flammation of the *sheath* of a nerve, or disease of the *spinal axis*, gives rise to pain referred by the patient to the *termination* of the nerve.

Subjective **hyperæsthesia,** or perversion of sensibility or psychical impressibility, may be, in its causation (as regards the nervous apparatus), either **functional**[1] or **organic**; and the difference between these is often practically important.

3. **Muscular Paralysis**; or that condition in which a central volition (or an excitation equivalent to it) fails to produce its normal effect of muscular contraction. Of this defect, also, the pathological origin may be, as to its seat, either **peripheral, intermediate, subcentral** (in the corpora striata or cerebellum), or **central** (in the convolutions of the cerebrum). Muscular as well as sensational paralysis, dependent on an affection of the brain, occurs on the *opposite side* to that of the encephalic lesion. Scarcely ever is palsy confined *exclusively* to sensation or to voluntary motion—although the *proportion* of impairment of the two functions may vary considerably in particular cases. Both kinds are occasionally *reflex* (Brown-Séquard).

4. Involuntary contraction of voluntary muscles, or **convulsion.** Only very *local*, and usually *transitory* spasmodic affections are (unless reflex) *peripheral* in their origin. Usually, convulsive affections are accounted for by functional excitement of the (spinal) **motor centres**; the causation of which is made up of three elements, in variable proportion, viz.: *a*, morbid **irritability** of the **spinal** *excito-motor apparatus itself; b*, imperfect *control* over the *subordinate nervous centres* by the **brain,** from an abnormal condition of the latter; *c*, the disturbing influence of a **peripheral irritant**—as, tension of the gums in teething, worms in the bowels, undigested food in the stomach, etc.

The three forms of spasmodic disturbance to which the muscles are liable under a morbid alteration of innervation, viz., the **tonic, choreic,** and **clonic,** are illustrated respectively in **tetanus, chorea,** and **epilepsy.**

5. **Excito-secretory** action (Longet, Campbell) becomes morbid under conditions like those which produce convulsion; for example, the diarrhœa of infants, so common at the time of dentition.

A subject of great interest, almost neglected until within the last dozen years, is that of the effects of various agencies, *through* the **nerve-centres,** upon the **bloodvessels.** But while the *vaso-motor nerves* are now recognized, and their special relation to the *ganglionic* or sympathetic system is beginning to be appreciated, much confusion on this subject still pervades medical literature.

A further important pathological subdivision exists as to the method of **origination** of those functional disturbances of the nervous system to which we have been alluding.

The source of any of the above forms of nervous disorder, hyperæsthesia, anæsthesia, muscular paralysis, or convulsion, may be (when not purely local) either—

[1] *Functional* nervous disorder results generally (Todd) from an abnormal state of the *blood*. See a valuable lecture on Hyperæsthesia, by Dr. C. Handfield Jones; Brit. Med. Journ., Sept. 30, 1871.

1. Central **organic** disease;
2. Blood-perversion, or defective **nutrition;**
3. Purely **sympathetic** disturbance.

It is far from easy, in many cases, to mark the diagnosis between these different modes of causation of nervous symptoms; but, when the decision has been made, in any instance, the *prognosis* is *most* favorable in the *last* case; less so in the *second;* and most unfavorable in the *first, i. e.*, when the symptoms have their origin in an actual organic lesion of an important nerve-centre.

Other subjects (hemorrhage, dropsy, etc.) which might be considered as belonging to general pathology, will be taken up in Part II. of this book.

MODES OF DEATH.

Death may occur—

1st. By **asthenia:** the dynamic force of the system being exhausted or destroyed, so that the heart ceases to beat; as in death from old age, lightning-stroke, poisoning by prussic acid, etc. **Syncope** (fainting) simulates or threatens this.

2d. By **anæmia:** the blood being rendered insufficient for life; as from hemorrhage after labor, surgical injuries, bursting of an aneurism, etc.

3d. By **apnœa,** or **asphyxia;** that is, arrest of respiration, either from disease of the lungs, obstruction of the air-passages, deficiency or impurity of the air.

4th. By **coma;** the brain and medulla being made incapable of sustaining innervation; as in apoplexy, opium poisoning, etc.

Sudden death may occur from

Apoplexy;

Valvular heart-disease (especially mitral);

Rupture of the heart (or syncope) in fatty degeneration;

Bursting of an aneurism, or an abscess, within the thorax or abdomen;

Suffocation;

Violent mental shock or alarm, producing fatal syncope.

SECTION II.

SEMEIOLOGY.

I. RATIONAL SYMPTOMATOLOGY.
II. PHYSICAL DIAGNOSIS.

Rational symptoms and **physical signs** are distinguished (somewhat arbitrarily) thus: a rational symptom is a sign of disease which is obvious to the patient himself or to the practitioner without close inspection. A physical sign is one determined by examination into the properties and material conditions of the organs of the body; as by palpation, ausculation, percussion, etc. Symptoms guide us, generally, by *physiological inference;* physical signs, by *anatomical necessity.*

Symptoms and physical signs together contribute to **diagnosis**; *i. e.*, the knowledge of the character of the morbid process or state in given cases; the answer to the question "what is the matter?"

