tification) of the tuberculous matter, and *improvement in the general* **hæmatosis** (*i. e.*, blood formation), so that *no new tubercle is formed.* The two great indications, therefore, in the treatment of pulmonary consumption (Radclyffe Hall) are, to gain **time** and **tone**; *time*, by allaying or preventing pulmonary or bronchial inflammation and irritation; and *tone*, by strengthening the patient's system by all possible hygienic and therapeutic measures.

Fig. 5.

Apex of tuberculous lung.

The following additional remarks upon tuberculosis are made necessary by recent discussions, especially in connection with the views of Virchow, Oppolzer, and Niemeyer, on the pathology of phthisis. A retrospect of the history of the subject will show that (after Bayle) Laennec[1] first established the doctrine, sustained afterwards with great vigor by Louis, that tubercle is a specific product of a diathesis; that there are two varieties of it, the miliary semi-transparent, and the yellow infiltrated; and that tubercle is, when deposited, *causative* of inflammation. Andral maintained about the same opinion. In consistency with his other pathological views, Broussais insisted that the yellow infiltration is certainly inflammatory, and that therefore the other variety must be so also. Louis, especially, denied this. Andral, Cruveilhier, Reinhardt, Addison, Condie, and others have, since Broussais, recognized the importance of inflammation as a *factor* in the history of pulmonary

[1] Dr. Thomas Percival asserted, in 1789, that "inflammation is only an occasional concomitant of the formation and softening of tubercles." This has been admitted by Graves, Trousseau, Bricheteau, and Jaccoud. See Teissier, London Med. Record, Feb. 5, 1873.

It appears to be Portal who first definitely applied the term *tuberculous* to what are now commonly called *caseous* deposits. Sylvius, Mead, Cullen, Reid, and especially Baillie, described the process of their formation. Bayle anticipated Laennec in teaching the existence of a tuberculous diathesis; he made, however, six kinds of consumption, of which one was "granular," and another "tubercular."

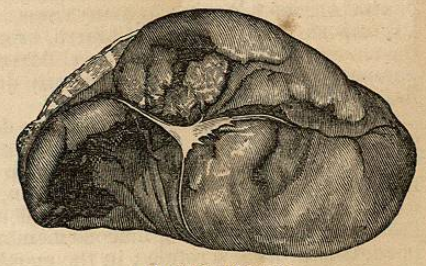
consumption. Rokitansky has clearly declared the belief, that both the semi-transparent granular and the opaque caseous forms ought to be considered as tuberculous exudates: the gray undergoing cornification when alone, and softening when present with the other variety, while the latter may either soften or become cretified. Virchow, Hérard, and Cornil now assert that only the miliary transparent and opaque *granular* matters are really tubercles.

Buhl proposed (1857) the theory of the *resorptive* origin of phthisis; yellow infiltration occurring first, and the absorption of a portion of this producing the semi-transparent granular tubercles. Waldenburg has supported a similar opinion. The idea of a specific tubercular *virus* is associated with this view. Niemeyer accepted the assertion of the dependence of miliary tubercle upon the presence of caseous masses; but without so positively stating the nature of the connection. The order of occurrence as described by him is—1. Catarrhal pneumonia or broncho-pneumonia; 2. Caseous pulmonary infiltration; 3. Secondary formation of tubercle. The first two of these stages may take place, in many cases, even of fatal consumption, without the third. Niemeyer's dictum has become quite famous, that "the greatest danger for the majority of consumptives is, that they are apt to become tuberculous."

On the other hand, the well-known experiments of Waldenburg, Cohnheim, Fränkel, Burdon Sanderson, and Wilson Fox lend support to the view that the miliary granulation may be primary, perhaps inflammatory, and the yellow infiltration the result of *degeneration* either of tubercle, or of *any* inflammatory or other morbid product. Dr. C. Ellis, of Boston, U. S., propounded this as the mode of origination of yellow tubercle, in 1860. Virchow, moreover, has shown that "caseous masses" occur sometimes entirely without the previous occurrence of pneumonia.

With Profs. Austin Flint and Alonzo Clark,[1] the writer is not prepared to accept, as a whole, the resorptive theory of consumption, or in any sense, the necessarily secondary nature of tubercular deposits. Dr. Austin Flint's careful analysis of 670 cases of phthisis (*N. Y. Med. Record*, Feb. 1, 1873) affords substantial clinical proof that *neither inflammatory affections within the chest, nor hæmoptysis*, can have much, if any, *causative* influence in the development of phthisis. Nor does the

Fig. 6.



