CHAPTER XXVIII.

THE INTERNAL SECRETIONS IN THEIR RELATIONS TO PATHOGENESIS AND THERAPEUTICS (*Continued*).

THE ADRENAL SYSTEM IN THE INFECTIOUS DISEASES OF THE LUNGS (*Continued*).

While the mortality of pulmonary tuberculosis has decreased during the last forty years, that of pneumonia has steadily increased. During the year 1890 the proportion of fatal cases in 1000 deaths from all known causes, in the United States, was 90.61; during the census year 1900 the corresponding proportion was 106.1: The deaths reported in the registration area during 1890 per 100,000 of population were 186.9. In 1900 this proportion had reached 192. In 1860 the corresponding ratio was only 44; in 1870 it was 102.4, and in 1880, 125.8. During the last forty years, therefore, the mortality of pneumonia has increased almost three and one-half times. As the deaths from "consumption" for the census year 1900 aggregated 109,750, while those from pneumonia reached 105,971, the latter disease may be said to be rapidly assuming the leading position among the foes of humanity. Thomas Darlington,[1] Health Officer of New York City, states that the death-rate has risen steadily from 1.95 per 1000 in 1870, to 19.5 per 1000 in 1904, and that it now leads all other diseases as a cause of death in our country's metropolis.

Referring to this appalling mortality of pneumonia, an editorial writer[2] recently asked: "Is this dreadful waste of human life inevitable? or is it the direct result of the nihilistic teaching of authorities who are grounded in the doctrine of 'self-limited' disease, and doggedly refuse to listen to the assertions of others as acute in observation and as honest in purpose as themselves, who claim that medicine is not powerless in the

[1] Thomas Darlington: Proceedings of the Phila. County Med. Soc., Mar. 23, 1905.
[2] Editorial: Medical Record, Oct. 28, 1905.

face of this devastating disease, and that pneumonia has been, and often can be, cured without waiting for the crisis on the seventh or ninth day?"

Analyzed impartially, this doctrine has its *raison d'être* with the prevailing ignorance of the physiological action of drugs as the foundation of a so-called "rational therapeutics." As urged by Hobart A. Hare,[3] referring specifically to pneumonia: "These are cases which remind us of the extraordinary bacteriolytic power of the blood, and the remarkable methods by which nature combats disease, and which should make us hesitate before we drop into the cog-wheels of such delicate machinery, drugs which may, if wrongly given, disorder or break down this complex mechanism." Indeed, of all diseases, pneumonia is the one which would bear the least the misuse of drugs—of vasoconstrictors when the sluices bearing a stream of antitoxin to the germ-laden areas should be widely opened; of oxygen-robbing alcohol when the blood's oxygenizing power should be in every way increased; of cardiac depressants when the ventricular contractions should be sustained, etc.

The doctrine that "pneumonia is a self-limited disease" disappears with the adrenal system as a foundation for the pathogenesis and treatment of pneumonia, for it points clearly to the measures that are productive of good and affords a logical, tangible, unequivocal explanation of the beneficial effects produced. A suggestive fact asserts itself, however, in this connection, viz.: that the agents indicated are precisely those which clinical experience, untrammeled by the theory of self-limitation, has sustained—those identical remedies urged upon the profession by men who have asserted that "medicine is not powerless in the face of this devastating disease," and that it "has been and often can be cured without waiting for the crisis on the seventh or ninth day."

PNEUMONIA.

SYNONYMS.—*Lobar Pneumonia; Pneumonitis; Fibrinous Pneumonia; Croupous Pneumonia.*

Definition.—Pneumonia, an infectious disease characterized by toxæmia and inflammation of one or more pulmonary

[3] H. A. Hare: Proceedings of the Phila. County Med. Soc., Mar. 23, 1905.

lobes, is due to the multiplication in the latter of the pneumococcus lanceolatus of Fraenkel and, less usually, of the bacillus pneumoniæ of Friedländer, when from any cause the auto-antitoxin and phagocytes in the fluids of the respiratory tract, which under normal conditions destroy these germs and their toxins, are deficient in quality or quantity.**

Symptoms and Pathology.—The morbid process may involve but one lobe, or a part of it, or extend to other lobes, and even to the other lung, thus constituting in the latter case the rare form of "double pneumonia." The physical symptoms may thus be circumscribed or widely distributed.