Prognosis is the anticipation of the *progress* and *results* or *terminations* of disease. The essential elements of prognosis are, a knowledge of the cause or causes of disease present; of the *condition of the organs;* and of the *general* vital state, or *degree of vital force* of the system. Prognosis depends, therefore, upon diagnosis; but is governed, in a majority of cases, chiefly by those rational symptoms which indicate the organic energy of the patient, and the kind and rate of change that his system is undergoing.

SYMPTOMATOLOGY.

Symptoms, or **rational signs**, are—

Local, or constitutional;
Idiopathic (primary), or secondary;
Premonitory (prodromata);
Critical;
Pathognomonic (characteristic);

We examine the symptoms of disease as connected with the digestive, circulatory, respiratory, tegumentary, secretory, motor, sensory, and psychical apparatus.

SYMPTOMS CONNECTED WITH THE DIGESTIVE SYSTEM.

The **tongue** may be natural, pale, cold, red, furred, brown, black, cracked, or fissured.

It is *pale*, in anæmia.

Cold, in *collapse*, as of cholera, etc.

Red, in scarlatina, stomatitis, sometimes in gastritis.

Furred, in indigestion, gastro-hepatic catarrh, fever, etc.

Brown or *black*, *cracked* or *fissured*, in low fevers: as typhus or typhoid.

Protruded with difficulty, in low fevers, and in apoplexy; *to one side*, in paralysis.

The *manner of cleaning* of the tongue during convalescence should also be noticed, as affording prognostic indications.

The **teeth** are *covered with sordes* in low febrile states.

They are *loosened* by severe salivation.

Their *rapid decay* shows some impairment of constitution; but this is unfortunately very common.

The **gums** are swollen, soft, and spongy, and prone to bleed, in scurvy.

A *blue line along the gums* is observed in lead poisoning.

A *red* line along their edge is sometimes noticed in phthisis.

Swelling and soreness of the gums, with tenderness of the teeth, and a coppery taste, occur in *salivation*.

Increased flow of saliva gives name to this effect of mercury on the mouth.

Deficiency and thickness or viscidity of the saliva occurs generally during fever; and often also in chronic diseases, especially of the throat and stomach.

The **taste** is morbidly

Bitter, in hepatic derangements, dyspepsia, etc.;
Sour, in gastric indigestion;
Saltish, in phthisis pulmonalis, hæmoptysis, etc.;
Putrid, in gangrene of the lungs.

Appetite is generally *deficient* (anorexia) in disease, especially of an acute character.

Excessive appetite (bulimia) is not often important; sometimes it occurs in nervous affections, in *diabetes*, and in persons having *worms* in the alimentary canal.

Perverted appetite is one of the symptoms of chlorosis, hysteria, etc.

Thirst is *excessive* in *two opposite* conditions: *high fever* and *low collapse*.

Difficulty of swallowing (*dysphagia*) may result from—

Inflammation of the fauces, tonsils, or pharynx;
Spasmodic constriction of the throat;
Stricture of the pharynx or *œsophagus;*
Obstruction by a foreign body, tumor, etc.;
Retro-pharyngeal abscess;
General debility, as in the moribund state.

Nausea and vomiting may occur from—

Indigestion: egesta.[1] partly digested food, mucus, etc.;
Colic: eg., ditto, bile, etc.;
Pregnancy: eg., mucus, food, etc.;
Gastritis: eg., abundant and altered mucus, etc.;
Hysteria: eg., gastric and biliary secretions, more or less altered;
Cholera morbus: eg., gastric and biliary secretions, diluted;
Cholera maligna: eg., copious watery fluid (rice-water);
Bilious fever: eg., altered mucus, bile, etc.;
Yellow fever: eg., (advanced stage) black vomit;
Ulcer of stomach. eg., mucus, lymph, blood;
Cancer of stomach: eg., ditto, with cancer-cells, fibres, etc.;
Disease of the brain: eg., not peculiar in character;
Bright's disease of kidney: eg., not peculiar;
Strangulated hernia: eg., stercoraceous (fecal);
Poisoning: as by tartar emetic, arsenic, etc.

Sarcinæ, or microscopic, wool-sack like vegetable parasites are occasionally found in matters vomited, in cases of disease of the stomach. Epithelial cells, starch granules, torulæ (also vegetable), and vibriones, are often discovered by the microscope.

SYMPTOMS CONNECTED WITH THE CIRCULATORY SYSTEM.

Palpitation or disturbed action of the heart may depend upon—

Pericarditis or *endocarditis;*
Hypertrophy of the heart;
Chronic valvular disease;

[1] *Egesta*, matters thrown out.

Anæmia;
Nervous irritability (nervousness);
Disorder of the brain;
Dyspepsia.