Cicatrix of lung.

[1] N. Y. Medical Record, Nov. 15, 1870. Mandl also rejects the "caseous-pneumonia" theory of phthisis; Traube and Skoda deny at least "phthisis ab hæmoptoë."

same experience present evidence that scrofulous disease of the glands has to do with the etiology of pulmonary tuberculosis. The following questions may be regarded as, at present, *sub judice*.

1. Are both the semi-transparent miliary granulations and the yellow caseous infiltrations to be called tubercles, or only one of these two forms; and, if but one, *which* is tubercle? This appears to be a question, not of fact, but of *nomenclature;* to be decided by the general suffrage of the profession. Meantime, accuracy in clinical description requires that terms be used which *define* the special lesions occurring in connection with phthisis, otherwise than by the use of the words tubercle or tuberculization alone. On the whole, it seems best to restrict the term tubercle (in accordance with etymology) to that which *began*, at least, as a nodule or nodules; while the terms caseous deposit and caseous infiltration may well apply to amorphous (steatomatous?) accumulations, in the lungs or elsewhere. Some pathologists (as Bastian) would drop the term tubercle altogether. *Granular* degeneration (Empis) is an expression used almost in the same sense by a few authors. But nothing seems as yet to be finally settled upon this point.

2. Does one of the above-mentioned deposits always precede and produce the other? And, if so, which is primary?

3. Does inflammation, as a rule, precede and produce tubercle, or tubercle inflammation? Likewise, does hæmoptysis produce tuberculization, or, as Laennec taught, tuberculization hæmoptysis? Against the general causative action of pulmonary hemorrhage, we have the valuable experiments of Perl and Lipmann[1] and Sommerbrodt, showing that *blood passing into the sound lung does not of itself act as an irritant*, but is gradually absorbed without producing any observable change, unless moderate emphysema.

Some facts in the clinical history of pulmonary and other affections in children militate against the "catarrhal broncho-pneumonia" and "resorptive" theories of consumption. Thus, 1. Scrofulous *caseous* formations or degenerations of glands, mucous membranes, and bones are common in children, without tubercle of vital organs following in many cases. Virchow has assented to the importance of this. 2. Catarrh, bronchitis, and even broncho-pneumonia are more common in children than in adults; and yet pulmonary phthisis is much more rare during childhood than later in life.

On the whole, without venturing to dogmatize on so difficult a subject, the following seems to be the most probable conclusion: That there is a certain constitution, often but not always inherited,[2] having a proclivity to the *precipitation*, under reduction of vital force, of semi-organized granular and corpuscular deposits from the blood;[3] in the glands, mucous membranes, and bones in childhood, constituting scrofula; in the membranes of the brain, at the same period, making tubercular meningitis; later in life, usually in the lungs, producing phthisis; that such deposition may occur

[1] Virchow's Archiv, Band xlix., Heft 2.

[2] "It cannot be assumed," says Virchow, "that caseous pneumonia creates no inheritable tendency."

[3] Dr. W. Minor Logan, of Cincinnati, asserts the importance of *phosphatic* deposits in tuberculosis; owing especially to deficiency of oxidation.

without inflammation as a cause, and may then produce inflammatory disturbance; while it is, not rarely, thrown down under the influence of local inflammation, especially when pus is formed; also, that resorption is not the usual, but may possibly be an occasional mode of origination of tuberculization, in the lungs or elsewhere.

If these views be correct, it must be recognized as a fact that fatal pulmonary phthisis may occur under several forms: 1. Most commonly, as a progressively degenerative affection of the lungs, with intercurrent, usually successive attacks of inflammation, which hasten very much the fatal termination. 2. As a tubercular or "caseous" pneumonia; subacute in character, or even chronic; but, sometimes, so rapid as to be named "acute phthisis," or galloping consumption. 3. As a simple, slowly progressing, cachectic, non-inflammatory, degenerative disease; constitutional depravation and pulmonary disorganization going on together, and death occurring sometimes from general vital decline, and sometimes from pulmonary obstruction or disability.

(On *Depression* and *Exhaustion*, see Sect. iii., General Therapeutics.)

GENERAL PATHOLOGY OF AFFECTIONS OF ORGANS.

HYPERTROPHY.

Hypertrophy is, strictly, *overgrowth;* an increase of the size and weight of a part without change of tissue. It is only in recent times that this has been clearly distinguished from *enlargement with alteration of tissue;* which is really, in many cases, a *degenerative* change, and therefore akin rather to *atrophy* than to hypertrophy.

