PART I.

PRINCIPLES OF MEDICINE.

SECTION I.

GENERAL PATHOLOGY.

Disease may be defined as a perversion either of the functions or of the structure of the body or of any of its parts. It is, in other words, a deviation from the normal physiological state or action of the organism, under the disturbing influence of **morbid causes.**

The **seat** of disease may be

In the **constitution**: *e. g.*, secondary syphilis; scrofula.
In special **tissues**: *e. g.*, mollities ossium.
In particular **apparatus**: *e. g.*, dyspepsia; neuralgia.
In individual **organs**: *e. g.*, pneumonia; cirrhosis; hydatids.
In the **blood**: *e. g.*, anæmia; scorbutus; typhus.

Of course, disease may be, and generally is, not limited to what is to be regarded as its principal or original seat. For example, in cholera, while its cause, no doubt, acts first upon the blood, the *ganglionic* system also is affected, as well as the stomach and bowels, etc.

Morbid states of the **system**:—

Fever;
Toxæmia;
Anæmia;
Plethora;
Cachexia;
Depression;
Exhaustion;

Morbid states of **organs**:—

Hyperæmia;
Hypertrophy;
Inflammation;
Hyperæmæsthesia, or "chronic inflammation;"
Atony; Exhaustion;
Atrophy;
Degeneration.

Of the above, the *most important general* or *systemic* morbid states may be included under **fever, toxæmia,** and **cachexia**; constituting a sort of "tripod" of systemic disease.

A similar tripod of the most frequent and important *local* disorders may be established, of **irritation, inflammation,** and **atrophy.**

GENERAL PATHOLOGY OF AFFECTIONS OF THE SYSTEM.

FEVER.

In using the term **fever,** as applied to a morbid *state* of the system, we must remember that the same word is also used as a part of the designation of several *complex diseases:* as typhus fever, yellow fever, remittent fever, etc. This double use of the word is unfortunate, but cannot now be avoided.

Symptoms of Fever.—*Increased heat* of the whole body;
Dryness of the skin, mouth, etc.;
Diminution in *bulk* of the *excretions;*
Muscular debility;
Frequency of the pulse;
Functional disturbance of stomach, brain, etc.

Heat is the most essential characteristic of the febrile state, having given name to it in all languages.

Notwithstanding the scantiness in *quantity* of the stools, urine, and perspiration in fever, it has been shown by Virchow, Vögel, Böcker, Parkes, Jenner, and Hammond, that the *actual amount of solid matter excreted,* especially by the kidneys, *is generally increased.* Although none of these observers have made chemical examination of the *expired air* during fever, we have, in the *heavy, offensive odor* of the breath, evidence that it, too, contains an excessive amount of decomposing organic material. It is highly probable, also, that *much excrementitious matter is,* during fever, *retained in the blood.* It has been observed, that if a *local inflammation,* as pneumonia, occurs during the febrile attack, the *excess of secreted solids disappears* until the inflammation has passed. Dr. C. Anton Errald, upon the basis of numerous careful observations, asserts that the amount of carbonic acid in the urine is always increased during fever.[1]

This increase of the disintegration of the substance of the body is, at present, one of the most prominent and interesting phenomena connected with the pathology of fever. The whole subject, however, is surrounded by obscurity, notwithstanding the fact that the *symptoms* and aspects of the febrile state have been familiar ever since man became a prey to disease.

On the basis of the facts observed and scrutinized at the present time, I think we may venture to throw out a comprehensive **theory** of fever. Thus—its essential phenomenon is increased **heat** of the body; this being produced by **excessive tissue-metamorphosis,**[2] under an abnormal "tension-condition" (Virchow)

[1] Philadelphia Medical Times, September 20, 1873; translated from Archiv für Anatomie, Physiologie, etc.

[2] The directness of the relation in the body between temperature and oxidation is well illustrated in the researches of Prof. H. C. Wood, Jr. (Am. Journal of Med. Sciences, July, 1871), upon the action of nitrite of amyl. This substance, whose influence in retarding oxidation can be demonstrated in a jar containing phosphorus, exhibits a remarkable power of lowering the temperature of animals to which it is administered.

of the **ganglionic nerve-centres,** which abnormal condition is the result (Addison) of either, 1, **corpuscular** toxæmia, or 2, **plasmic** toxæmia, or 3 (Campbell and Müller), sympathetic **irritation** from local inflammation.

