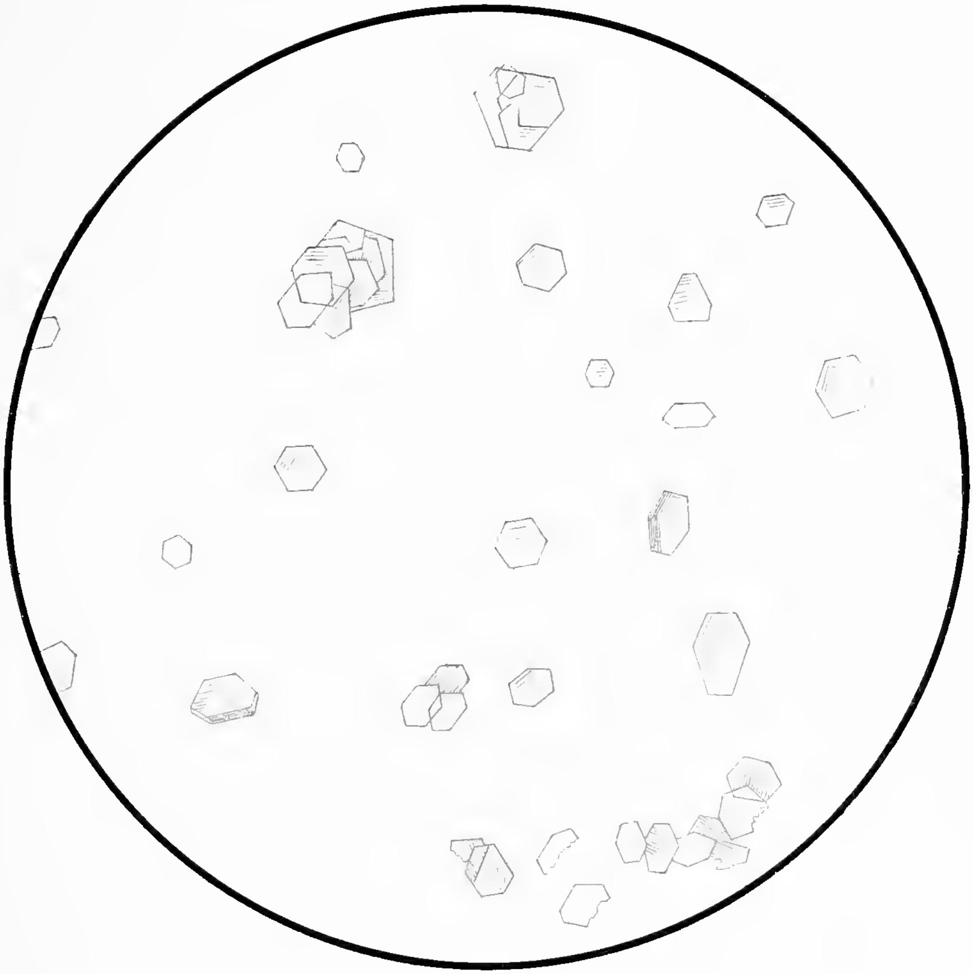
CRYSTALS OF CYSTINE.

Magnification × 216.

CYSTINE or Cystic Oxide is deposited in the urine as colourless, six-sided tablets, frequently lying super-imposed on one another, and marked with lines of crystallisation. They are insoluble in acetic acid, but are readily soluble in ammonia; they are also dissolved by hydrochloric acid, by which means they can be distinguished from similar looking plates of uric acid.

Cystine ($C_3H_6NSO_2$) is a rare urinary deposit; its occurrence is associated chiefly with deranged nutritive processes, in which the liver plays a part. It occasionally collects to form gravel and calculi, the tendency to such formation running frequently in families.

PLATE XIII.



CRYSTALS OF CYSTINE.

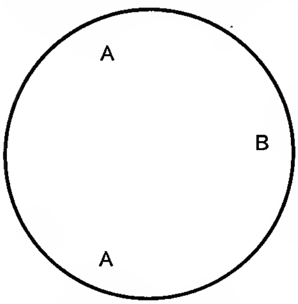
Magnification × 216.

PLATE XIV.

TYROSINE AND LEUCINE.

Magnification × 216.

A. TYROSINE (C_9H_{11} NO_3) is deposited in the urine as fine, acicular crystals, arranged usually in sheaf-like bundles; occasionally they form rosettes, or lie loosely. They are insoluble in acetic acid, alcohol, and ether; soluble in ammonia, hydrochloric acid, and hot water.



B. Leucine ($C_6H_{13}NO_2$) when pure crystallises as delicate plates; when less pure it forms spheres with a slightly radiating structure. When very impure it appears as refractile, globular masses, sometimes showing a light centre and a dark margin, or *vice versa*; this being the form in which it generally appears in the urine. It is insoluble in ether (which distinguishes it from oil globules) and in hydrochloric acid; soluble in caustic alkalies.

Tyrosine and leucine are derived from the decomposition of proteids within the system; they are extremely uncommon urinary deposits, and usually occur together. They have been found in leucæmia, typhus, acute phosphorus poisoning, and frequently in acute yellow atrophy of the liver.

PLATE XIV.

TYROSINE AND LEUCINE.

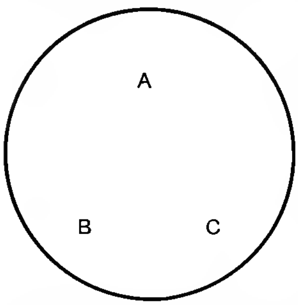
Magnification × 216.

PLATE XV.

CRYSTALS OF HIPPURIC ACID, CREATININE, AND CHOLESTRINE.

Magnification × 216.

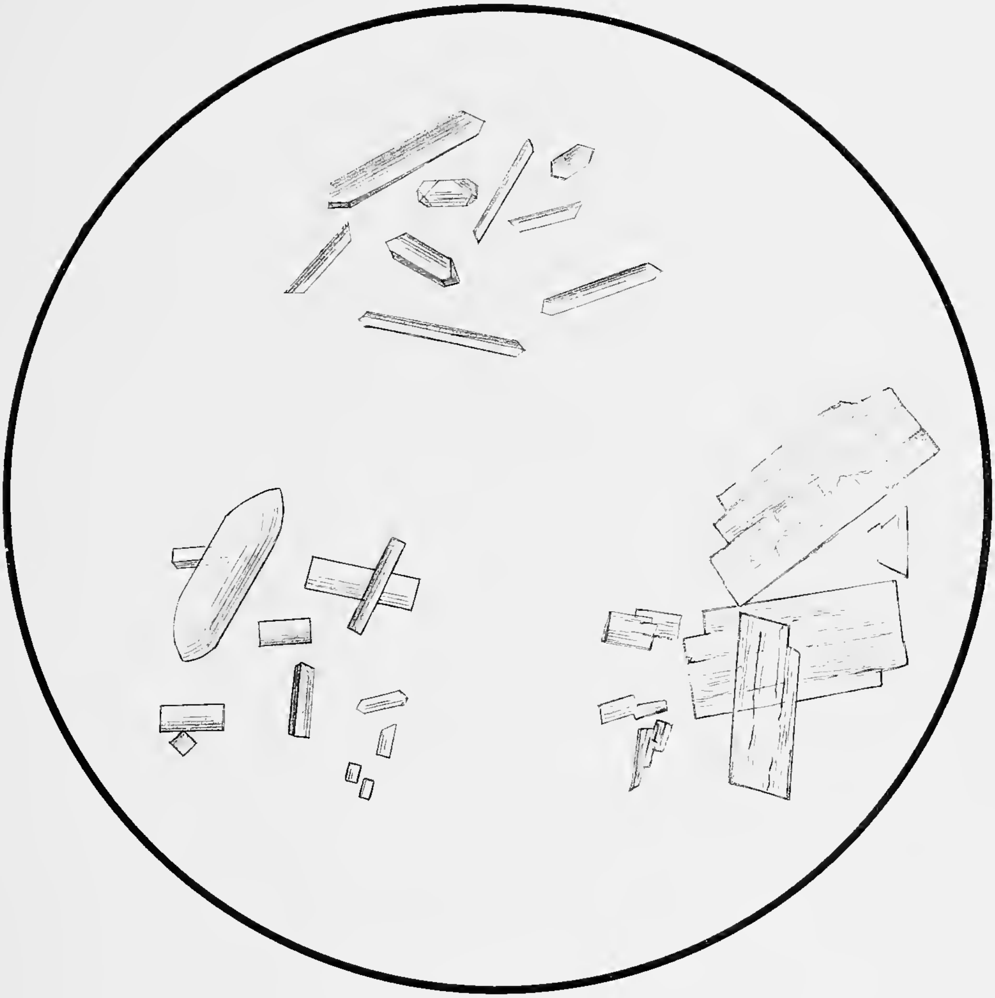
A. CRYSTALS of hippuric acid ($C_9H_9\ NO_3$) occur in the urine as four-sided prisms with bevelled ends. They are soluble in ammonia, and insoluble in hydrochloric acid. They may appear after partaking of certain drugs—especially benzoic acid—and fruits—cranberries, bilberries, and prunes.



B. Creatinine never occurs as a spontaneous deposit. It can be crystallised from the urine artificially as colourless, oblong prisms. It is a constant constituent of normal urine, from one-half to one gramme being excreted daily. It is closely allied to creatine, the latter containing one more molecule of H_2O.

C. Cholestrine is found as clear, rhombic plates, with a tendency to be marked by longitudinal striae, and to have a rectangular niche in the corner. They are insoluble in acids and alkalies. The spontaneous deposition of cholestrine in the urine is of great rarity.

PLATE XV.



CRYSTALS OF HIPPURIC ACID, CREATININE, AND CHOLESTRINE.

Magnification × 216.