Hypertrophy is often, *per se*, physiological or natural; although depending on a morbid or pathological cause. When the bladder, for instance, becomes hypertrophied in consequence of obstruction by an enlarged prostate, although the *latter* is morbid, the increase in the strength and thickness of the muscular coat of the bladder is as normal as is that of the uterus during gestation; in due proportion to the necessities of its use.

A constant law of the animal economy is, that, within certain limits, the **growth** of an organ is in proportion to its **exercise;** provided that this exercise is not too violent, and is alternated with sufficient periods of repose.

The three **causes** of hypertrophy, then, are (see *Paget's Surgical Pathology*):—

1. Increased *exercise* of a part in its healthy functions.

2. Increased accumulation in the blood of the particular *materials* which a part appropriates in its nutrition or secretion.

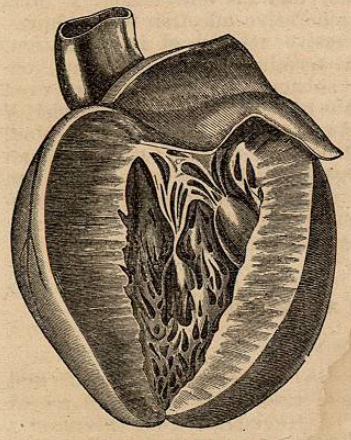
3. Increased *afflux* of healthy *blood* to the part.

We may illustrate the first of these modes of causation by the blacksmith's arm, the legs of the *danseuse*, the cuticle of the *laborer's hand*, *the heart* in cases of *valvular obstruction*, etc.

An example illustrative of the second is found in the enlargement of a healthy kidney, when the opposite one fails, from disease, to remove from the blood its due share of urea, etc.

The third is exemplified in the large growth of hairs around an inflamed ulcer or osseous fracture; by the growth of the bones of the limbs when their nutrition is increased by exercise; by hypertrophy of a bone, a portion of which has been subject to disease with vascular excitement; and by Hunter's interesting experiment of the transplantation of the spur of a cock to its *comb*.

Fig. 7.



Hypertrophy of heart.

Adaptive hypertrophy is remarkably seen in the changes undergone by the skull in proportion to its contents. The cranium is subject to—1, eccentric, and, 2, concentric hypertrophy. The first occurs in cases of *hydrocephalus*, the second in cerebral *atrophy;* the bony case in the one instance *expanding* with its contents, in the other *thickening* so as to fill up the abnormal void.

Corns illustrate hypertrophy extremely well.

Intermittent pressure, or attrition, causes *hypertrophy; constant* pressure, *atrophy* or absorption.

The formation of corns upon the foot illustrates the former of these laws; the wasting away of vertebræ under the incessant pressure of an aortic aneurism affords an example of the latter. *A Chinese woman's bandaged foot, with corns*, exemplifies *both*.

ATROPHY.

Atrophy requires but a few words in this place. Simple atrophy is exactly the reverse of simple hypertrophy; viz., wasting and diminution of a part, without change of structure. But most pathologists include also under the same term such defects of nutrition as result in **degenerative** changes; constituting the two classes of—1, **quantitative**, and 2, **qualitative** atrophy. The latter (*e. g.*, fatty degeneration) is frequently attended by *increase* instead of diminution of *bulk* in the parts affected.

The **causes** of atrophy are:—

1. Deficient *exercise* of a part;
2. Deficiency in the *supply of blood;*
3. Defective supply of *nervous influence;*
4. *Disease* (inflammation, etc.) in the part.

Of the first of these, the atrophy of the mamma of the old maid may afford an example.

Of the second, softening of a portion of the brain from the obstruction of one of its arteries by a coagulum.

Of the third and first together, the muscles of a paralyzed limb.

Of the last, there are many instances familiar to the pathologist, although obscurity often attends their individual history; as the *gouty kidney*, etc.

Quantitative or qualitative atrophy may affect the heart, arteries, brain, muscles, bones, liver, kidneys, pancreas, testicles, etc., and also *morbid products*, *e. g.*, inflammatory exudations, cancer, etc.

Qualitative atrophy, or **degeneration,** will be again alluded to presently.

IRRITATION.

Irritation and inflammation are at once the most familiar in their phenomena, and the most obscure in their nature, of all pathological processes or occurrences. I shall confine myself to a broad statement of what I believe to be most important truths concerning them; although a somewhat argumentative tone may be unavoidable, upon topics which are subject to so much controversy.