A pathological classification of fevers, convenient for some purposes, is, into **irritative, reactive,** and **toxæmic** fevers. The *ganglionic* nervous system would seem to be most involved in the first, or *phlegmasiæ* (pneumonia, pleurisy, etc.); the *spinal* system in the second (intermittent and remittent fevers); the *whole* nervous system, and prominently the brain, in the third, as typhus, typhoid, and "spotted" or cerebro-spinal fever. Dr. Allbutt compares *intermittent* fever, as a periodic discharge of tension with disengagement of *heat,* to epilepsy, which is a periodic discharge of tension in the form of *motion.*[1]

TOXÆMIA.

Toxæmia, more properly *toxicohæmia* (from τοξικον, poison, and αἷμα, blood), is a term used to indicate *poisoning of the blood.*

After all the long and reiterated disputes between the advocates of the exclusive *solidist* and *humoral* pathologies, it has become a matter of general recognition that *both* the fluids and the solids are involved in almost every disease—their mutual interdependence making the contrary impossible.

Certain diseases, however, more than others, are believed, upon the strongest evidence, to depend upon a chemical and functional *change in the blood,* to which the name of toxæmia is applied.

Toxæmia may originate: 1. By the introduction into the blood of morbid poisons *from without;* as in syphilis, smallpox, remittent fever, etc. 2. By morbid alteration from processes occurring *in the blood itself.* 3. By *absorption* of poisonous material, by the vessels, from diseased parts of the body; as in purulent infection after wounds, etc. 4. By the non-excretion, and consequent accumulation in the blood, of post-organic or excrementitious substances, which, by their own properties, or by the chemical changes they undergo, prove injurious to the system. Obstructive jaundice and uræmia afford the best examples of this last occurrence.

1. All of the *zymotic* diseases (*e. g.*, exanthemata, yellow fever, diphtheria) have their origin explained by the first of the above modes of blood-poisoning.

Yet, our knowledge of the very existence of several of these "morbid poisons" is inferential only. Our idea of their nature is conjectural; and our reasonings upon their mode of action upon the blood and system at large are entirely speculative.

Some facts, however (see *Simon's Lectures on Pathology*), are well deserving of notice.

a. The effects of these poisons, when introduced into the body, are both *local* and *constitutional* symptoms. The *constitutional* symptoms, which begin the attack, are *nearly alike for them all;* the *local* symptoms are *peculiar for each one.*

b. The smallpox virus is the most readily studied of all of these causes. The material is evidently **volatile,** or at least portable by

[1] Brit. and For. Medico-Chirurg. Review, July, 1870, p. 155.

the air,[1] as it acts often through *considerable distances;* and it is **soluble,** because it infects, sometimes, the fœtus in utero, which has no communication of fluids with the mother, except by placental *endosmosis.* The poison of **primary** *syphilis* is not thus transmissible, although it is directly **contagious** by *inoculation;* that of **secondary** syphilis is not[2] contagious, but *is* **transmissible** by descent.

c. **One** attack of smallpox, scarlatina, measles, whooping-cough, usually gives **immunity** for the rest of a lifetime. It may, from this, be argued that besides the *materies morbi* or *causative* matter, *another* material must exist *in the blood* of the susceptible person, which *combines* with the former (thus producing *the disease*), and which is *exhaustible.*[3] (Illustration: the saturation of a carbonated alkali by an acid; after a certain portion of the latter has been added, it will *cease to effervesce* with any subsequent addition of the same.) Paget believes that the change effected occurs in the solid structures of the body.