The pulse should be examined when the mind and body of the patient are as tranquil as possible. It is most rapid in the standing posture, less so when sitting, slowest in the recumbent position. Dr. Guy asserts it to be most rapid in the morning. It is increased in force and frequency by exercise, food, and emotional excitement. The pulse of the female is slightly more rapid, as a rule, than that of the male sex. It diminishes in rapidity from infancy to old age; but in very aged people it again becomes somewhat accelerated.

In obscure cases we should examine the pulsation of other arteries beside those at the wrist; and should especially observe the character of the *impulse of the* **heart.**

In adults, the average number of beats in health is, for the male, 70; for the female, 75.

Infancy, 120 to 100.	Middle life, 75 to 65.
Childhood, 100 to 90.	Old age, 70 to 60.
Youth, 90 to 75.	Decrepit age, 75 to 80.

We judge by the pulse (inferentially) of the force of the **heart's** action, of the force of the **arterial** impulse, of the **excitability** of the nervous system, of the **fulness** of the bloodvessels, and of the tone and physical **condition** of the arteries.

The pulse in disease may be *natural*, or *strong*, *weak*, *firm*, *yielding*, *full*, *small*, *bounding*, *compressible*, *rapid*, *slow*, *quick*, *jerking*, *hard*, *soft*, *tense*, *gaseous*, *corded*, *wiry*, *thready*, *imperceptible*, *regular*, *irregular*, *intermittent*, *dicrotous*.

Not considering it necessary here to define each of these terms, it may be remarked that an important difference exists between a *rapid* pulse and a *quick* pulse, and between one that is merely *full* and *large* and one that is *strong*.

The pulse of **fever** is characterized by *moderate* **acceleration,** with variable increase of force in the beat.

The pulse of **inflammation** (with constitutional excitement) is not only *accelerated*, but **hard** or *tense*, and, commonly, full. Whatever may be said to the contrary, this character of the pulse is, in acute inflammations, of great consequence as one indication for treatment; although, of course, it must not be depended on *alone*.

The pulse of **nervous irritation** is usually **quick,** and **variable** in rapidity and force, under excitement or repose.

A *jerking*, abrupt pulse is associated (Stokes) with *deficiency of the aortic valve*.

The pulse of *extreme* **debility** is nearly always (as in the dying state) very **rapid** and very **small,** or "thready."

Irregularity of the pulse is occasionally congenital; sometimes it comes on with old age. It is of the least importance in young children. It may be a transient symptom, accidental, as it were, during the progress of an acute malady; or at the commencement of convalescence, as from remittent fever. It is directly related to

the nature of the disease, in certain cases of *disease of the heart*, and in *meningitis* (inflammation of the membranes of the brain) during the stage of effusion. Dr. B. W. Richardson has laid stress upon the frequency with which *mental depression* is a cause of irregularity of the heart's action.

The **dicrotous** or double pulse is observed especially during *continued fevers*, either typhous or typhoid. It is explained in some cases, at least, by a loss of muscular tone in the arteries, so that the arterial impulse is separated from that of the ventricles by a perceptible (though slight) interval.

The state of the **capillary and venous circulation** often affords signs of disease. Torpor of the circulation is marked by slowness in the return of the blood after it has been displaced by pressure; for instance, upon the cheek or back of the hand. The *veins* of the hand or arm may be similarly examined with advantage; as in *cholera*, *pernicious intermittent*, *low continued fever*, etc. The venous circulation is affected not unfrequently in *heart-disease: e. g.*, pulsation of the jugular veins, from valvular disease involving the right side of the heart; *cyanosis*, or *blueness*, from imperfect separation of the arterial from the venous blood, etc.

Pulsation of the veins does not, however (notwithstanding the dictum of authorities), *always* depend upon disorder of the heart. The author has seen three cases in which jugular pulsation was evidently the result of *local irritation*, exaggerating the muscular activity resident in the organic muscle-fibres of the vein.

The **blood** itself is perhaps the most important of all subjects of inquiry in connection with disease. Little, however, as yet, is known of its morbid changes. The principal facts are, that—

In *anæmia*, there is a deficiency of the red corpuscles;
In *plethora*, an excess of red corpuscles;
In *leucocythæmia*, an excess of the colorless corpuscles;
In *inflammation*, and in *chlorosis*, excess of fibrin;
In *anticipation* of *suppuration*, excess of colorless corpuscles;
In *gout*, excess of uric acid;
In *rheumatism* (probably), excess of lactic acid;
In (malarial) *melanæmia*, excess of free pigment;
In *jaundice*, excess of biliary matter;
In *Bright's disease*, excess of urea, etc. (uræmia);
In *diabetes*, excess of sugar;
In *malignant cholera*, deficiency of water and salts.

These peculiarities require minute inspection, with the aid of the microscope or of chemical reagents. To the eye, differences sometimes exist which may be instructive; *e. g.*, as to the bright red or very dark color of the blood; as to the magnitude, form, and firmness of the clot, and the rapidity of coagulation, etc.

In cases of lingering prostration, clots may form in the heart or large arteries before death. After very rapid malignant diseases, the blood is sometimes found uncoagulable.