Stimulation and **irritation** are often inconveniently confounded. It would be desirable to confine the *former* term to excitation *within physiological limits;* applying the *latter*, irritation, only to such an *excessive* action upon a part as produces *morbid* effects.

With this, which seems to me a necessary postulate—I would define **irritation** as an *arrest of vital movement in a part.* This could only be elucidated by an extended allusion, inappropriate here, to the *correlation of physical and vital forces; life* being considered as *a molecular motion.*

In regard to the *circulation*, to the old and accepted maxim—

Ubi *stimulus*, ibi *affluxus*—

may be added a second—

Ubi *irritatio*, ibi *stasis*,

and, anticipating the account about to be given of *inflammation*, a third—

Ubi *phlogosis*, ibi *effusio.*

The **stasis of irritation** may be either *partial* or *complete; limited* to a very small surface, or widely *extended;* and *transient*, or *continued* for a considerable time.

If *complete*, *extended*, and *continued* in a tissue at all vascular, **inflammation** *follows.*

If the influence of the irritant be very *limited* and *transient*, a temporary stasis, with functional and sensational disturbance only, follows.

If it be *extended* and *continued*, or *repeated*, and yet of power enough to produce a *partial stasis only*, a condition may result to which the name of **chronic inflammation** has been commonly given; of which more will be said hereafter.

The *effects*, or **symptoms** of irritation differ according to the tissue or organ affected. When a nervous expansion or centre is in-

volved, *pain* is the most familiar result.[1] Functional disorder, of the part innervated, also occurs. Irritation of muscular tissue causes tonic *spasm*.

INFLAMMATION.

Inflammation must be considered briefly, as to its *symptoms*, *minute phenomena*, *products*, *terminations* or *effects*, and *post-mortem* appearances.

Its recognized **symptoms** or signs, in a part open to inspection, are *redness*, *heat*, *swelling*, and *pain*.

In **internal** organs inflammation is detected chiefly by *pain*, *increased by pressure* or *motion; obstruction* or *alteration* of the *functional action* of the organ; and *general* (sympathetic) *vascular excitement*. Certain *physical signs* also aid in the diagnosis of inflammation of particular organs (see *Semeiology*).

The **minute phenomena** of inflammation, as seen under the microscope, have been variously construed by different observers. The use of the *term* itself has been, of late years, distorted (Virchow) from its old meaning; and attempts have been made by some (Andral, Eisenmann, Bennett) to do away with it entirely; attempts which fail, because, in proposing other terms, a *part* only is substituted for the *whole*. It is curious, that, of the three terms proposed by three leading pathological writers, **hyperæmia, stasis,** and **exudation,** to take the place of the old word inflammation, *each* expresses a *single* part or element of the process, which *can only be defined by including them all;* while cell-distension and cell-multiplication, made by Virchow so prominent in the process, are only incidental attendants upon it.

The essential minute phenomena of inflammation are, as regards the circulation—

Central stasis;
Concentric hyperæmia;
Exudation.

Other changes affecting the red and white corpuscles, etc., occur, but are of secondary consequence.

The *nature* and *cause* of these phenomena require, for their comprehension, a close consideration of the laws which govern nutrition, the capillary and arterial circulation, and innervation, in their mutual relations, under the influence of *normal stimuli* and of *morbid irritants*.

What are the actual causes of inflammation?

Not section of the nerves; nor division of the arteries (*per se*); nor divisions of the veins; nor ligation of arteries nor of veins; nor (*per se*) of lymphatics. Only such causes as modify the **molecular state** of the tissue, and arrest, for the time, the usual interchange of material between the tissue and the blood, can induce a true inflammation.

Let us, then, revert to our maxims. *Ubi stimulus, ibi affluxus. Stimulation* causes *active hyperæmia. The arteries thus exhibit*

[1] Inman and Radcliffe, especially, have insisted that pain is always a sign of *local diminution of vital energy;* it is on the way toward death.

reflex action; a fact which, in spite of the teachings of Unzer, Hunter, and C. Bell, has been denied or misunderstood by nearly every other physiologist and pathologist down to the present day.[1]

Next, *ubi irritatio, ibi stasis.* Stimulation, carried to *morbid excess*, interrupts, by the molecular disturbance it induces, the *normal life-movement* of the part, and checks the interchange of particles going on between the capillaries and the tissue. Thus *the circulation in the capillaries of the part is arrested;* **stagnation** ensues.

Both of these results, active arterial hyperæmia and capillary stasis, follow from the *same* or *similar* causes acting in *different degrees*. They may and do exist *together;* the one (capillary stasis) at the very **point** of irritation, the other (active hyperæmia) in the vessels **surrounding** it.