Vaccination can be best explained upon this view. Just as more than one acid will neutralize potassa or soda, etc., so that after it has been saturated with sulphuric acid it will not react with nitric—so the virus of the vaccine disease appears capable of *saturating* and *exhausting that material in the body, the presence of which constitutes the susceptibility to variola.*

No such immunity after a single attack is found to exist in the case of the miasmatic fevers (remittent, intermittent). That element in the blood for which *their* morbid poison has affinity is, therefore, not exhaustible. Several reasons exist for conjecturing this element to be the *red corpuscles* themselves.

Of the different hypotheses propounded in regard to the *modus operandi* of **zymotic** (epidemic, endemic, infectious, or contagious) causes upon the blood, that which has best stood its ground is that of **catalysis,** or continuous molecular action.

Liebig first urged this theory, upon the analogy between the action of yeast in producing fermentation, and that of a virus, as of smallpox, in producing its effects upon the system, through the blood. It is true, that the blood does not *ferment;* the action is therefore not *similar*, but **analogous.** Chemical action, of a certain kind, going on in the particles of the yeast, or of a virus, is by their contact with another substance, communicated to or instigated among the particles of the latter. A *mechanical* analogy to illustrate this is the setting in motion of one cog-wheel by another. A physical illustration, less remote, is that of the extension of fire from a burning body to other combustibles near it.

Davaine, Hallier, Salisbury, and others assert the discovery of

[1] Chauveau's experiments, if accepted as entirely exact, disprove the *volatility* of the virus.

[2] Recent experiments have occasioned doubt as to the correctness of this commonly accepted statement. Indeed it appears to be proved by Lee, Waller, Pelizarri, and others, that *occasional* transfer of constitutional syphilis by inoculation is possible. See Lectures on Surgical Pathology, by Henry Lee, F.R.S., 1870.

[3] Reflection will show that there is no real incompatibility between this theory and that of *zymosis*, to be mentioned presently. We have not space, however, for the further discussion of so speculative a subject.

special organic forms, chiefly of microscopic fungous vegetation, in the blood and other fluids of patients suffering with carbuncle, cholera, intermittent fever, syphilis, gonorrhœa, etc. The importance of these forms in the causation of disease is denied positively by Thudichum among medical authorities, and by Berkeley, a distinguished naturalist. The question is certainly yet *sub judice.*[1]

2. Toxæmia from *spontaneous changes in the blood itself*, under causes or conditions which do not affect the solid structures of the body, if it occur, must be rare. *Heat-stroke* may be, in part, an example; that is, the dangerous or fatal effect of extreme heat, with exhaustion, away from the direct influence of the rays of the sun. Here the blood probably undergoes a chemical change which renders it unfit to vitalize the nerve-centres and other organs.[2]

Perhaps **pyæmia, ichoræmia,** or **septæmia** (pus-forming blood—contaminated blood—blood-decomposition), as nearly the same affection is called by different authors, may be supposed to occur sometimes spontaneously. Much more often, however, such an affection is ascribed to the next mode of causation.

3. Absorption of deleterious material, by the bloodvessels or lymphatics, from parts of the body in a state of disease, may cause *purulent infection*, or **pyæmia.**

Absorption of pus (containing pus-cells) is unlikely to occur without a solution of continuity in the vessels. But in *arteritis*, or *phlebitis*, suppuration of artery or vein may introduce pus into the blood.

In the greater number of cases, it is not pus, but an unhealthy material of a less definite nature, which contaminates the blood by its absorption. This may take place after wounds or surgical operations—from the womb in the puerperal state, etc. Pyæmia is attended by great prostration, rapid pulse, copious perspirations, low delirium, and the depositing of pus and formation of abscesses in different parts of the body. It very often begins with a chill.

4. Toxæmia from **non-elimination** of the secretions may follow, of course, upon prolonged constipation, obstruction of the biliary duct, inaction of the liver, or suppression of the action of the kidneys.

Effort is made (according to the adaptations of nature), when one emunctory fails to act, to carry out its *excreta* by other channels. Especially do the skin and kidneys act thus *vicariously* for each other.

When the blood is in no way rid of those effete particles which should make the *solids of the urine*, the resulting condition is called **uræmia.** Its symptoms are, pain in the head, dulness of sight and hearing, vertigo, nausea, and vomiting; ending, unless relieved, in convulsions, coma, and death. Pregnant women sometimes have

[1] See an article upon it by H. C. Wood, Jr., M.D., Am. Journ. of Med. Sciences, October, 1868.