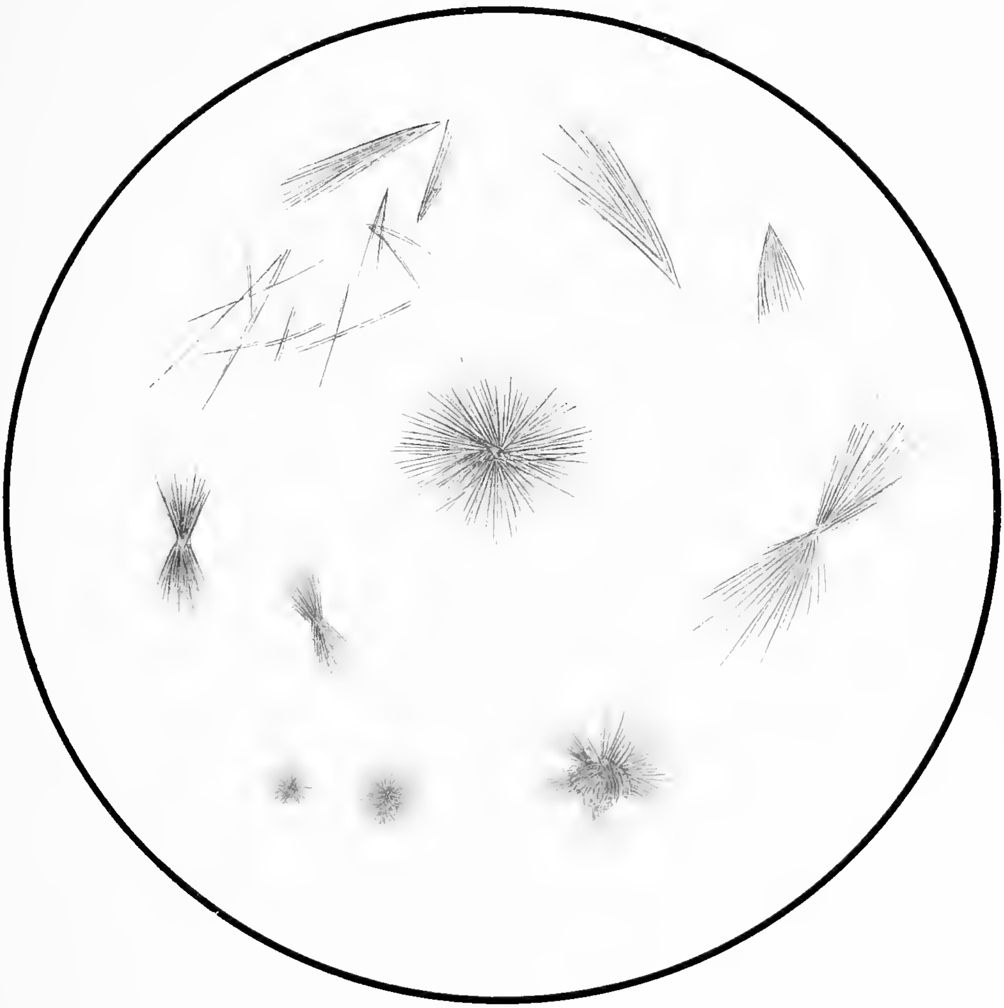
PLATE XVI.

CRYSTALS OF PHENYL-GLUCOSAZONE.

Magnification × 216.

PHENYL-GLUCOSAZONE is deposited when the phenylhydrazine test is applied to urine containing glucose (see p. 30), as fine needle-like crystals of a canary-yellow colour, lying loose, or in bundles like sheaves and rosettes. They melt at a temperature of 204° C., which distinguishes them from a similar compound of a glycuronic acid, melting at 150° C.

PLATE XVI.



CRYSTALS OF PHENYL-GLUCOSAZONE.

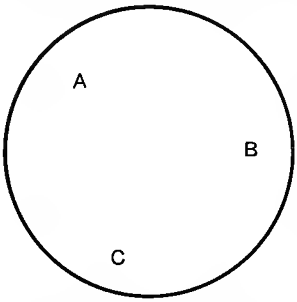
Magnification × 216.

PLATE XVII.

PUS-CELLS FROM THE URINE.

Magnification × 300.

A. PUS-CELLS appear as granular spheres, about one-third larger than a red blood-corpuscle, and of a grey or yellowish-grey colour.



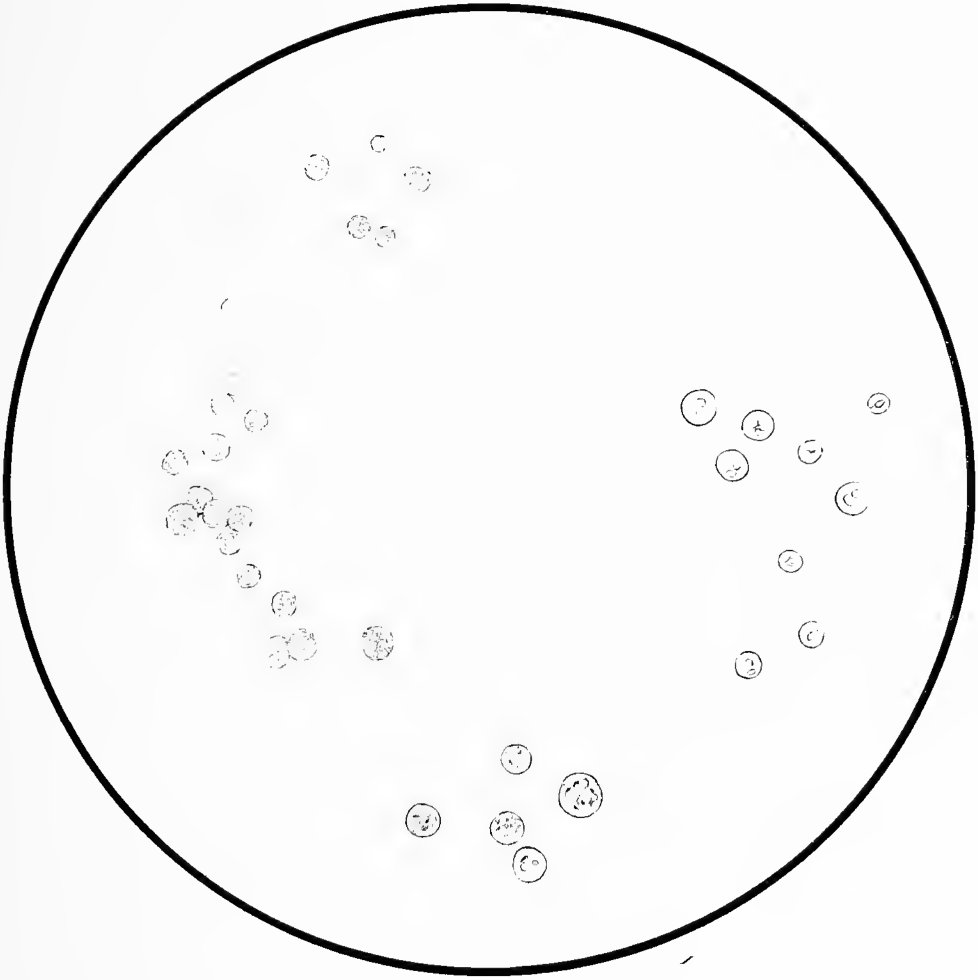
They have a well-defined, circular outline, and may show one, two, three, or four nuclei. By insinuating a little acetic acid under the cover-glass, their substance swells and loses its granular appearance, making the nuclei and outline stand out prominently as at B, enabling them to be distinguished from red blood-corpuscles. In ammoniacal urine they frequently have the appearance shown at C.

Pus deposits in the urine-glass as a greyish-yellow layer with a sharply-defined, horizontal margin. The addition of liquor potassæ gives it a glairy gelatinous consistence, so that it adheres to the glass, or pours out as a stringy viscid mass.

Urine containing pus is turbid, and necessarily contains pathogenic bacteria. It generally shows the presence of albumen on applying the usual tests, even when the pus cells are removed by filtration: the quantity is, as a rule, small; when large and apparently out of proportion to the amount of pus, a diligent search should be made for casts.

The most frequent source of pus is the urethra in the male, and the vagina in the female. It may come from any part of the urinary tract, and the simultaneous presence of epithelium and other structures from the affected part will help to indicate its seat of origin. It must be remembered that a few mucous or lymph-corpuscles, indistinguishable from pus-cells, are to be found normally in urine, especially if the centrifuge is used to collect the deposit.

PLATE XVII.



PUS-CELLS FROM THE URINE.

Magnification × 300.

PLATE XVIII.

RED BLOOD-CORPUSCLES AS FOUND IN URINE.

Magnification × 300.

RED BLOOD-CELLS vary in appearance according to the length of time they have been in the urine, and according to its density, reaction and composition. In a fresh urine they usually appear as at A, their biconcave character being made out by focusing, or by getting them to roll over on their edge as at A′. In a urine of low density they tend to become pale in colour, swollen, and globular, as shown at B. In a highly acid and concentrated specimen they may become shrunken, globular, and of a deeper tint, as at C. If retained long in the urine their pigment becomes washed out, but their biconcave formation is still in evidence, for they show a dark centre and a light margin, which changes to a light centre and dark margin on focusing (D). When long in contact with the urine they also become crenated (D′, E, and E′). In ammoniacal urine they rapidly become crenated and broken up. Red blood-corpuscles may resemble pus-cells; the latter are distinguished by their colour, larger size, granular and spherical appearance, and by the addition of acetic acid. Yeast-cells and spores of fungi, if isolated, sometimes closely resemble them; these, however, tend to form chains or buds, to be oval rather than round, and to show a nucleus. Small, colloidal spheres and ovoids may occasionally cause confusion; they are to be identified by their chemical reactions, and by the fact that they transmit light between crossed Nicols.

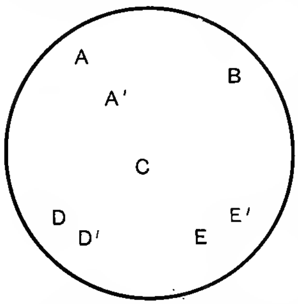


PLATE XVIII.

RED BLOOD-CORPUSCLES AS FOUND IN URINE.

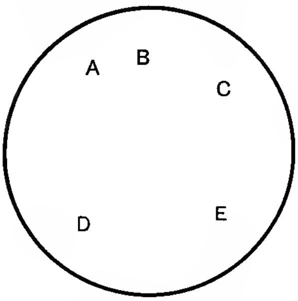
Magnification × 300.

PLATE XIX.

EPITHELIAL CELLS FROM THE URINARY TRACT.

Magnification × 216.

STRATIFIED EPITHELIA, like those at A, may come from the bladder, prepuce, or vagina. Cells like those at B, C, and D represent transitional cells, and come from the deeper layers of epithelium in any part of the urinary tract, from the meatus urinarius to the pelvis of the kidney. Epithelia like those at D are frequently glandular, and come from the deep urethra and vagina in health and disease. Some of them cannot be distinguished from the cells at E, which bear the characters of kidney epithelia, and are drawn from an abundant epithelial deposit in a case of acute tubal nephritis.