What follows? Hydraulics may answer this question. A quantity of fluid in (minutely or potentially) porous vessels, being forced upon a centre whose condition allows little or none of it to be transmitted, an **effusion** must result, through the more or less distended coats of the vessels.

This is expressed by our third maxim: *Ubi phlogosis* (inflammation), *ibi effusio.* This phenomenon, the "exudation," has attracted almost all the attention of many pathologists, to the exclusion of other occurrences, which precede and accompany it; an exclusion which has had detrimental results (J. Hughes Bennett) as regards the practical and therapeutical deductions made therefrom. The importance of the part taken by the arterial circulation in inflammation is well illustrated by the control exercised over it in acute cases, as recorded by a number of observers, by the *ligation* or *compression* of the *main artery* of the limb or other part affected.[2]

Virchow has another theory of inflammation, forming a part of his "Cellular Pathology." He identifies (confounds) **stimulation,** which is physiological or normal, with **irritation,** always abnormal or pathological in kind or degree. All irritation, in vascular or non-vascular parts, is, according to him, either *functional*, *nutritive*, or *formative*. Exudation, or transudation of fluid into the substance of an inflamed part, is admitted only of the more vascular, soft, and superficial tissues. In others (parenchymatous inflammation) the essential effect of irritation of a high degree is said to be, the taking, by their own action or attraction, of more fluid into

[1] Much of the accepted pathology of to-day, and some current notions in therapeutics, are founded upon an erroneous view of the physiology of the circulation, especially in regard to the mode of action of the arteries. The error is, the statement (based upon experiments whose results were only *morbid*, not normal) that the *normal, active* contraction of the arteries always *diminishes* the supply of blood through them; as Virchow expresses it "*the more active the vessel, the less the supply of blood.*" Another generation will attain to the correction of this; and, with it, a revolution in the pathology of inflammation must occur. See the author's *Prize Essay on the Arterial Circulation*, Trans. Amer. Med. Association, 1856. More recently (1858) *Lister* asserted reflex action as occurring in the vessels in inflammation; as well as the central arrest of nutrition. A brief summary of the argument is given by the author in Am. Journal of Med. Sciences, July, 1868, p. 288; and again, in the Trans. of the Amer. Med. Association, 1872, p. 181. An admission of the true view, based on the recent experiments of Legros and Onimus, occurs in the Brit. and Foreign Medico-Chirurg. Review, April, 1871, pp. 291, 303.

[2] See an article on this subject by S. W. Gross, M.D., Philadelphia Medical Times, Jan. 16th, 1871.

the *cells* of the organ or tissue. Thus they swell, and become clouded in aspect under the microscope. Next the *nucleus* divides; and afterwards the cells themselves multiply by division, or *proliferate*. The origination of pus or other cells from entirely liquid lymph, as asserted by Paget, Robin, and others,[1] Virchow denies, in accordance with his maxim "omnis cellula e cellulâ." Either the epithelial cells, or those of the connective tissue (common germ-stock of all tissues) must give rise, by change, to pus-cells. At a certain stage, cell-enlargement and proliferation become destructive of function; the parts then degenerate. But Virchow does not with any distinctness at all state the relation between this **degeneration** and that *nutritive* or *formative* **action** which he considers the one effect of "irritation;" nor does he allow to the condition of the bloodvessels much importance in what, in any tissue, he calls inflammation.

This eminent pathologist has *added* to previous knowledge that of the changes going on in the cells of an organ, a part of which is inflamed. These are important. But he omits, in his account of the process, much; and makes, on the whole, the least satisfactory theory of it now held by any authority.

To return to our account of it: an example of the three stages or processes of stimulation, irritation, and inflammation may be well studied in the action of a mustard plaster applied to the skin. Its first effect (the only one if the mustard be diluted) is **stimulant** merely; the skin grows warmer, and redder, and its sensibility is moderately heightened. Next (if it be strong and allowed to remain), **irritation** is produced; shown by *pain*, tenderness on pressure, and a deeper or more purple redness. If the irritating matter be now withdrawn, all of these may subside without going further. But if the irritation be continued up to a certain point of duration and intensity, **inflammation** occurs. Then we have redness, heat, pain, and swelling, with effusion of lymph, which, after a sinapism, or cantharidal plaster, raises the cuticle in a blister.