[2] The experiments of Wood and others, which have been thought to show the absence of a fatal blood-change in heat-stroke, do not appear to me to establish that conclusion.

uræmic convulsions (C. Braun), from fœtal pressure obstructing the renal circulation.

The term **cholæmia** is less used, though quite as justifiable as uræmia. It means, retention in the blood of the excrementitious matter of the bile, from inaction of the liver. **Cholesteræmia** is a term preferred by Dr. A. Flint. Jr., who asserts cholesterin to be the excretory ingredient of bile.

Jaundice is of two origins: 1, **obstruction** of the biliary ducts, with reabsorption of bile into the blood; 2, **suppression** of the secretion of the liver. (A third is possible—perhaps present in the jaundice of infancy; viz., **excessive formation** of yellow pigment in the blood, and its deposit in the skin, etc., without disorder of the liver.)

In jaundice from obstruction and reabsorption, the symptoms are milder and the state less dangerous than in that from suppression of the action of the liver. Severe, and even fatal disease of the liver may occur, however, without jaundice.

Dr. Harley has shown that the diagnosis between these two forms of jaundice may be made, on analysis of the urine, by finding the *coloring* matter of the bile always in the urine in *both*, but the biliary *acids* only in the *obstructive* form.

Slight and transient cholæmia is, no doubt, common. Although the term "biliousness" is much abused, it is not always quite a misnomer. As signs of the condition mentioned, we find nausea, bitter taste in the mouth, constipation, and dizziness, with yellowness of the tongue, conjunctiva, and skin.

ANÆMIA.

Anæmia (*spanæmia*, *hydræmia*) is the common term indicating poverty of the blood. The density of that liquid is diminished, and there is a deficiency in the number of the red corpuscles. Exhausting hemorrhages or discharges, severe attacks or long continuance of disease, insufficiency of food, etc., may cause this condition.

It is shown by paleness (sometimes with occasional flushes) of the face, even of the lips, and tongue, as well as of the hands; debility; feebleness and excitability of the pulse; frequently, palpitation of the heart, and a *bellows murmur*, audible especially near the base of the heart, to be carefully distinguished from the valvular murmurs of organic disease. *Nervousness*, and neuralgic pains, are also very common in the anæmic.

Chlorosis, although sometimes separated from anæmia, is generally associated with it, occurring in young females. The name is given on account of the peculiar sallowness of the complexion. *Perverted appetite*, as for charcoal, slate-pencils, etc., is one of its occasional symptoms.

Plethora involves an *excess* in the density of the blood, and in the number of its red corpusles; the opposite to anæmia. It is shown by a high color of the face, distension of the bloodvessels, a full, strong, but rather slow pulse, and general roundness of the figure. It may exist without actual deviation from health; but the plethoric are especially liable to acute inflammation, active congestions, and hemorrhages.

CACHEXIA.

Cachexia (from κακος, bad, and ἕξις, habit) is usually understood to mean a *depraved habit of system;* an error of development and nutrition, affecting the general state of the blood and organs with perversion.

There is, at the same time, no good reason why we should not speak of *local* as well as general cachexiæ; although this has not been usual.

Melanæmia, *Addison's disease*, and *leucocythæmia*, may be regarded as cachexiæ.

Melanæmia is the name given (Frerichs) to a state of the blood common in severe malarial fevers, in which the coloring matter (pigment) escapes from the corpuscles, and is deposited in the liver and other organs.

Morbus Addisonii, Addison's disease, is a rare constitutional malady, in which anæmia coincides with bronzing of the skin, disease of the suprarenal capsules, and progressive debility, usually ending in death. The capsular disease is not shown by any definite local symptoms during life; and its frequent connection with the *cachexia* has not been explained.

Leucocythæmia (leukæmia) has been, after Virchow, and Bennett, recognized as a condition in which the number of colorless corpuscles in the blood is *increased;* sometimes numbering one to four, three, or two, instead of one to fifty (normal) or more, of the red corpuscles. This, of course, can be ascertained only by means of the microscope.