Hemorrhage from different parts of the body is often important as a symptom, but requires to be interpreted with care. Its consequence varies much with its *quantity*, and the *source* of the blood thrown out.

Thus, in **epistaxis,** or bleeding at the nose, the flow may result from—

Mechanical injury;
Congestion of the Schneiderian membrane;
Congestion of the brain;
Typhoid fever;
Hemorrhagic diathesis;
Suppressed menstruation.

This variety of hemorrhage is, however, *most* frequent during *childhood* and early adolescence.

In hæmoptysis, or spitting of blood, the source of the hemorrhage may be the—

Gums;
Posterior nares;
Throat (*e. g.*, ulcerations, etc.);
Bronchial mucous membrane;
Lungs;
Stomach.

In the *last* case, being *vomited* into the mouth, it is properly called **hæmatemesis.** Sometimes it requires care to determine *what is* the source of blood coming from the mouth. We must notice what are the *symptoms preceding* the hemorrhage; and the *manner of its ejection*, whether by *coughing* or *vomiting*, etc., as well as the appearance of the blood, whether mixed with food, gastric fluid, etc.

True **pulmonary hæmoptysis** may arise from—

Active congestion of the lungs;
Passive congestion, from heart disease;
Tubercular phthisis;
Hemorrhagic diathesis;
Vicarious monthly flow, in the female;
Mechanical injury, as fractured rib, etc.;
Rupture of aortic aneurism.

Hæmatemesis, or vomiting of blood, may be—

Hysterical;
Ulcerative;
Cancerous;
Vicarious; etc.

Uterine hemorrhage, other than the normal menses, may be—

Congestive;
Ulcerative;
Cancerous; as well as, in the *pregnant* female, *placental*, technically called "unavoidable hemorrhage;" that of *abortion;* or *after parturition.*

Hemorrhage from the bowels may be connected with—

Hemorrhoids, or piles;
Dysentery;
Ulceration of the bowels;
Intussusception;

Cancer of rectum, etc.;
Rupture of aneurism;
Hemorrhagic diathesis;
Typhoid or *yellow fever;*
Vicarious menstruation.

Hæmaturia, or bloody urine, may result from—

Mechanical injury of the bladder, prostate gland, or urethra;
Renal inflammation;
Calculus;
Hemorrhagic diathesis;
Passive senile congestion of the kidneys;
Scarlatina.

SYMPTOMS CONNECTED WITH THE RESPIRATORY ORGANS.

The normal, average rate of breathing in the adult, while at rest, is sixteen or eighteen respirations in the minute. In fever it is much accelerated. In *extreme narcotism* it becomes slower than natural. In some cases of *fatty degeneration of the heart* it is sighing and interrupted.

Dyspnœa, or difficulty of breathing, when great, is called **orthopnœa,** from the erect posture required by the patient. **Cervical** respiration, *i. e.*, auxiliary action of the muscles of the neck, occurs in cases of great exhaustion, or of obstruction of the respiratory function by disease.

Dyspnœa may be caused by—

Chlorine or other irrespirable gases in the air;
Morbid change of the blood, as in cholera;
Laryngeal or tracheal obstruction, as in croup, etc.;
Bronchial spasmodic constriction, as in asthma;
Bronchitis; pneumonia; pleurisy; phthisis;
Heart disease; aneurism of thoracic aorta;
Cancer within the chest; hydrothorax; ascites.

Coughing may depend upon a variety of causes, the nature of which may often be concluded upon from its character. Thus, usually,

Cough is *dry and hollow*, or *hacking*, when nervous or sympathetic;
Dry and tight, in early bronchitis;
Soft, deep, and loose, in advanced bronchitis;
Hacking, in incipient phthisis pulmonalis;
Deep and distressing, in confirmed consumption;
Short and sharp, in pneumonia;
Barking and hoarse, in early or spasmodic croup;
Whistling, in advanced membranous croup;
Paroxysmal, and whooping, in pertussis.

Expectoration is—

Mucous, in catarrh, and early bronchitis;
Purulent, in severe and protracted bronchitis;
Rusty, in the early and middle stages of pneumonia;
Bloody and muco-purulent, in phthisis;

Nummular and heavy, etc., in advanced phthisis ;[1]
Putrid, in gangrene of the lung.

The **temperature** *of the breath* is *increased* during the *febrile state.* It is *lowered*, sensibly, only in aggravated prostration ; as in the collapse of cholera. Coldness of the breath is an almost certain prognostic of dissolution.

The **odor** of the breath is rarely perfectly agreeable except in the healthy infant or child. It is very heavy at the commencement of fever ; sour during indigestion ; offensive, often, from decayed teeth ; rotton, in gangrene of the lung.

Hiccough (singultus) is produced by a spasm of the diaphragm. It may depend upon *indigestion*, *nervous disorder*, or *exhaustion.* It is serious in prognosis only when the latter is present or is anticipated.