I express, then, what I hold to be a correct theory of the nature of the inflammatory process, in this definition: *Inflammation is a local lesion of nutrition, with concentric vascular excitement;* resulting either in *exudation* or *cell-distension* and *proliferation;* being *destructive* at the centre of the inflamed part, but often *formative* (*hyperplastic*) *around* and at some distance from it.

The observations of Cohnheim, of Berlin, have, since 1867, attracted great attention. He announced (after Addison, 1841, and Waller,[2] 1846), that, in an inflamed part, some of the colorless corpuscles of the blood (leucocytes) migrate through the walls of the capillaries, and become blood-corpuscles. Without waiting, even for the complete establishment by others of this, as a fact, some have rushed at once to the assumption of a new theory of inflammation, making it to consist *entirely* in this cell-emigration! Rashness in pathological speculation could hardly go further than this. As Stricker[1] has shown, however, Cohnheim's labors have recalled attention to the changes in the circulation during inflammation; while other sources of pus-cells must be admitted. It may be added that, in view of the known fact that pressure increases osmose, this escape of corpuscles through the walls of the capillaries must find its best explanation, partly at least, in the *increased pressure* already alluded to in our description of inflammation.[2]

[1] Hérard and Cornil (on Pulmonary Phthisis, 1867), after minute and prolonged investigation, declare that "the theory of proliferation cannot altogether take the place of that of new formation at the expense of pre-existing blastema." Paget appears to be inclined, recently, to give it up.

[2] Philosoph. Magazine, vol. xxix. pp. 271, 398.

The **products** of inflammation (by exudation) are (see *Paget's Surgical Pathology*): **1. Serum. 2. Blood. 3. Mucus. 4. Lymph.**

The inflammatory effusion of non-fibrinous **serum** is *rare*. The term is often applied, however, clinically, to a serosity which contains a small proportion of fibrin; as the effusion which follows pleurisy, etc.

Blood is exuded *occasionally* only; *e. g.*, in dysentery, in nephritis, and (dissolved) in pneumonia.

Mucus, a certain portion of which constantly moistens the surface of mucous membranes in health, is altered both in character and in amount by inflammation. The general statement is, that when a mucous membrane is inflamed (*e. g.*, in bronchitis) its secretion of mucus is *at first arrested*, then *increased*, and lastly *perverted* in character.

Coagulable **lymph** is, however, the characteristic ingredient of inflammatory exudations.

Inflammatory lymph is divided by Paget into—**1, fibrinous,** and **2, corpuscular** lymph; with the assertion that, as a general fact, the more fibrin a specimen of lymph contains (provided it be *healthy* fibrin), the greater the probability of its being organized into tissue; while the larger its proportion of *corpuscles*, the greater is the likelihood of suppuration or some other degenerative process, and the more tardy its development into any kind of tissue. (Note an *at least apparent exception to this* in the case of *diphtheritic* exudation.)

Fibrinous (coagulable or plastic) lymph is very well seen in the autopsy of any case of acute pleurisy, peritonitis, meningitis, etc. It is a whitish or yellowish-gray substance, opaque or semi-translucent after coagulation, arranged in fibrous bands, meshes, or layers, and causing adhesions between contiguous portions of the tissues affected.

Corpuscular lymph may be studied in the fluid of the vesicles of herpes, or of an ordinary blister; especially if the surface of the latter have been exposed to the air for a short time.

The lymph- or exudation-**corpuscles** which it is found (under the microscope) to contain, are about $\frac{1}{2500}$ of an inch in diameter, "round or oval, pellucid, and appearing, as if through irregularities of surface, dimly nebulous or wrinkled." Examined after a few hours under the action of water, a round and pellucid nucleus is observed within and attached to the cell-wall. It is, however,

[1] Studien aus dem Inst. für experiment. Pathol., Wien, 1869.

[2] Stricker thus states the results observed in experimental inflammation: "traumatic interference, disturbance of circulation, exudation of fluid and morphological constituents, disturbances of nutrition, new formation."

impossible, in a given instance, to make a *positive* microscopical diagnosis between these corpuscles of inflammatory lymph, and the normal lymph or chyle corpuscles, colorless corpuscles of the blood, and pus corpuscles.

The "biography" of the lymph of exudation consists in its **resorption,** or its **development** into connective, fibrous, elastic, osseous, cartilaginous, or vascular tissue, or into epithelium, etc. (rarely into muscular or nervous tissue); or its **degeneration** into *pus*, or *granule cells*, *exudation granules*, etc.

The rapid **resorption** of a moderate amount of exuded lymph, constitutes the **resolution** of an inflammation.