Most recent writings tend to show that it is not possible to determine the *exact* locality from which urinary epithelia are derived; the preponderance of any particular type would, however, help to indicate their seat of origin. Thus, a large number of long-tailed comma-shaped cells would suggest the existence of a pyelitis, and the presence of many circular epithelial cells, like those at D, in an otherwise normal urine, would suggest some change about the deep urethra.

PLATE XIX.

EPITHELIAL CELLS FROM THE URINARY TRACT.

Magnification × 216.

PLATE XX.

HYALINE CASTS.

Magnification × 216.

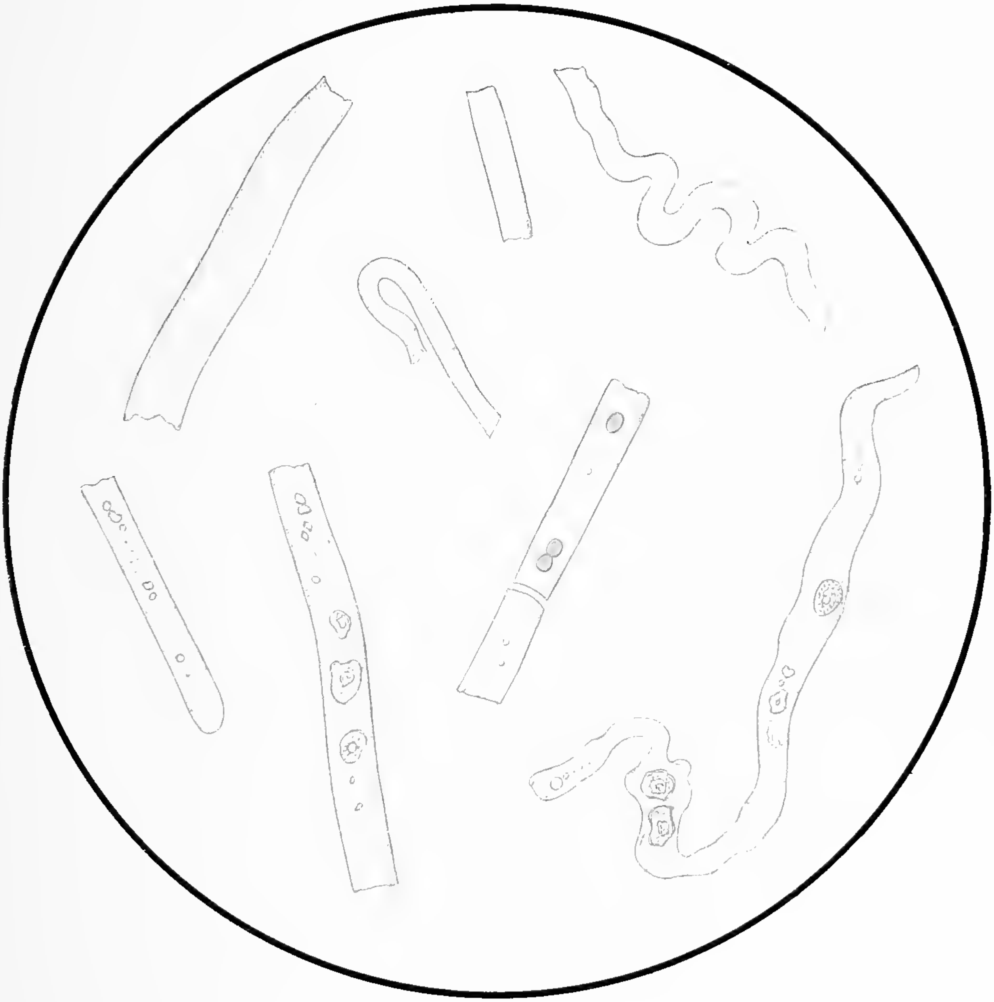
HYALINE CASTS are transparent, cylindrical bodies differing in size and shape, according to the variety of kidney tubule, in which the hyaline material has coagulated. As a rule, they are difficult to detect unless stained (see Plate XXIII.), and are best searched for with an inch power and narrow diaphragmatic aperture.

They occur in all varieties of kidney disease. Henle, Nothnagel, and von Jaksch have found them where the kidneys were quite healthy. Bartels and Purdy hold that they occur only in urines which are albuminous, or have recently been so. What experience I have had leads me to the belief that their presence is equally indicative of kidney affections as the presence of albumin, and that they should be regarded with equal suspicion (see also p. 24).

Hyaline material is probably one of the derivatives of albumin excreted in the urine, and may be regarded as the ground substance or stroma of all casts. It is common to see granular and fatty debris, epithelium, and blood-cells within its substance or adhering to it.

The long ribbon-like cast, in the right lower quadrant of the plate, is like one of the "cylindroids" of Thomas. It was drawn from a case of mitral disease, with slight passive kidney congestion.

PLATE XX.



HYALINE CASTS.

Magnification × 216.

PLATE XXI.

WAXY CASTS.

Magnification × 216.

WAXY CASTS are found in various acute and chronic kidney affections, but are most common in amyloid disease. They closely resemble hyaline casts in appearance, but are somewhat less transparent, and differ also in their micro-chemical reactions. They are said to occasionally give the characteristic amyloid colour with iodo-potassic-iodide solution and methyl-violet.

PLATE XXI.

WAXY CASTS

Magnification × 216.

PLATE XXII.

GRANULAR CASTS.

Magnification × 216.

GRANULAR CASTS occur in all forms of kidney disease, but are chiefly associated with the chronic varieties. As a rule the granules are fine and grey in colour, but may be brownish and coarse. Large quantities of fine granular cylinders are usually found in chronic interstitial nephritis, and may be detected in the centrifugalised sediment, even when no trace of albumin is to be found. Their presence is always indicative of an organic kidney lesion.

PLATE XXII.

GRANULAR CASTS.

Magnification × 216.

PLATE XXIII.

EPITHELIAL AND FATTY CASTS.

Magnification × 216.

Fatty and Epithelial Casts are found together, a portion of the cast usually showing epithelial cells in various stages of fatty degeneration. Large numbers are to be found in the urine in "large white kidney." Their structure is well brought out by staining. All varieties of casts may be readily stained as follows:—Fill up a test tube of the urine containing them, including, by preference, a good deal of the sediment. Add a few drops of carbol-fuchsin solution—such as is used for staining tubercle bacilli—and leave it to stand. The sediment will show the pigmented casts in eight to twelve hours. Casts, when stained, are less liable to escape detection.

PLATE XXIII.

EPITHELIAL AND FATTY CASTS.

Magnification × 216.

PLATE XXIV.

BLOOD CASTS FROM THE KIDNEY.

Magnification × 216.

Blood Casts of the kidney tubules may be formed solely of closely packed blood-corpuscles. It is more common to find them composed of epithelial cells, granular debris, and red blood-corpuscles, held together by hyaline material. They are a certain indication of hæmorrhage from the kidney substance, and are found in acute and chronic nephritis, in tubercular and surgical kidney, in passive renal congestion, and in certain abnormal blood states like purpura and leucocythæmia.

PLATE XXIV.

BLOOD CASTS FROM THE KIDNEY.

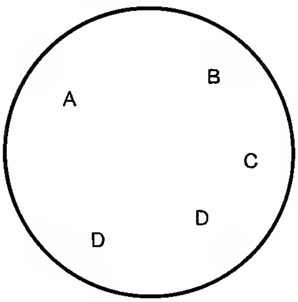
Magnification × 216.

PLATE XXV.

FALSE CASTS.

Magnification × 216.

VARIOUS structures may be arranged so as to resemble casts of the kidney tubules.



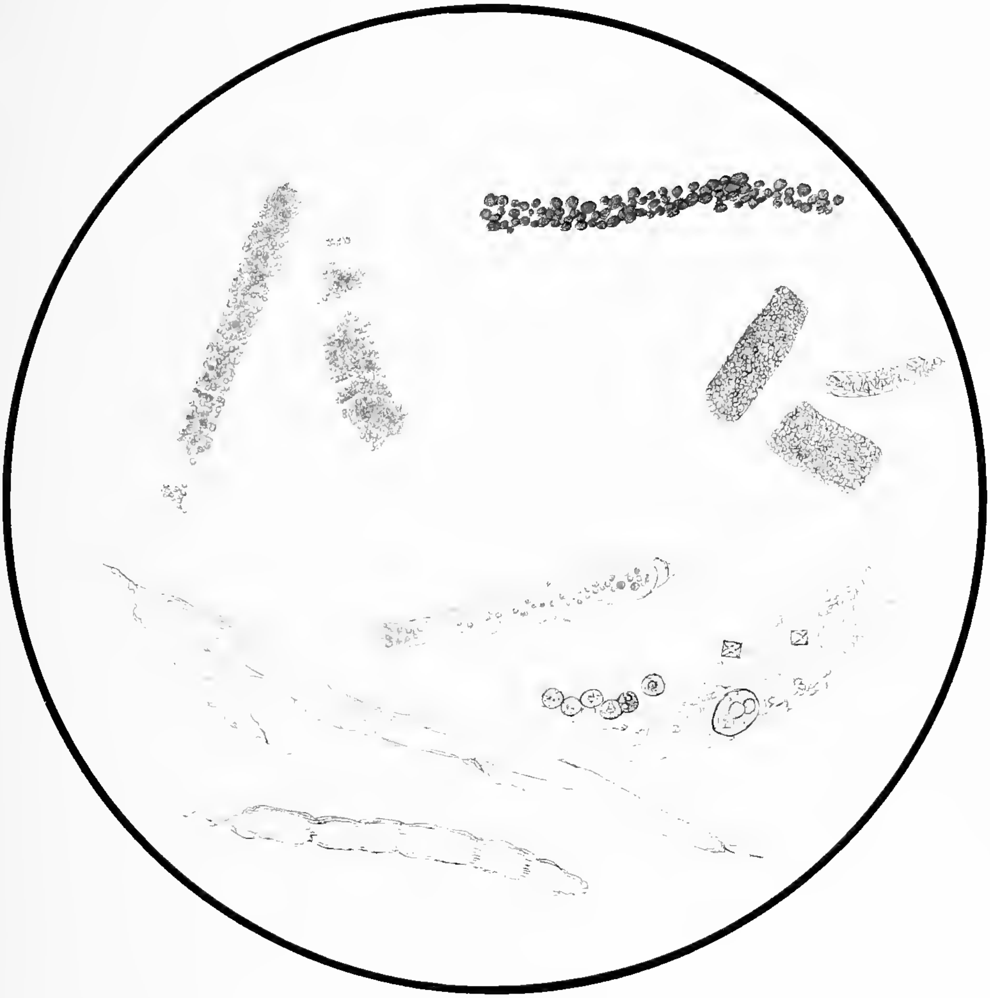
At A are two aggregations of amorphous urates, not unlike some forms of granular casts.