Enlargement of the liver, and still more of the spleen, and disease in these organs as well as in the thyroid and lymphatic glands, are found to attend this disorder. It most frequently affects men. Its symptoms are, pallor, emaciation, diarrhœa, epistaxis (bleeding from the nose), or other hemorrhages, and dropsy.

Much more frequent, and therefore important—the most common and destructive of all cachectic affections, is tuberculosis.

While diverse opinions exist as to the essential nature of tubercle and its origin, there is a general agreement among pathologists and clinical observers, upon many of the following points:—

1. **Tuberculosis** and **Scrofulosis** are identical as a *diathesis.*[1] The term *scrofula* is generally applied to certain slow inflammations, abscesses, ulcerations, and other disorders of the skin, mucous membranes, glands, and bones, which occur especially in young persons, and are characterized by the *moderate* degree of vascular excitement attending them, with the great *obstinacy* or *chronicity* of their career. In many cases, also, of external scrofula, particularly in the glands, a deposit of curd-like or cheesy material is found.

[1] Schönlein, Virchow, Jenner, and C. West are among those who have denied this.

2. Of the **causes** apparently connected with the production of the tubercular or scrofulous diathesis, the most obvious and constant is *hereditary* **predisposition.**

3. This diathesis may, however, undoubtedly be **acquired** without inheritance. *Change of* **climate,** from a warm to a *cold and damp* locality, will often induce it. Other **depressing** influences promote it, such as want of *food*, *light*, *pure air*, and *warmth*, sedentary habits, etc. But all of these often fail to generate any form of tubercular disease. Dr. Wilks, of Guy's Hospital, London, reasserts the opinion of Barlow, that "tubercle is apt to be developed in an organ according to its functional and vascular activity;" supporting this by Rokitansky's observation, that cyanotic patients, having a preponderance of venous blood, are especially free from tubercular disease of the lungs. But Baudelocque, McCormack, Parkes, and others have adduced abundant proof that *close living*, in an impure air, is highly promotive of phthisis. Probably the *normally* active function and vascularity of the lungs render them particularly *susceptible* to injury when, in an impure atmosphere, this functional activity is impeded and depressed.

4. Tuberculosis may be pathologically defined as a constitutional tendency to the formation of blood, the plasma of which is defective in organizable capacity; so that, in nutrition, instead of healthy tissue, it forms in one, or very often in many, of the organs, **aborted blastema,**[1] which accumulates as a deposit. This deposit is called tubercle; the process, **tuberculization.** This view of tubercle has been denied of late, it appears to me upon insufficient grounds, by Dr. T. K. Chambers and others. Some even assert a *specific* character in tubercle. Villemin, Colin, and Lebert declare that they have propagated it in animals by inoculation. It would appear probable, however, from the Report of a Commission of the French Academy of Medicine upon Villemin's experiments, that "*all inflammatory products* have, when inoculated, similar effects to those of tubercle." Dr. Marcet of London has confirmed, by his own experiments upon animals, the inoculability of tubercle from the sputa of patients. The investigations of Lebert and Wyss, Cohnheim, Fränkel, Sanderson, and Wilson Fox may be considered to have overthrown Villemin's hypothesis of *specific* tubercular inoculation.[2] Niemeyer believed that while a peculiar caseous metamorphosis, most frequently the result of inflammation, may, by resorption and infection, propagate phthisis in animals inoculated, yet "a purely contagious origin of actual tuberculosis has not been proven."

5. But the tubercular **diathesis** may exist *without tuberculization.* Its influence is then shown, especially, in **modifying** *inflammatory* or *other morbid processes;* giving them a *lower*, *slower*, and more persistent or intractable type. Thus, many cases of what is called *tubercular meningitis* in children occur, with fatal result, in which

[1] From βλαστανω, I *bud;* used to mean *tissue-forming material.*

[2] See Brit. and For. Medico-Chirurg. Review, July, 1868, p. 26. Chauveau, however, is asserted to have shown that when tubercle and pus were injected in *filtered solutions*, only tubercle produced tubercle. (See Damaschino on the Etiology of Tubercle, Archives Gén. de Méd., Oct. 1872.)