Its **development** is also a form of resolution, but with modification of the condition, dimensions, etc., of the part. This is, in some instances, merely restorative.

The **degeneration** of the exudation results in its being *thrown off*, as pus, or *finally absorbed*, in the form of molecular exudation-granules.

Whether immediate absorption, development, or suppurative or granular degeneration shall occur in any particular case of inflammation will depend—

1. On the state of the **blood;**
2. On the **seat** of the inflammation;
3. On the **degree** of inflammation.

(See Paget's[1] experiments as to the influence of the state of the **blood** on the lymph of vesication.)

As to the **seat** of the attack, *generally*, *serous* and *synovial* tissues (pleural, peritoneal, arachnoid, articular) are most subject to *adhesive* inflammation, *i. e.*, with the exudation of *fibrinous* lymph. *Mucous* tissues seldom exhibit this, being more prone to *suppurative* inflammation. (Exceptions in *croup*, *diphtheria*, etc.) *Parenchymatous* tissues, as those of the lungs, liver, etc., when inflamed, may suppurate, or the lymph exuded may degenerate into exudation granules, and be finally absorbed.

The **degree** of the inflammation exercises an important influence. The greater its intensity or severity (*i. e.*, the more decided and extended the *local lesion of nutrition* and the *concentric hyperæmia*), the further will the lymph exuded be removed, in its primary character, from that transuded in the natural state of the part, and the more will its subsequent changes differ from those of normal nutrition and development.

Degeneration may affect both the *fibrinous* and the *corpuscular* portions of inflammatory lymph.

The **fibrinous** part is subject to—

1. Drying into *horny concretions* (as on the valves of the heart, from endocarditis).
2. *Fatty softening.*
3. *Liquefactive* degeneration.

Both of these last contribute, no doubt, to the process of **suppuration**. Calcareous and pigmental degeneration are also described as occurring occasionally, but they are less important.

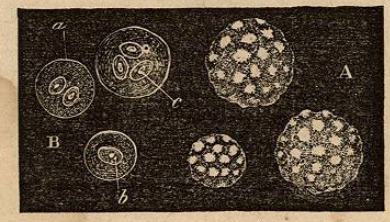
[1] Surgical Pathology, Phil. ed., p. 220.

The **corpuscular** portion of lymph may also undergo—

1. *Withering* and *drying* (as in scrofulous inflammation of glands).
2. Conversion into *granule cells* (inflammatory globules of Gluge), by *fatty degeneration.*
3. *Calcareous,*
4. *Pigmental* degeneration.
5. *Most commonly*, degeneration of the lymph-cells into **pus-cells;** the whole of the lymph being transformed into **pus.**

Pus is a greenish-yellow, creamy fluid, consisting, under the microscope, of the *liquor puris* and *pus-cells* or corpuscles. The latter are definite cell forms, larger than blood or lymph corpuscles, somewhat more irregular, and often containing several nuclei. Their characters, however, are *not invariably distinctive;* as might be anticipated, from their being merely *transformed or degenerated* lymph, blood, or epithelial corpuscles; or, in a wound or ulcer, cells of granulation. The assertion of Dr. Cohnheim,[1] of Berlin, that pus-corpuscles are white blood-cells which have emigrated through the unbroken walls of the bloodvessels, should not be finally accepted without very full confirmation. Professor Koster, of Utrecht, thinks he has obtained similar results. Prof. K. Balogh,[2] of Pesth, after great care, failed to confirm them; and the same is reported by Feltz and Picot. Lebert, Stricker, Caton, Woodward, and others find their observations to accord with them. If the fact of migration (Recklinghausen) of the colorless corpuscles be granted, it does not follow, as Stricker has shown,[3] that *all* pus-cells are of this origin. Some, most probably, are, as before taught by Paget and Virchow, of local origin. Stricker announces his discovery of the *division* of the "nomadic cells or pus-corpuscles." It has, perhaps, also, not yet been proved to be quite impossible, that what Cohnheim believes to be emigrating corpuscles, may be nuclei of the capillary walls, which, as described by Beale[4] "are at definite intervals, often on alternate sides of the tube," and, according to his opinion, "may give origin to the white corpuscles."

Fig. 8.



A. Pus corpuscles (magnified 350 diameters). B. Same made transparent with acetic acid. *a.* Cell wall. *b.* Nucleus *c.* Nucleolus. (After Lebert.)

Chemically, pus may be approximatively tested by its solubility in *liquor potassæ.*

Suppuration is either **circumscribed** (as in abscess), **diffusive** (in erysipelas), or **superficial** (in leucorrhœa, etc.)