At B is a somewhat similar collection of small spheres of ammonium urate from an alkaline urine.

The bodies at C were repeatedly found in a case of chronic cystitis. They looked like fine granular casts, but high magnification showed them to be cylindrical masses of micro-organism, held together by fibrinous material. They probably represent moulds of the glandular follicles about the bladder and prostate.

At D are fibrin coagula, with epithelial cells and granular matter adherent; they sometimes bear a close resemblance to hyaline casts; and are to be distinguished by their less regular outline, and ragged or bifurcated ends.

PLATE XXV.



FALSE CASTS.

Magnification × 216.

PLATE XXVI.

SPERMATOZOA FROM THE URINE.

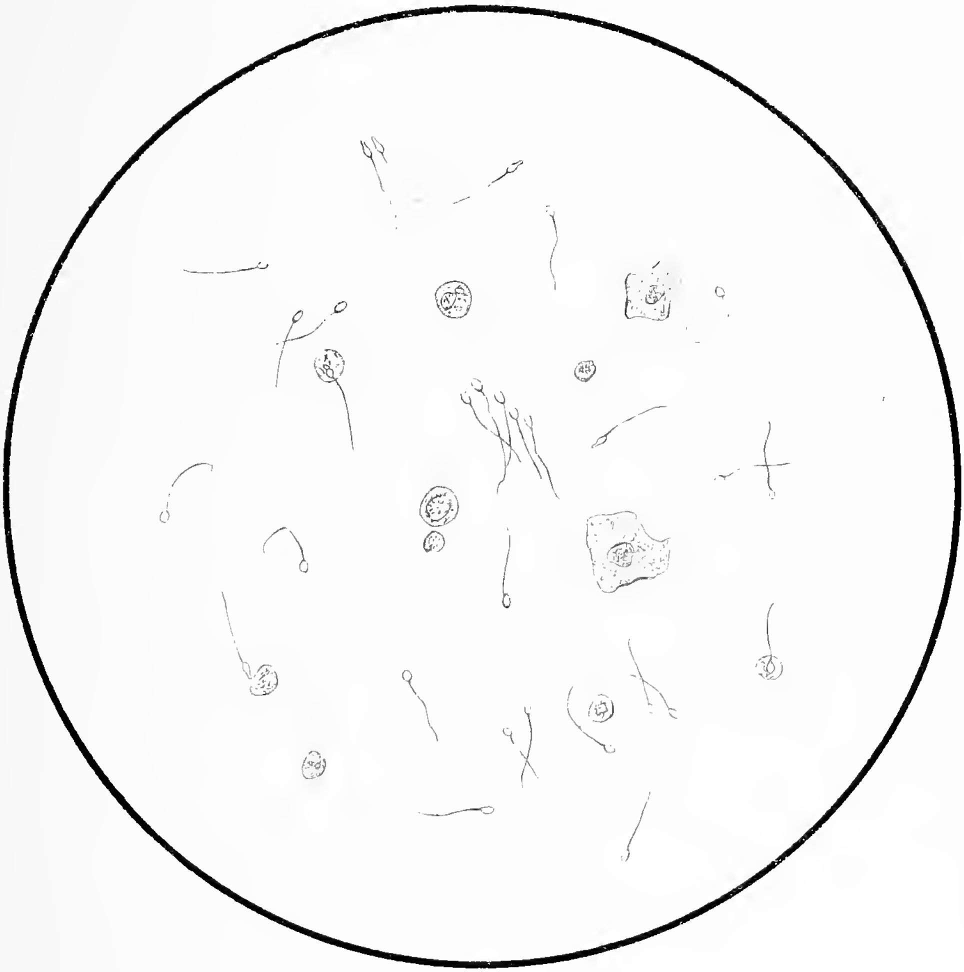
Magnification × 216.

SPERMATOZOA appear under the microscope as delicate filaments, made up of a flattened oval head and a long whip-like tail, which in the urine is quiescent. They measure $\frac{1}{600}$ of an inch long.

They may be found in the urine, male or female, after the ejaculation of semen, from whatever cause. They are highly resistent, and can be detected when alkaline decomposition is well advanced.

Their presence is suggested to the naked eye by a dense glairy cloud, with floating shreds and particles, in an otherwise clear or slightly turbid urine.

PLATE XXVI.



SPERMATOZOA FROM THE URINE.

Magnification × 216.

PLATE XXVII.

OVA AND EMBRYOS OF BILHARZIA HÆMATOBIA.

Magnification × 216.

SOME seven or eight ova of Bilharzia are depicted in the plate: they appear as oval granular bodies, with a small beak at the anterior end of the shell, and show the outline of the enclosed embryo. The ova are frequently held together in clumps by mucus, fibrin, and debris. Two free embryos are also shown; they are covered with cilia, and retain their power of active movement for a considerable time after the urine has been passed, sometimes running rapidly across the field, or elongating and contracting their bodies. Empty shells—two are represented here—from which the embryos have escaped, epithelia, and blood corpuscles are also usually found in the deposit. This plate was drawn from the urine of a young girl with Bilharzia disease.

The sediment can be collected by the centrifuge, or by getting the patient to express the last few drops of urine into a watch glass. The ova are best looked for with a half-inch power.

Bilharzia disease is common in many parts of Africa, especially in Egypt. The adult worms find access to the portal vein from the intestine, and, when sexually mature, migrate to the smaller veins of the mucous membrane of the bladder, ureter, and pelvis of the kidney. Here they shed numerous ova and embryos, giving rise to symptoms of cystitis and pyelitis, with profuse hæmaturia.

PLATE XXVII.

OVA AND EMBRYOS OF BILHARZIA HAEMATOBIA

Magnification × 216.

PLATE XXVIII.

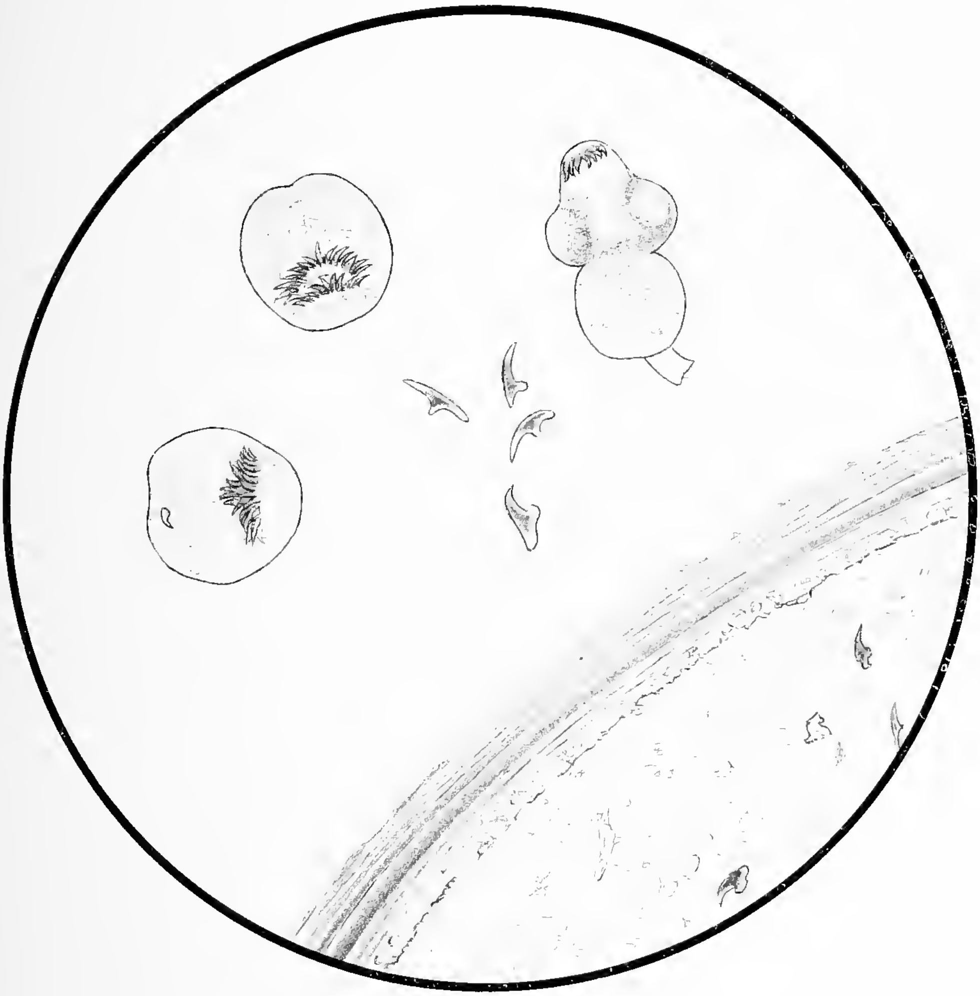
URINARY SEDIMENT IN HYDATID DISEASE OF THE KIDNEY.

WHEN the ovum of Tænia Echinococcus finds its way into the body of man, the embryo is liberated and carried by the blood stream to various organs, in which it develops into a hydatid cyst. Its seat of growth is usually the liver, more rarely the lung, still more rarely the kidney. When located in the kidney, its presence is indicated by various symptoms, such as renal tumour, attacks of pain and feverishness, and the presence in the urine of portions of the cyst wall, secondary vesicles of variable size, echinococcus heads, and hooklets.

The upper portion of the plate shows three echinococci (magnification × 216): the one to the right has the head protruded from the caudal vesicle, with its crown of hooklets at the point, and the suckers rather indistinct at each side. In the other two echinococci the head is retracted within the caudal vesicle, as one usually finds it, the double row of hooklets representing the situation where the head is invaginated. Between the echinococci are four loose hooklets (magnification × 400).

Below and to the right of the plate a portion of a small vesicle is drawn (magnification × 216). It shows the two layers of the cyst wall, the outer thick, tough, and laminated; the inner thin, soft, and granular. Among the granular gelatinous contents several loose hooklets are to be seen.

PLATE XXVIII.



ECHINOCOCCI, HOOKLETS, & PORTION OF HYDATID VESICLE.

PLATE XXIX.