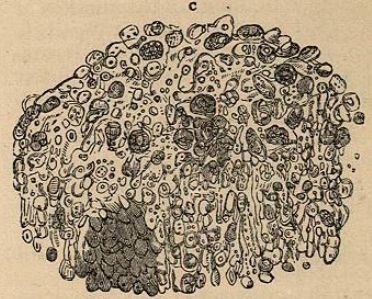
(Bouchut, Hughes Wilshire, etc.) *no tubercular* **deposit** *is found;* yet the disease is modified by the diathesis. Some regard phthisis as, in like manner, a *tubercular pneumonia;* but this designation can only apply, properly, to a certain proportion of cases.

6. Tubercle is distributed either in **regularly-formed** masses (miliary tubercles, etc.), or **irregularly,** through the tissues of organs. The most amorphous (shapeless) and homogeneous examples of it have been called **infiltrated** tubercle. The *size* of the masses of tubercle varies from that of a pin's head to that of a hen's egg.

7. The two varieties of tubercle heretofore described are the **semi-transparent, gray, granular,** and the **yellow, opaque, caseous** tubercle.[1]

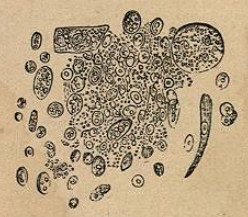
8. **Neither** of these forms ever undergoes **organization.** They are never *vascular.* They are deposited outside of the bloodvessels, and not in *non-vascular* tissues, such as cartilage, etc. Dr. Beale inclines to the opinion[2] that tubercle corpuscles, sometimes at least, originate in the *nuclei of the vascular walls.* Cornil and Bastian[3] entertain analogous views. Schrœder van der Kolk, Radclyffe Hall, and others consider pulmonary tubercle to result from a degeneration of the epithelial lining of the pulmonary air-cells. But the very existence of this epithelium is not admitted by all micrologists. Dr. C. J. B. Williams has applied the term *phthinoplasm* to the degraded bioplastic material of the blood whose formation is attended by the wasting and other characters of "consumption." Virchow describes infiltrated tubercle of the lung as the cheesy metamorphosis of an *exudation.*

Fig. 1.



Yellow tubercle.

Fig. 2.



Gray tubercle.

9. The **gray** tubercle, when *alone*, may be subject to **cornification;** *i. e.*, drying into a horny substance as hard as a shot. When

[1] Several pathologists now assert that only the miliary semi-transparent and opaque granular matters are really tubercular. Further allusion to this question will be made presently.

[2] Microscope in Pract. Medicine, 1867, p. 305.

[3] Edin. Med. Journal, 1867, p. 875.

with the yellow tubercle, or after conversion into it (Rokitansky),[1] the gray may undergo **softening.**

10. **Yellow** tubercle *usually* **softens;** sometimes it **cretifies;** *i. e.*, becomes chalk-like, by degeneration.

Fig. 3.

Tubercle corpuscles.

11. The softening of tubercle is sometimes, at least, *spontaneous;* not depending upon any agency of surrounding parts. In regularly formed tubercles it commences at the **centre;** in the irregular, at *any* part. *Tuberculous* **softening** must not be confounded with *suppuration* of inflamed tissue; although they are often mingled, and have been considered identical by some pathologists.