[1] Virchow, Archiv, Band 40, s. 38.
[2] Brown-Séquard's Archives de Physiol., etc., 1869.
[3] Studien aus dem Instit. für experiment. Pathol., 1869. See, also, a paper by Dr. S. H. Chapman; Am. Journal of Med. Sciences, October, 1872.
[4] Microscope in Practical Medicine, 1867, pp. 165, 166.

The **effects** of inflammation *upon the part or organ involved* are—

Enlargement;	*Degeneration;*
Induration;	*Ulceration;*
Softening;	*Mortification.*

We thus see that very different or even *opposite* results may follow from different *degrees* or *kinds* of inflammatory action.

Specific inflammations require merely to be mentioned here. They are, chiefly, **scrofulous, erysipelatous, rheumatic, gouty, exanthematous, syphilitic, gonorrhœal.** These are distinguished from ordinary inflammation and from each other in that—1, each exhibits a peculiar *plan* of morbid process; 2, each depends upon a peculiar *cause;* 3, the effects of the said cause are irrespective of its *quantity* mostly; 4, they are especially *diffusible* from one part of the body to another; 5, they sometimes exhibit definite *stages* of the morbid process (*e. g.*, *primary* and *secondary* syphilis); 6, they are *nearly all*, in a more strict sense than other inflammations, *self-limited;* the morbid process *dying out* after a certain time. (This *last* statement applies especially, if not only, to *exanthematous*, *rheumatic*, *gouty*, and *gonorrhœal* inflammations; hardly to the scrofulous, erysipelatous, and syphilitic.) It may be questionable whether erysipelatous inflammation is a truly specific process; as reason has been shown for believing that its peculiar character may be owing to the tissue which it chiefly affects; viz., that of the vessels of the lymphatic system.

Fig. 9.

Bands of lymph in peritonitis.

The **post-mortem appearances** of inflammation are important. They can be *generalized*, so as to avoid, to a great extent, the necessity of their reiteration in connection with the description of

particular diseases. It is, at the same time, necessary for the student to *familiarize* himself with them, in their local manifestations, by availing himself of every opportunity for autopsic study.

A part which has been inflamed will exhibit after death some, or perhaps all of the following signs:—

Redness;	Coagulable lymph;
Enlargement of bloodvessels;	Pus;
	Softening;
Tumefaction;	Induration.

The **redness** of inflammation must be distinguished with care from—1, *hypostatic* injection, or cadaveric settling of blood in the lowest parts, by gravitation; and 2d, *physiological* redness, as of the stomach during digestion, the ovaries during menstruation, etc. Inflammatory redness is usually more *unequal* than either of the above, and is *stellated*, or in *streaks* and *patches*.

Enlargement of the **bloodvessels** of a part may occur as the result of a *chronic* affection, different from acute inflammation. This sign, therefore, is to be interpreted with great caution. The same is true of *tumefaction*.

Softening, if not *cadaveric* (as when the body has been long defunct), may have been produced by *chemical action*, as in poisoning by corrosive sublimate, etc., by *acute and rapid inflammation*, or by *slow*, *non-inflammatory degeneration*.

Induration may also follow either acute inflammation, or slow, atrophic degeneration.

The presence of bands or membranes of coagulable **lymph** is indisputable evidence of inflammation having occurred in the part. But it is not easy, in *all* cases, to determine with certainty whether such formations are *old* or *new*.

The existence of **pus** is a still stronger sign of the recent existence of inflammation; but, occasionally, instances occur in which pus, produced by inflammation in *one* part, is conveyed (as in phlebitis) by the veins, etc., and *deposited in another*. This, although a rare event, is *possible* at least.

Clearly, therefore, *no one* of the above post-mortem signs of inflammation is sufficient *alone*. *Several of them together* will make the diagnosis certain. *Redness* and *enlargement of bloodvessels*, with *lymph* or *pus*, and softening or slight induration of tissue, will leave little or no doubt in any case.

The variations in the appearance of different organs and portions of the body, in fatal cases of inflammatory disease, are not such as to interfere with the correctness and availability of this general description.

CHRONIC INFLAMMATION.

The term "**chronic inflammation,**" *as commonly* applied, is a *misnomer*. Although the cases so designated exhibit more or less redness, heat, swelling, and pain, yet they are wanting in *exudation;* without which, pathologically, there is *no inflammation*. There is also, in the same cases, only a *partial stasis* or *none;* and