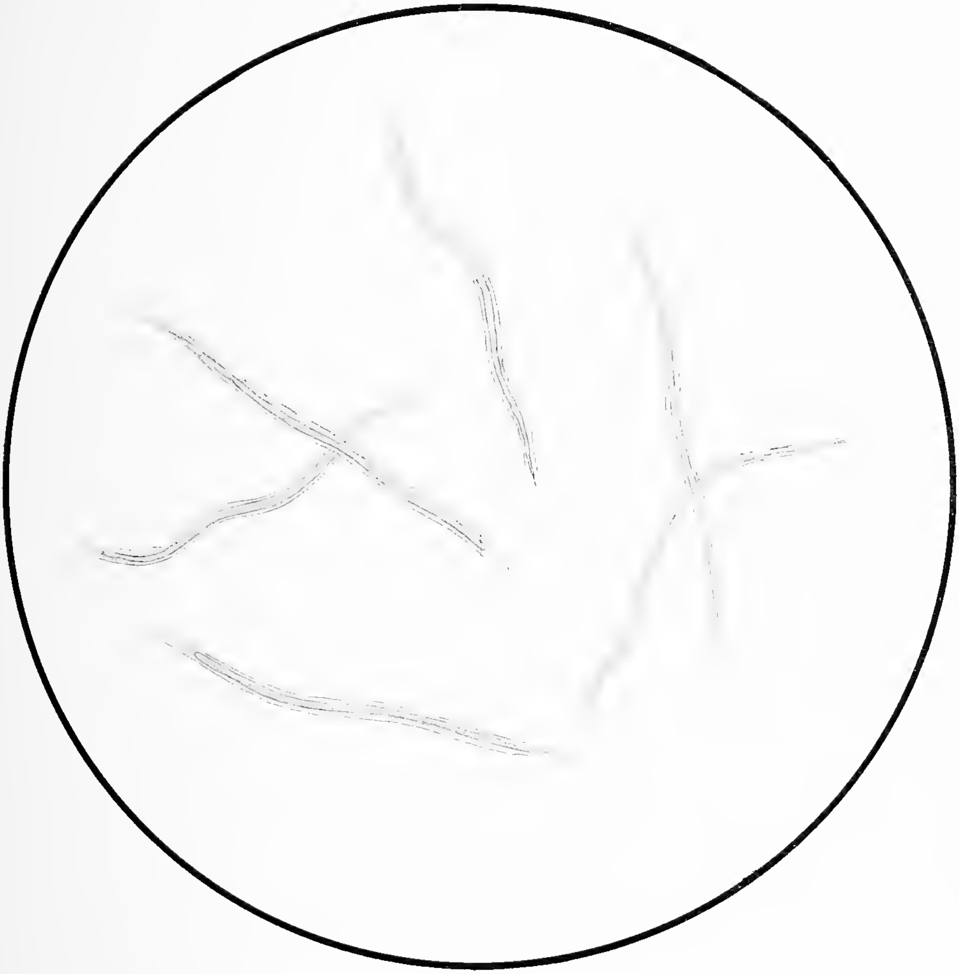
FILARIA SANGUINIS HOMINIS IN THE URINE.

Magnification × 360.

ENDEMIC CHYLURIA is due to the Filaria Sanguinis Hominis invading the blood and urinary tract. It is chiefly met with in India, China, and the West Indies. Chylous urine has a milky appearance, and on standing separates into three layers—an upper cream-like layer, a lower of reddish-brown colour; and a middle, by far the most bulky, of salmon-coloured fluid, in which the contracting clot of fibrin forms. To find filaria tease out a portion of the clot, and search with a half-inch objective, or, better still, break up the clot as soon as formed and search the sediment. The filaria will be found entangled in strings of fibrin, perhaps feebly moving. They show on high magnification a delicate hyaline envelope, in which the somewhat opaque and granular body of the parasite lies encased. They measure $\frac{1}{75}$ of an inch in length, and are about the diameter of a red-blood corpuscle.

Chylous urine contains lymph-corpuscles, blood-like corpuscles, fibrin, and much fatty matter.

PLATE XXIX.



FILARIA SANGUINIS HOMINIS IN THE URINE.

Magnification × 360.

PLATE XXX.

RARE ANIMAL PARASITES FOUND IN URINE.

EUSTRONGYLUS GIGAS. After Balbiani (*Journal de l'Anatomie et de la Physiol.*, 1870, p. 180).

Fig. A. Ovum. Magnification × 400. Fig. B. Embryo. Magnification × 250.

THE adult worms are cylindrical and of a reddish colour, the male measuring 1 foot in length and the female 3 feet. When they lodge in the kidney they destroy the glandular substance, ultimately reducing it to the condition of a sanguino-purulent cyst. The ovum and embryo are found in the urine.

The ovum is elliptical in shape. On the surface of the brown chitinous shell are numerous little clear areas surrounded by a border, being the orifices of small canals which penetrate the shell. Measurements, ·068 mm. long by ·042 mm. broad.

The embryo has pointed head and simple mouth, is circular in section and measures ·24 mm. long by ·014 mm. broad.

RHABDITIS GENITATIS. After Scheiber (*Virchow's Archiv*, 1880, p. 161).

Fig. 1. Female, from ·9 to 1·32 mm. in length. Magnification × 80.
,, 2. Embryo, about ·21 mm. in length. Magnification × 80.
,, 3. Ovum, ·06 mm. long by ·03 mm. broad. Magnification × 250.
,, 4. Sexless worm, ·54 mm. in length. Magnification × 120.

Scheiber found this worm in the urine of a female, the subject of pneumonia and pyelitis. The urine was acid, contained much pus and epithelia, innumerable living and dead parasites and ova. He concluded they came from the vagina.

Baginsky (*Deutsch med. Wochen.*, 1888, p. 604), and others have reported cases with this parasite in the urine. Peiper and Westphal (*Centralb. f. klin. Med.*, 1888, p. 145) report the case of a boy with hæmaturia, whose urine showed many of the parasites, no ova or parasites being found in the stool or about genitals.

INFUSORIA.

Fig. *α*. Trichomonas found by Dock (*Amer. Journ. Med. Scien.*, 1896, p. 1) in the urine of a man suffering from attacks of hæmaturia, with difficult and painful micturition. The organism was 15 to 22 mm. in length, and 10 to 15 mm. in width, usually elliptical in shape, substance granular and finely vacuolated. At anterior end were 3 or 4 flagella, the posterior end being prolonged into a tail-like process. Marchland and Miura have described similar urinary parasites under the name of Trichomonas Vaginalis.

Fig. *β*. The Trichomonas Vaginalis found by Donné (*Kuchenmeister's Manual of Parasites*) in the urine. He believed them to find their way into the bladder, both of male and female subjects, from the vagina.

Various infusoria, such as Bodo Urinarius and Cercomonas Urinarius described by Hassall, appear in the urine when kept standing.

Bälz (*Berl., klin. Woch.*, 1883, p. 237) found an amœba in the bladder and vagina of a Japanese woman suffering from phthisis. The urine contained also blood, pus, and necrotic debris.

Various other urinary parasites have been described, such as the Dactyleus Aculatius of Curling (*Trans. Roy. Med. Chir. Society*, 1839), the Spiroptera Hominis of Lawrence (*Trans. Roy. Med. Chir. Soc.*, 1809), but their identity is doubtful, and they seem to have been introduced by catheters.

PLATE XXX.

RARE ANIMAL PARASITES FOUND IN THE URINE.

PLATE XXXI.

THE TUBERCLE BACILLUS IN URINARY SEDIMENT.

STAINED BY THE ZIEHL-NEELSEN METHOD.

Magnification × 720.

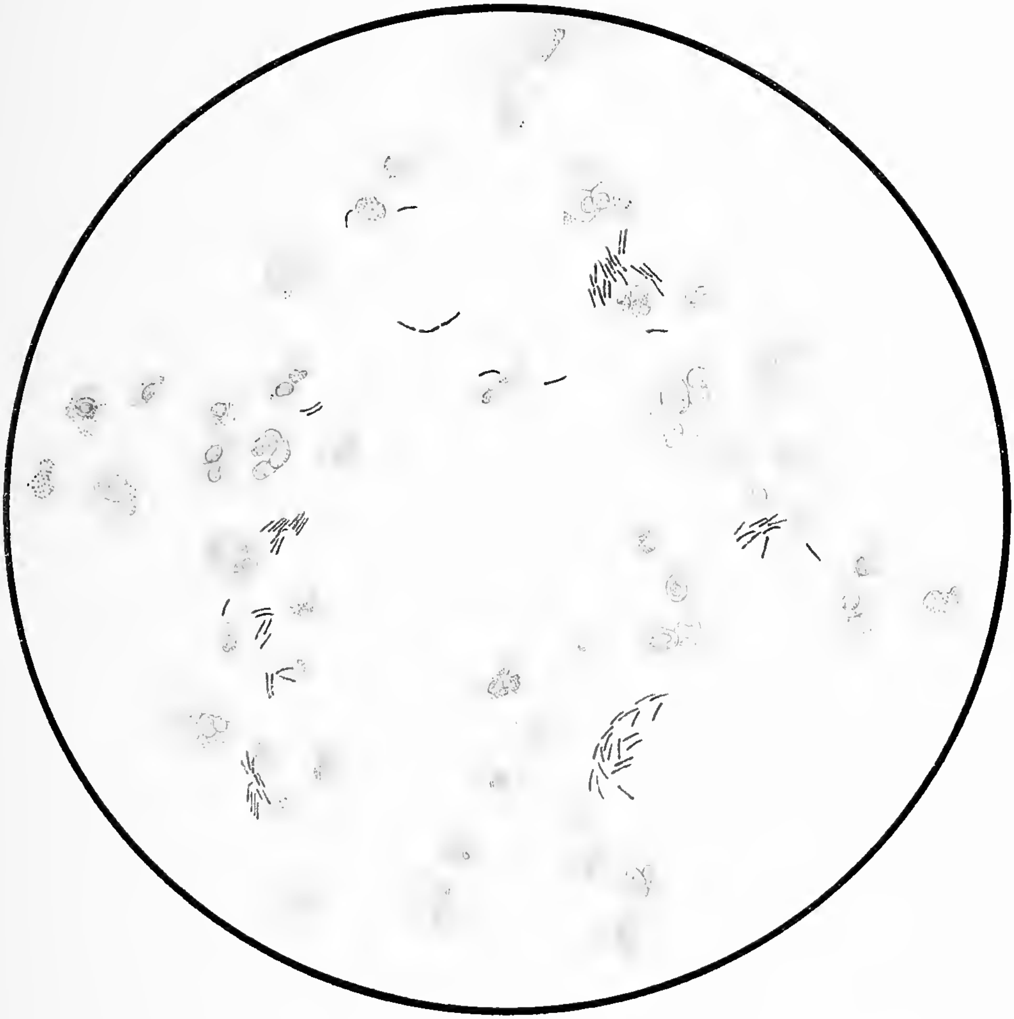
THE tubercle bacilli stain bright red in distinction to the blue colour of the pus-cells and other structures. They tend to arrange themselves in close clusters, often lying side by side. Curved or S shaped groups are extremely typical.