Stertorous *respiration*, from relaxation of the *velum palati*, results from *cerebral oppression ;* the cause of which may be *apoplexy*, *fracture of the skull*, *dead drunkenness*, or *narcotism* by opium, etc.

SYMPTOMS CONNECTED WITH THE TEGUMENTARY APPARATUS.

The skin is **hot** and **dry** during the presence of **fever.**

Moisture is almost always a *favorable* sign.

The exceptions are, the profuse *colliquative* sweats of phthisis, etc., and the *cold and clammy* perspiration of extreme prostration. Coldness of the skin, or inequality of temperature, is always more or less unfavorable.

Emaciation is often an important sign. It generally occurs in severe chronic diseases, but is sometimes rapidly brought on in acute affections ; *e. g.*, diarrhœa or dysentery. The changes which occur in the adipose tissue, and in the plumpness and roundness, or flabbiness and shrunken appearance of the surface of the body, are often *extremely rapid in children.*

The **color** of the skin varies much in disease. Thus, the face is—

Pale, in anæmia, syncope, etc. ;
Flushed, in fever, congestion of brain, etc.;
Cheeks brightly flushed, in hectic fever ;
Forehead and eyes flushed, in early stage of yellow fever ;
Purple or livid, in low continued fever ;
Yellow, in jaundice, bilious fever, yellow fever ;
Sallow, in chlorosis, dyspepsia, cancer ;
Blue, in the collapse of cholera, and in cyanosis ;
Black, almost, in asphyxia.

Eruptions upon the skin are characteristic of certain diseases. Their description belongs to Special Pathology.

SYMPTOMS CONNECTED WITH THE SECRETIONS.

These must always be considered along with *other* explanatory symptoms ; and the **character** of the *discharges* should never be overlooked. Thus,

[1] Microscopic examination has discovered (Schrœder von der Kolk) portions of disintegrated *lung-tissue* in the expectoration of phthisical patients ; arched and anastomosing fibrils of pulmonary and bronchial elastic tissue, with abrupt or square fracture.

Constipation may denote—

Torpor of the muscular coat of the bowels ;
Deficient *secretion* of the liver, or intestinal glands ;
Defective *innervation*, from spinal or encephalic disease ;
Stricture of rectum or *colon ; pregnancy ; cancer ;*
Intussusception, *strangulated hernia*, etc. ;
Sympathetic disturbance from fever, etc.

Diarrhœa and **Dysentery** will be considered in another department. It may be mentioned, however, that in *dysentery* the discharges contain blood, mucus, lymph (in small quantity), and, when ulceration has occurred, pus. In *diarrhœa* they are either fecal, mucous, bilious, or serous—the last being of importance, especially in the diagnosis of *cholera.*

Symptoms Connected with Urination.

Dysuria, or difficult urination (strangury).
Ischuria, retention of urine.
Enuresis, incontinence.
Diuresis (diabetes), excessive discharge of urine.
Morbid character of the urine itself.

The average quantity of urine passed by a healthy adult in twenty-four hours, is from thirty to forty ounces—greatest in the winter.

In reaction to test-paper, the urine is normally *acid ;* reddening litmus, or restoring to turmeric its yellow after it has been made brownish-red by an alkali.

The *color* of healthy urine is that of *amber.*

The average *specific gravity* of human urine (water being 1000) is 1017-24 ; containing about twenty grains of solid matter to the ounce.

Deviation, to a certain extent, from any or all of the above standards as to quantity, reaction, color, and weight, is quite compatible with ordinary health ; but a very decided and persistent deviation is a proof of disease.

Retention of urine may be caused by—

Deficiency of contractile power in the bladder ,
Spasmodic constriction of the vesico-urethral muscular fibres ;
True *stricture of the urethra ;*
Enlargement of the prostate gland ;
Calculus in the bladder or urethra.

Percussion and palpation, as well as catheterism, are sometimes necessary to determine the fact of retention of urine.

Suppression of urine, from inaction of the kidneys, is a most serious symptom under all circumstances. If long continued, it becomes fatal by *urœmic poisoning*—coma, and often convulsions, preceding death. *Partial* suppression of urine occurs, sometimes transiently, in cholera, scarlet fever, etc.

Excessive urination is frequently present in hysterical cases—the water being pellucid, and of low specific gravity (diabetes insipidus). The influence of cold and of diuretic medicines produces a similar watery excess, mostly with little increase in the solids of the urine.

Diabetes mellitus is, however, a more important affection; in which the urine is not only excessive in quantity, but *heavy*, and *loaded with sugar*.

For the accurate estimation of the changes occurring in the urine in disease, scientific skill is requisite. To pursue *original investigations* upon the subject, considerable practical knowledge of *analytical chemistry*, and of the use of the *microscope*, is indispensable. But for the *application* of the conclusions of pathological chemists and micrologists to *diagnosis*, a much more moderate amount of skill will suffice. There is wisdom in the remark of Dr. Todd (*Clin. Lect. on Urinary Organs*, etc., p. 73), that, "while it is clearly a duty not to neglect any means of observation and investigation, it is desirable that you should be as little as possible dependent on means which are not always at hand, and which it does not fall to the lot of every eye and hand to use with equal readiness and skill."[1]

I shall state, on this principle, only the *most important* and *available* points in urinary pathology and diagnosis.