12. Examined with the microscope, tubercle is found to consist essentially of: 1. An *amorphous, granular* material, containing *irregular solid* corpuscles (tubercle-corpuscles), considered (Virchow) to be *shrivelled nuclei.* 2. Elements of *disintegrated tissue* of the part involved; as epithelial cells, fibres, etc. 3. Results of *degeneration; e. g.* oil-globules, pigment, calcareous particles, etc. 4. Results of *inflammation* of surrounding parts; lymph, pus, exudation-corpuscles. 5. Extravasated *blood-corpuscles,* from hemorrhage, the effect of obstruction or ulceration of vascular trunks. Sometimes *giant cells* are observed. Rokitansky, Virchow, Wagner, and others have recognized these, under different names. Prof. Schüppel (of Tubingen) gives the following account of the constituent elements of a tubercle: 1. Multinuclear giant-cells, resembling the "myeloplaxes" of myeloid or giant-celled sarcoma; 2. Large cells of an epithelial type; 3. Small round cells like lymph-corpuscles; 4. A peculiar reticulum, in the meshes of which these cells lie. This pathologist considers the giant-cells to be the essential element of tubercle; although the bulk of it consists of cells of an epithelial type. As observed in the lymphatic glands, he asserts that, after a short stage of "development," each tubercle undergoes retrogressive changes. These are: 1. Necrosis, leading to *caseation* and its sequelæ or terminations, cretification, and softening; 2. Transformation into *fibrous tubercle;* 3. Formation of calcareous or other concretions in the centre of the tubercle; 4. Resorption, which is probably always preceded by necrosis of the tubercle.

13. Tubercle contains **no truly specific,** heterologous **form.** All that it holds is the consequence of abortion and degeneration.

14. The process of **tuberculization** or deposit of tubercle in an organ may occur (Rokitansky)—

a. **Insensibly,** in the course of ordinary nutrition.

b. With **hyperæmia,** or local determination of blood.

c. With **inflammation;** *i. e.*, as a *product* or concomitant of the inflammatory process.

15. The effects of the tubercular deposit *upon the part* are: 1. **Obstruction,** and arrest or impairment of function. 2. **Inflammation;** *e. g.*, in phthisis pulmonalis (consumption), which has, in its usual form, been designated (Condie) *tubercular pneumonia,* from the common occurrence of inflammation of the lungs with deposit of tubercle. 3. **Ulcerative destruction** of the tissue by the repeated new formation and softening of tuberculous matter, producing **cavities.**

[1] Laennec accepted this conversion, which Rokitansky for a long time denied, but now considers frequent. Virchow admits it, and Hérard declares it to be universal.

16. Tubercle, once thrown out, is never (as a whole) *absorbed.* It can only be *eliminated, cretified, or cornified.* Elimination is the most common. After this has happened, sometimes *callous* cavities are formed by a process of **cicatrization.**

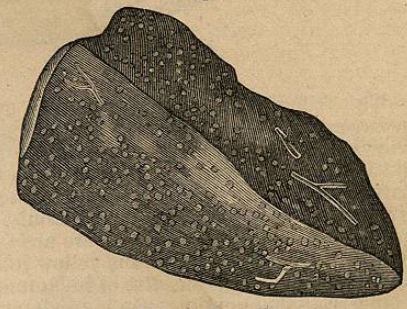
17. The *order of frequency* with which different organs are affected with tubercle is (Rokitansky) as follows:

Lungs;	Spleen;
Intestines;	Kidneys;
Lymph-glands;	Liver;
Larynx;	Bones;
Serous membranes;	Uterus;
Brain;	Testicles.

Except in the case of *children,* in whom the *lymph glands* and the spleen stand first on the list.

18. But the organs most frequently *first invaded* by tubercle are, at all times of life, the **lungs** and **lymph-glands.**

Fig. 4.



Miliary tubercles.

19. The *parts especially preferred* by tubercle for its deposit are, in the lungs, the apex; in the pia mater, about the base of the brain; in the brain, the gray substance; in bones, the cancellated structure; in the bowels, the lowest part of the ileum; in the testicle, the epididymis; in the female generative apparatus, the Fallopian tubes and fundus of the uterus.

20. The immense experience of Rokitansky gives origin to the statement that tubercle has certain general **incompatibilities;** the most important of which are, with cancer, with typhus, with ague, and with goitre (bronchocele, enlargement of the thyroid gland). These incompatibilities are, however, *general,* not *universal;* as, for instance, a considerable number of cases have been observed, in which cancer and tubercle were undeniably present in the same patient. Burdel (1869) has observed this in more than a hundred instances.

21. The only possible cure of tubercular disease (*e. g.*, of the lungs in phthisis) after the deposit has occurred, consists in the total **elimination** (or absolute quiescence by cornification or cre-