The bacillus can be detected in many cases of tubercular disease of the urinary system. The urine is generally acid, and contains pus in variable quantity; or it may show fragments of caseous material like crumbs of bread or cheese, with or without the presence of blood, pus, and albumin.

The centrifugalised deposit is spread on a cover glass, or one of the caseous particles compressed, and stained by the Ziehl-Neelsen method—first in a warmed solution of carbol-fuchsin, decolourising in 25 per cent. sulphuric acid, washing in absolute alcohol, and counter-staining in methylene-blue. When this fails, Biedert's method sometimes leads to their detection. Take 15 cc. of the deposit at the bottom of the urine-glass, mix with about 25 cc. water and four to eight drops of caustic soda. The mixture, after being well shaken, is boiled, and diluted with twice its own amount of water, and allowed to stand in a conical urine-glass for a couple of days, or put in centrifuge. The sediment is then stained as before.

Inability to find the bacillus does not necessarily mean absence of urinary tuberculosis.

PLATE XXXI.



THE TUBERCLE BACILLUS IN URINARY SEDIMENT.

STAINED BY ZIEHL-NEELSEN METHOD.

Magnification × 720.

PLATE XXXII.

THE SMEGMA BACILLUS.

STAINED BY THE ZIEHL-NEELSEN METHOD.

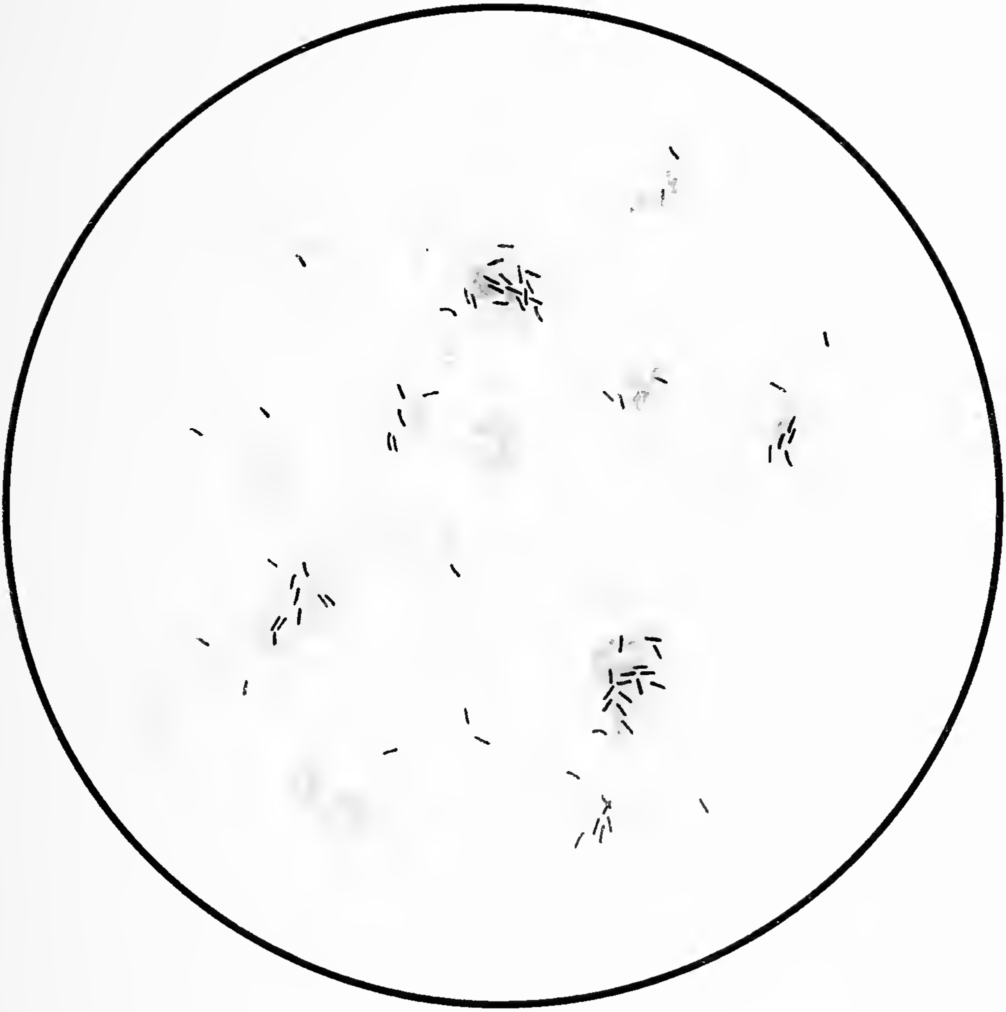
Magnification × 720.

THE smegma bacillus is common in the urine, especially of females, and is so like the tubercle bacillus in staining reactions and other respects, that its presence has on more than one occasion led to the diagnosis of renal tuberculosis, the error being detected only after removal of the kidney by operation.

In carbol-fuchsin solution it stains red, and is not decolourised by 25 per cent. sulphuric acid. Subsequent washing in absolute alcohol removes a good deal of the red tint, the bacillus appearing rather of a purple colour, different from the bright red of the tubercle bacillus. Moreover, the smegma bacillus is usually found adherent to the larger epithelial squames—*e.g.*, those of the vulva. It is never arranged in curved groups, nor do the bacilli tend to lie so closely aggregated, although, as a rule, they are much more plentiful in the specimen. It does not show a beaded appearance, such as tubercle bacillus sometimes does on high magnification.

In all doubtful cases, especially before operation, the urine should be drawn off by the catheter, the absence of the smegma bacillus thus being ensured.

PLATE XXXII.



THE SMEGMA BACILLUS.

STAINED BY ZIEHL-NEELSEN METHOD.

Magnification × 720.

PLATE XXXIII.

THE GONOCOCCUS OF NEISSER IN PUS.

STAINED IN EOSINE AND METHYLENE-BLUE.

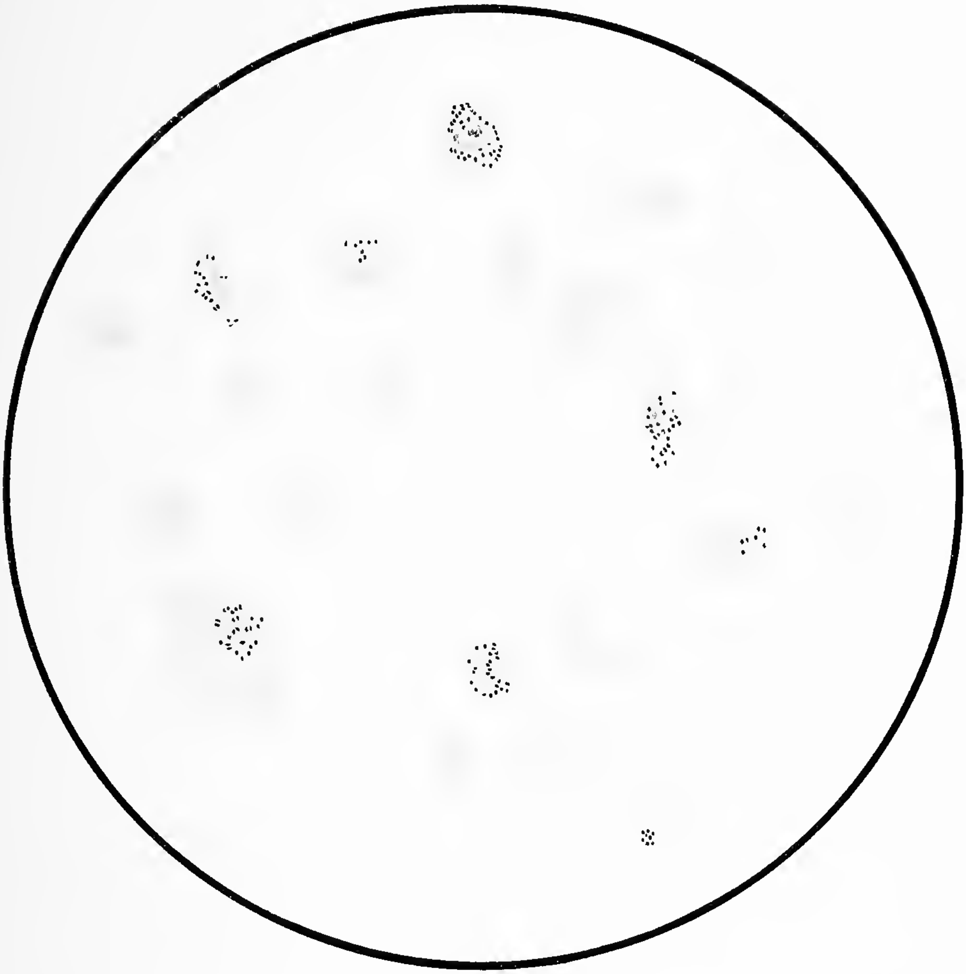
Magnification × 720.

GONOCOCCI are minute oval, coffee-bean-shaped organisms; they tend to be distributed in pairs—diplococci—and to lie inside the pus-cells, in some cases dotted over the entire substance of the cell: such an arrangement is absolutely typical of this organism.

Their detection is often of much importance. Spread a thin film of the suspected discharge on a cover glass, pass through the spirit-lamp flame to dry, and float for two or three minutes on the surface of a little methylene-blue or fuchsin solution in a watch glass; wash off excess of stain, dry thoroughly, and mount in canada-balsam. Unless the cocci are distributed within the pus-cells as shown, it is not safe to conclude they are gonococci. By Gram's method they lose the purple colour of the aniline-gentian violet after using Gram's iodine solution, and can thus be distinguished from other similar pus-forming cocci.

The present specimen was first stained in weak eosin, colouring the ground substance of the cells pink, and then in methylene-blue, colouring the nuclei and organisms. The polynucleated character of the pus-cells and the distribution of the cocci are thus well brought out. The organisms nearly always appear under the microscope as rounded dots, arranged in pairs or in groups, as shown in the plate; it is very seldom that a coffee-bean shape can be made out.