Allowance must always be made, or correction obtained, for the *variation* the urine undergoes in the course of the same day. It is divided technically into the *urina* **sanguinis**, *urina* **chyli**, and *urina* **potus**: the first being that after a night's rest, the second that after dinner, the third after a very light meal with fluid, as tea. All of these should in each case be examined and compared.[2]

The questions in regard to any given specimen of urine are (see Bowman's *Medical Chemistry*), as to its **general appearance, specific gravity, acidity** or alkalinity, the chemical or microscopical character of its **sediments**, and the **effects of reagents** upon the clear fluid.

General appearance. If *clear*, after standing a few hours, note the *color*. *Deep-colored* transparent urine, of high specific gravity, indicates excessive metamorphosis of tissue. In *jaundice*, the urine is generally very yellow, and sometimes as dark as porter.

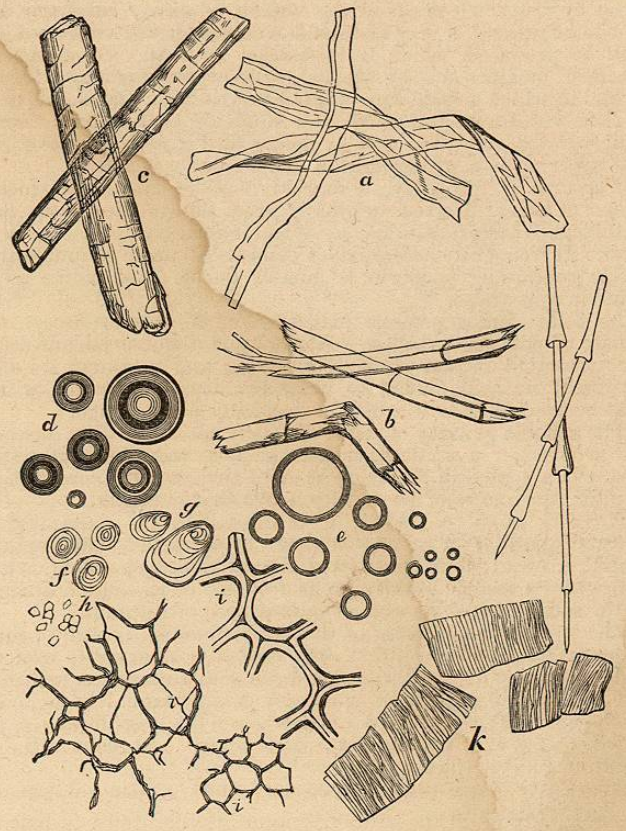
If the urine be *opaque*, it is either *white* or *dark*. *White* opaque urine contains either *mucus*, or *pus*, or undissolved *earthy salts*, or all of these together. Mucus floats more distinctly in a separate cloud than pus; purulent urine is generally opaque throughout, and of a creamy yellow color at bottom. Pus can, however, be more readily diffused by agitation than mucus. Purulent urine is oftenest *acid;* mucous urine, generally *alkaline*. Pus contains *albumen*, as shown by testing; mucus does not. Acetic acid coagulates mucus, not pus.

Dark-colored opaque urine is most frequently tinged with *blood*, giving it a pinkish or brownish hue. The latter color prevails especially in cases of passive hemorrhage from the kidney—the former in fresh hemorrhage from the bladder, or acute renal hemorrhage. Urine may also be dark from the presence of *bile* (as in jaundice), or of *purpurin*.

[1] A similar remark has been made by Virchow, in reference to "naked-eye pathology." He asserts that almost always microscopical appearances may be anticipated by a *careful* and *skilful* examination with the unaided sight.

[2] It is well also (in some cases at least) to avoid the complication of *urethral* discharges, by having the first ounce or two passed into a different vessel from that which is to receive the urine to be retained.

Fig. 16.



Foreign bodies in urine. *a*. Cotton fibres; *b*. Flax fibres; *c*. Hairs; *d*. Air-bubbles; *e*. Oil globules; *f*. Wheat starch; *g*. Potato starch; *h*. Rice starch; *i, i, i*. Vegetable tissue; *k*. Muscular fibres.

For biliary coloring matter (biliphæin, cholepyrrhin) a good **test** is (Gmelin's) the addition of nitric acid, drop by drop, to a little of the urine on a white dish. It will become pale green, violet, pink, and yellow, in succession. Or (Heller's) shake a little solution of albumen (white of egg) with the urine, and then add a slight excess of nitric acid; if bile be present, the coagulum will be dull

green or bluish. Or (Cunisset), add half its bulk of chloroform to the urine; the yellow coloring matter will be carried down.

Pettenkofer's test for bile consists in the addition (after separating albumen, if it be present in the urine, by coagulation and filtration) to the fluid, of a grain or two of white sugar, and then, drop by drop, two-thirds of the volume of strong sulphuric acid. If bile be present, a very distinct and characteristic violet-red color will be produced, which is intensified by heat.[1] Strassburg, of Bremen, modifies this by dipping a slip of filtering paper into the urine, to which a little cane sugar has first been added. The slip is then withdrawn and dried. On applying to it a drop or two of concentrated sulphuric acid by means of a glass rod, and holding the paper up to a strong light, a violet color appears.

Purpurin is, probably, a morbid modification of the *coloring matter* of urine; derived, originally, from that of the *blood*. Some pathologists believe it to be one of the indications of disease of the liver. It frequently accompanies deposits of urate of ammonium. Urine containing purpurin is pink or purple; not unlike bloody urine in appearance.

As tests—liquor potassæ makes purpurin *greenish-brown;* carbonate of potassium, *yellow*. Alcohol will dissolve purpurin from an evaporated extract of urine, receiving and retaining its color. Hydrochloric acid, added to urine containing purpurin, will, if heat be applied, give it a lilac or decidedly purple tinge.

The **specific gravity** of urine is easily ascertained by means of the *urinometer;* a small glass instrument so weighted and marked that, when floated in the urine at 60° Fahr., it will show, in thousandths, the excess of its density above that of water.

Excessive weight of the urine is caused by its containing an unusual quantity of *salts* or of *urea;* or by *sugar*. The quantity passed in twenty-four hours must always be considered in connection with its specific gravity, so as to judge of the actual quantity of the solids passed, as well as their degree of dilution.

The *heaviest* urine is that of *diabetes mellitus* (glycosuria); sometimes reaching 1060 to 1070. The *lightest* is observed in hysteria, and in Bright's disease; running down sometimes even to 1003.

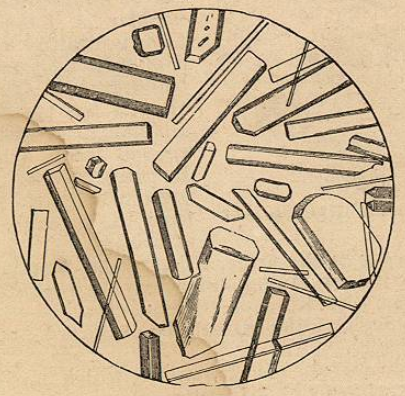
The degree of **acidity** of urine may be approximately estimated by the more or less decided redness given by it to litmus paper. If it be *alkaline*, it will make turmeric brown, and restore the blue to litmus reddened by an acid.

Alkalinity of the urine is *uncommon;* unless (Bence Jones) immediately after a meal. If it does occur at other times, it depends upon either fixed (potassa, soda) or volatile alkali (ammonia). If

[1] Practical difficulties in the use of this test have led Hoppe-Seyler to propose the following process: "Precipitate with subacetate of lead and ammonia; wash the precipitate somewhat with water; boil precipitate with alcohol, and filter hot. (The alcohol takes out the bile-acid salt of lead.) To this solution, after filtration, add enough solution of caustic soda to decompose all the bile-acid salt of lead). We now have a bile-acid salt of sodium.) This solution is to be evaporated to dryness; residue to be extracted with absolute alcohol. (This takes up the bile-acid salt of sodium, leaving the lead behind.) Filter. Precipitate filtrate with excess of ether; allow to stand; pour off supernatant fluid; dissolve precipitate in water, and apply Pettenkofer's test. Much care is needed in this; for some lead flows on closely throughout."

the former, it is usually associated with *nervous debility* or general depression of vital power; except when accounted for by the medicinal use of potassa, soda, or lithia. Excess of the phosphatic salts, and of oxalate of calcium (oxaluria), often accompanies alkalinity of the urine. The importance of the presence of oxalate

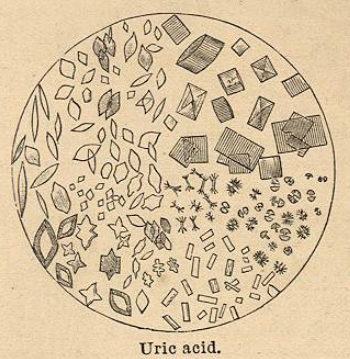
Fig. 17.



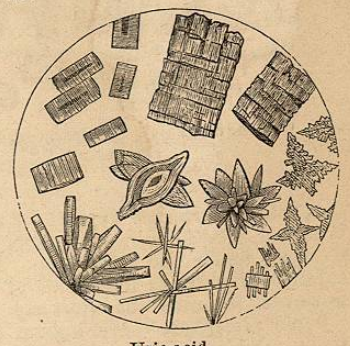
Urea.

of calcium has probably been overrated. Garrod and Parkes have shown reason for believing that a little oxalic acid exists naturally in the blood; and Leared and Duckworth have caused artificial oxaluria by the free ingestion of lime-water into the stomach.

Fig. 18.