PLATE XXXIII.



THE GONOCOCCUS OF NEISSER IN PUS.

STAINED BY EOSINE AND METHYLENE-BLUE.

Magnification × 720.

PLATE XXXIV.

A PSEUDO-GONOCOCCUS IN PUS.

STAINED BY GRAM'S METHOD.

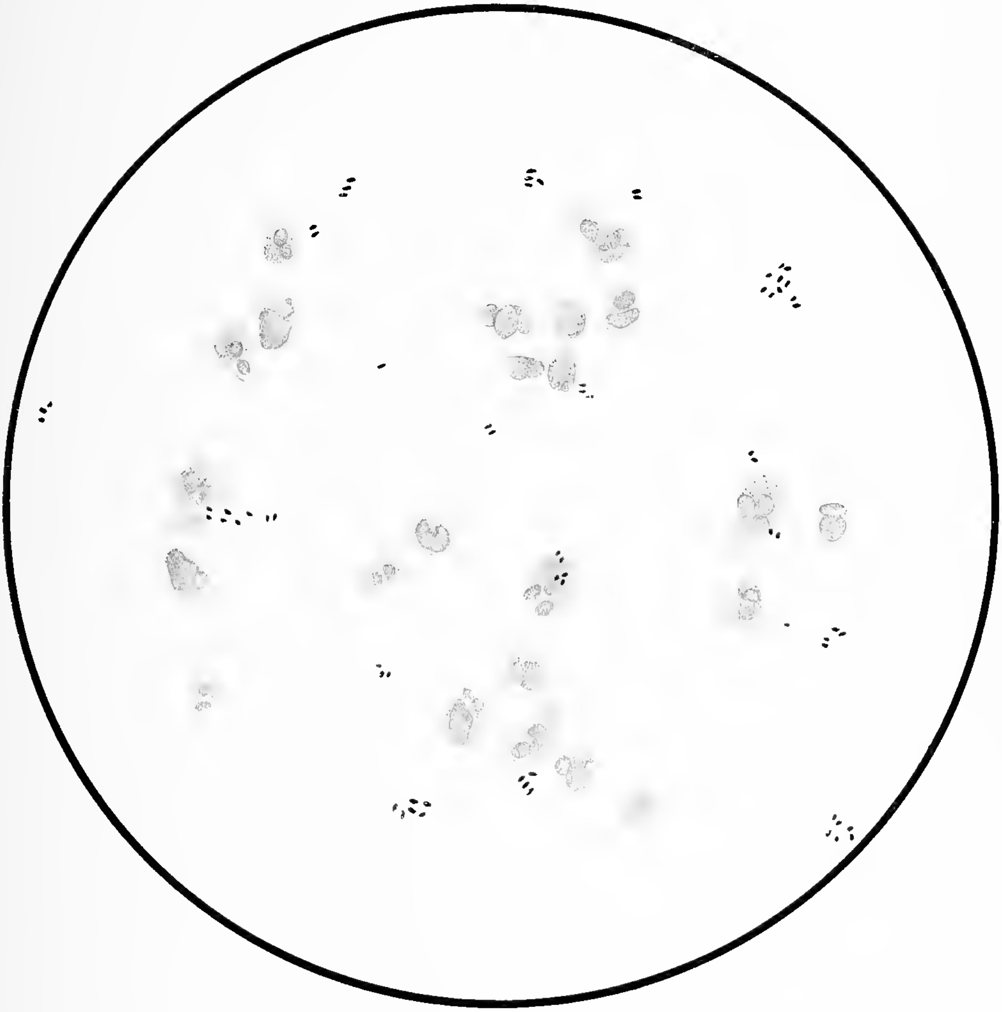
Magnification × 720.

VARIOUS diplococci bear a close resemblance to the gonococcus of Neisser. This specimen was prepared from an acute urethritis occurring in a young man, who had apparently not run the risk of infection. By simple staining in methylene-blue or fuchsin, such an organism would be difficult to distinguish from the gonococcus.

By using Gram's method—aniline-gentian violet, Gram's iodine solution, and counterstaining in weak fuchsin—it will be noticed that the organism has *not* lost the purple colour of the aniline-gentian violet in Gram's iodine solution, as the gonococcus would have done. Although it tends to be arranged like a diplococcus, very few of the cocci are in contact with or in the substance of the pus and exfoliated epithelial cells. They are also somewhat larger than gonococci.

Intra-cellular diplococci, similar in appearance and in staining reactions to the gonococcus of Neisser, have been found in cases of spinal and basal meningitis; they can, however, be distinguished by their growth on artificial media.

PLATE XXXIV.



A PSEUDO-GONOCOCCUS IN PUS.

STAINED BY GRAM'S METHOD.

Magnification × 720.

PLATE XXXV.

A SEDIMENT OF ACUTE BRIGHT'S DISEASE.

Magnification × 216.

THIS sediment was collected from a case of acute tubal nephritis, during the second week of illness. The plate shows hyaline, fatty, and blood casts, a few red blood-corpuscles, and many epithelial cells having the characters of tubal epithelium.

PLATE XXXV.

THE SEDIMENT OF ACUTE BRIGHT'S DISEASE.

Magnification × 216.

PLATE XXXVI.

THE SEDIMENT OF GOUTY KIDNEY (GRANULAR NEPHRITIS).

Magnification × 216.

THE diagnosis of gout can in doubtful cases often be made from the *microscopic* examination of the urine. The association of gout and granular kidney is very close, and indeed some writers maintain that an organic kidney affection is a necessary adjunct in the causation of gout. Even when no trace of albumin can be found in the urine, the centrifugalised sediment may show day after day finely granular and hyaline casts, which in a case of arthritis would be strongly indicative of gout. The accompanying plate was drawn from the sediment of a gouty subject affected with early granular nephritis, and shows finely granular and hyaline casts, uric acid crystals, amorphous urates, and one or two epithelial cells and leucocytes.

PLATE XXXVI.

THE SEDIMENT OF GOUTY KIDNEY (GRANULAR NEPHRITIS).

Magnification × 216.

PLATE XXXVII.

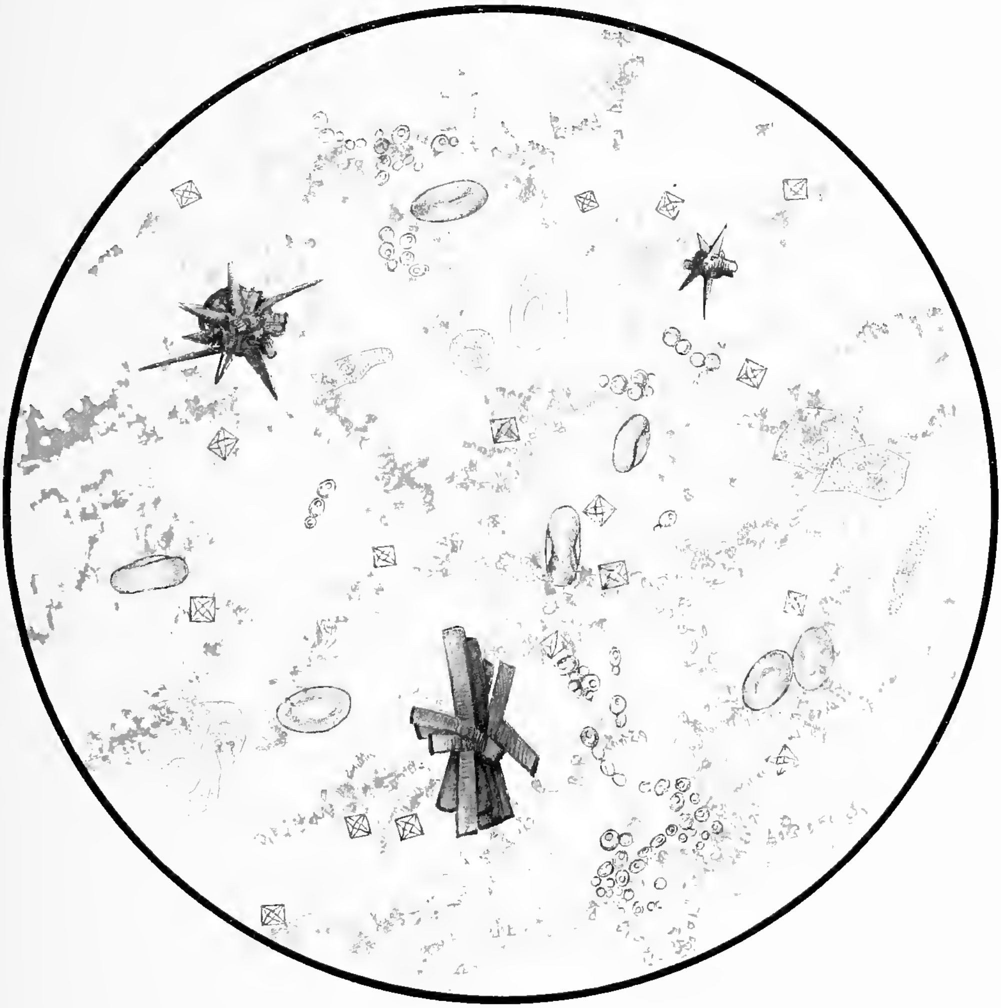
DEPOSIT FROM A CASE OF DIABETES.

Magnification × 300.

THIS plate was drawn from the urinary sediment of a female affected with glycosuria. Though this disease possesses no characteristic deposit, the drawing represents many of the structures commonly found in diabetic urine.

The most interesting feature is the presence of many oxalate of lime crystals, most of them as the biconcave biscuit-shaped colloidal form, which is not unfrequently found in urine loaded with sugar. Several groups and chains of yeast fungi are visible; these are derived from the atmosphere, and readily grow in diabetic urine. When a vulvar eruption exists, many epithelial cells are common. The deposit also shows uric acid crystals and amorphous urates.

PLATE XXXVII.



DEPOSIT FROM A CASE OF DIABETES.

Magnification × 300.