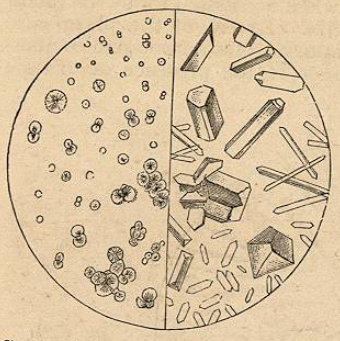
Uric acid.



Uric acid.

Carbonate of *ammonium*, when present in the urine, causes it to **effervesce** on the addition of an acid, from the escape of carbonic

Fig. 19.



Carbonate of calcium. Hippuric acid.

acid gas. The change of color produced by ammonia in turmeric paper will, also, *disappear when it is heated.*

Ammoniated urine becomes so by the decomposition of urea, and its conversion into carbonate of ammonium. When the bladder is inflamed, and contains unhealthy mucus, this decomposition occurs, either in the bladder, or in the urine shortly after it has passed; making it alkaline in reaction, and effervescent when acid is applied. In cases of *much less frequency*, urine will effervesce with acid from the presence of *carbonate of calcium.*

Sediments occur in the urine, either when first passed or after standing, from its containing substances (1) insoluble in it, or (2) precipitated upon its cooling, or, (3) from chemical changes rapidly taking place. Such sediments may be examined both chemically and microscopically.

A *fawn-colored* deposit, not crystalline, which is *redissolved when the urine is heated*, consists of **urates** of *ammonium* and sodium. Urate of ammonium is also immediately dissolved by solution of ammonia or of potassa.

Fig. 20.

Octahedral oxalate of calcium crystals. (Bowman.)

Fig. 21.

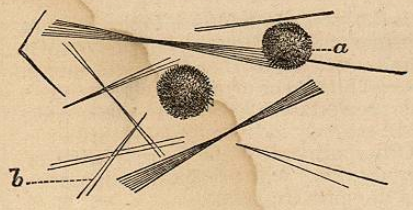
Dumb-bell shaped crystals of oxalate of calcium. (Bowman.)

A much more *rare* deposit, of **cystine,** has a similar color; but it is not soluble by heat, and is but slowly dissolved by alkalies. Cystine, under the microscope, shows rosette-like or hexagonal crystals, sometimes like those of chloride of sodium; the latter, however, is much the most soluble in water. The crystals of "triple phosphate" are known from those of cystine by being freely soluble in *dilute acids.*

Heavy red sand, at the bottom of the vessel, insoluble in hydrochloric acid, but *dissolved by nitric acid* and also by *alkalies* (as liquor potassæ), is **uric** (lithic) **acid.** When *strong* nitric acid is

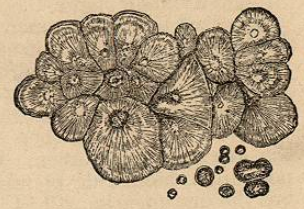
added to deep-red urine containing urate of ammonium in excess with purpurin, solution occurs, with effervescence; and a brownish deposit falls, of uric acid chiefly. When evaporated to dryness, the addition of ammonia to the deposit will produce the purple *murexide.* Similar reactions occur with uric acid itself, acted upon by strong nitric acid.

Fig. 22.



Crystals of tyrosin obtained by the evaporation of urine. (Frerichs.)

Fig. 23.



Crystals of leucin. (Beale.)

Blood-corpuscles sometimes fall to the bottom of urine as a colored sediment. They are not soluble in acids or alkalies, and may be distinguished by aid of the microscope.

A *whitish* deposit, *not at all dissolved by heat*, but dissolved by nitric acid, consists of **earthy salts,** phosphatic or oxalic. If *oxalate* of calcium, it will not be dissolved by *acetic* acid; if *phosphates*, that acid will render the liquid clear. Phosphatic deposits occur (even if not excessive in amount) when the urine is alkaline. Nevertheless, excess of the phosphates is indicated, by the urine becoming turbid when heated, and clearing up when acetic acid is added.

A *creamy-white* flocculent and *ropy* deposit, not dissolved on agitating the liquid, is probably **mucus.** A greenish-yellow settling, diffused when shaken, and which is dissolved by and forms a *jelly* with liquor potassæ, may be concluded to be **pus.**[1]

The **microscope** may detect, even in urine scarcely opaque, or in its residue after evaporation—

Blood-corpuscles, disk-like, or jagged and out of shape;
Mucus-corpuscles, mingled with epithelial scales or cells;
Pus-corpuscles, granular, containing several nuclei;
Epithelial cells or scales, from the kidney or bladder;
Tubular casts from the kidney (desquamative nephritis);
Spermatozoa; thread-like, with one end ovate and expanded;
Uric acid crystals, variously shaped, as lozenges and square prisms;

[1] Dr. Day, of Geelong, tests for pus with a fluid prepared by exposing a saturated alcoholic solution of guaiacum to the air until it has absorbed oxygen enough to cause it to become green with iodide of potassium. A drop or two of this will give a clear blue color with a very small amount of pus moistened with water.