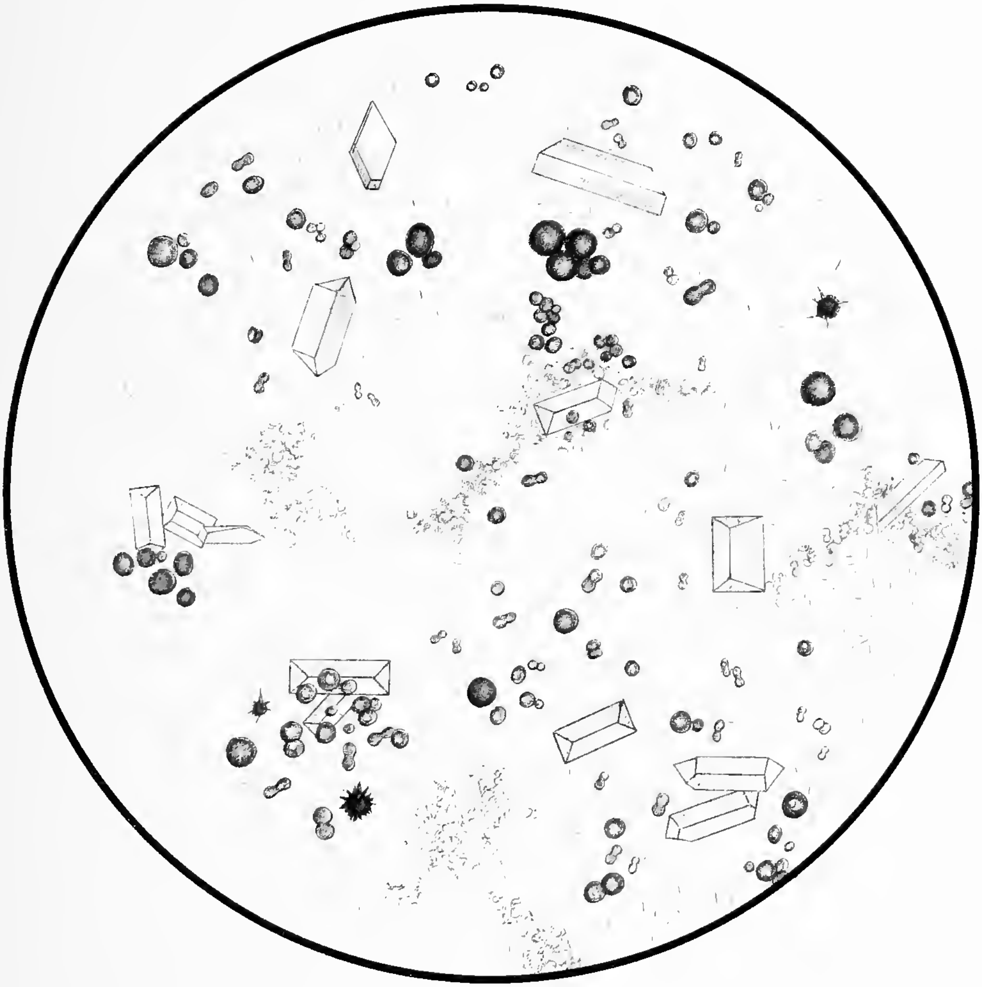
PLATE XXXVIII.

THE SEDIMENT OF AMMONIACAL DECOMPOSITION.

Magnification × 216.

THIS sediment shows crystals of ammonio-magnesic phosphate, spheres and dumb-bells of ammonium urate, several clumps of amorphous phosphate of lime, and innumerable bacteria. It is found in certain forms of cystitis, pyelitis, and pyelonephritis, or when normal urine is kept exposed to the air for a length of time, and depends upon the fermentative action of those bacteria, which have the power of decomposing urea, whereby the urine is rendered ammoniacal.

PLATE XXXVIII.



THE SEDIMENT OF AMMONIACAL DECOMPOSITION.

Magnification × 216.

PLATE XXXIX.

DEPOSIT FROM A CASE OF CYSTITIS (ACID).

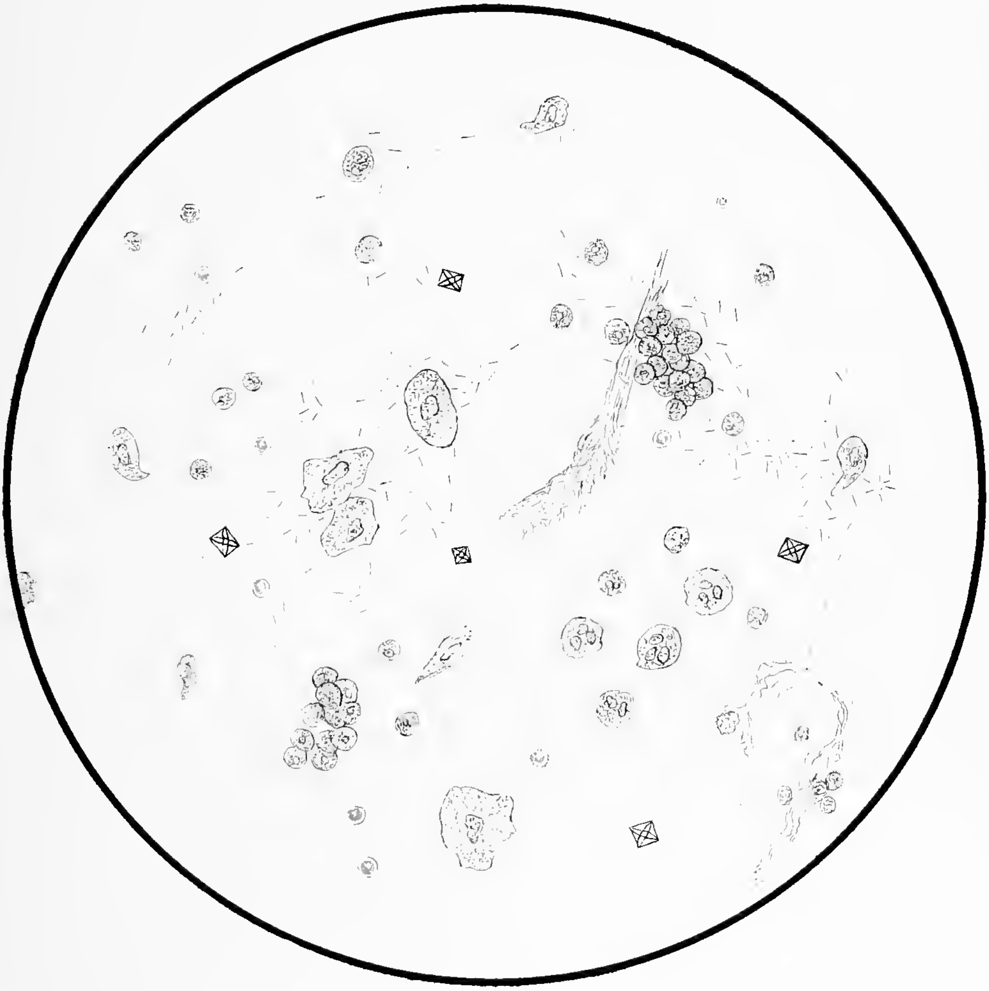
Magnification × 216.

CYSTITIS and Pyelitis are frequently caused by bacteria which do not possess the quality of decomposing urea. The urine in such cases, therefore, retains its acid reaction, and may remain acid after standing exposed to the air for several weeks.

The plate was drawn from a case of chronic cystitis. The urine when passed was acid, slightly turbid, and contained a trace of albumin; the collected sediment showed pus-cells, isolated and in groups, many epithelial cells from the bladder and urethra, shreds of fibrin, octahedra of oxalate of lime, a few red-blood corpuscles, and actively moving bacteria.

A similar deposit is found in acid pyelitis, with the addition in some cases of several long-tailed, comma-shaped, epithelial cells, suggestive of the pelvis of the kidney.

PLATE XXXIX.



DEPOSIT FROM A CASE OF CYSTITIS (ACID).

Magnification × 216.

PLATE XL.

EXTRANEOUS MATTER FOUND IN THE URINE.

Magnification × 216.

Extraneous matter finds its way into the urine from the atmosphere, from the clothing, and from the towelling of urine vessels.

Cotton fibres can be recognised by their twisted form and flat appearance; linen fibres are more cylindrical, and have joints like a bamboo; blanket fibres are broader, and show a segmented surface. Human hair is diffusely striated and pigmented, and usually shows a medulla.

Starch grains, especially those of the pea and wheat, may give rise to confusion. They stain blue with solutions of free iodine, and show a dark cross with polarised light.

Air-bubbles are recognised by their strong refraction, broad dark border, and clear centre. Oil globules have a less-defined outline, and are usually of a yellow colour.

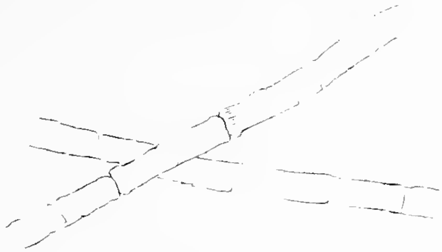
Yeast cells and spores of fungi readily gain access to the urine. They have usually an oval outline, and tend to be arranged in chains or bunches, and to show buds and a nucleus. The spores grow to form filaments, ultimately appearing as a thick net-work, which constitutes on the surface of the specimen the familiar patches of mould.

Many other extraneous substances are to be found in the urine, and not infrequently are added for the purpose of deception.

I have refrained from including any of the structures that may gain access from the intestine, either by fistulous communication or otherwise, as they would open up a sphere too wide to be dealt with here.

PLATE XL.

Cotton Fibres.



Linen Fibres.

Wool Fibres from Blanket.

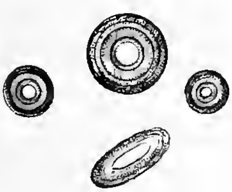
Human Hair.

Rice Starch.

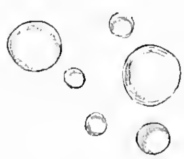
Pea Starch.

Wheat Starch.

Potato Starch

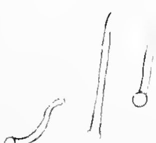


Air Bubbles.

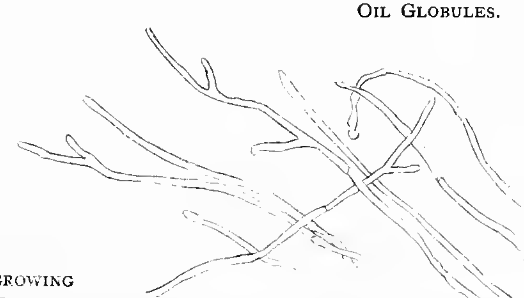


Oil Globules.

Yeast Cells and Spores of Moulds.



Mould Filaments growing from Spores



Mould Filaments.

INDEX

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