The onset of pneumonia is occasionally preceded by headache, a slight cough, oppression and pain in the chest, and general malaise of a couple of days' duration, but as a rule it is abrupt, and is marked by a *severe chill* in adults and vomiting and convulsions in children. What fever may have been present rises rapidly, reaching 104° to 105° F. (40° to 40.5° C.) within a few hours, and remains high. The face is flushed and shows deep-red spots on the side of the affected lung, and the skin is dry and hot to the touch. The pulse is generally strong and full, varying between 100 and 120. Both the temperature and pulse are apt to be high in children. The capillaries, especially those of the surface, are flooded with blood.* In children it is the rush of blood to the skin which, by exciting the sensory end-organs therein, provokes reflex convulsions.*

The *chill* is due to a temporary depression of the functions of the vasomotor center by the toxins when the toxæmia reaches a certain limit. As this is followed by a general relaxation of all the arteries, the blood accumulates in the great central trunks, depleting the surface. The peripheral temperature being lowered, the cutaneous muscles are caused reflexly to contract and relax rapidly—the "chill." The physiological purpose of this phenomenon is to conserve heat, if possible, through enforced motion. That there is depletion of the peripheral capillaries was shown by Maragliano[4] plethysmographically, the volume of the arm being decreased during the rigor. Moreover, Geigel[5] found that the temperature fell at this time.

The connection between the reaction (the rush of blood to the periphery shown by the sudden rise of temperature) and the convulsions in children, may be illustrated by the fact, demonstrated by Poulsson,[6] that even strychnine convulsions can be prevented by anæsthetizing with cocaine a frog's skin, thus paralyzing the sensory end-organs. The convulsions are obviously reflex.

* *Author's conclusion.*
** *Author's definition.*
[4] Maragliano: Zeit. f. klin. Med., Bd. xiv., S. 309, 1888.
[5] Geigel: Allbutt's "System of Med.," "Fever," vol. i, 1905.
[6] Poulsson: Arch. f. exp. Path. u. Pharm., Bd. xxvi, S. 22, 1889.

An early symptom is a sharp pain on the affected side. This is accompanied by a short, dry cough which necessarily increases the pain (due to involvement of the pleura) and is therefore suppressed. It is often absent in the aged. Simultaneously, the respirations are increased in frequency, ultimately reaching in some cases 60 and even more a minute.

The fever remains high until the crisis, unless the subject be debilitated through alcoholism, squalor, a previous disease, etc. The fluctuations correspond very nearly with those observed during health, though exaggerated at times, especially when nearing the crisis; the nocturnal remissions averaging in most cases slightly above 1° F. When the crisis is near at hand a marked rise, 106° F. (41.1° C.) and over, may occur. In debilitated subjects the temperature range is lower; when it is very low, *i.e.*, below 101° F. (38.4° C.), the chances of recovery are greatly reduced.

The febrile process is evidently a protective one, the purpose being to increase the bactericidal and antitoxic properties of the blood.* Not only is marked leucocytosis present in cases that end in recovery, but this hyperleucocytosis is not observed in most cases that terminate fatally. It is usually absent in greatly debilitated, very young, and aged subjects, which constitute a large proportion of the mortality lists.

Leucocytosis may be low, however, in mild cases, and also in cases attended with very great intoxication. In the latter, the test-organ, overwhelmed by the toxins, is unable to enhance sufficiently general metabolism, and, therefore, the functional activity of the leucocytogenic organs.* Here, however, the febrile process is likewise low, and the depression is commensurate with the intense intoxication present. While a high fever ranging between 103° and 105° F. (39.5° and 40.5° C.), therefore, indicates a marked intoxication, it also shows that the protective functions are actively combating it.* Conversely, when in a severe case the temperature remains low, the probability is that the toxins are steadily overcoming these functions.*

Norris,[7] in a series of 500 cases treated at the Pennsylvania Hospital, found that the greatest number of recoveries occurred among those in which the temperature ranged between 103° and 105° F. (39.5° and

* *Author's conclusion.*
[7] Norris: Amer. Jour. Med. Sci., June, 1901.

40.5° C.), while the highest mortality occurred in cases in which it fluctuated between 99° and 101° F. (37.3° and 38.4° C.). Many other examples of this kind are available in literature. The influence of debility is well illustrated by the fact that while the average mortality of the 500 cases was 25 per cent., the 34 known to have been drunkards showed a mortality of 67 per cent. The protective rôle of leucocytes, not only as phagocytes, but as the source of antitoxic, *i.e.*, proteolytic ferments, has been shown.[8] That leucocytosis occurs in favorable cases—subject to the conditions outlined above—is now generally recognized. Thus Ewing,[9] thirteen years ago, concluded that in most cases of lobar pneumonia "there is a marked leucocytosis. This may be absent or inconsiderable" adds this histologist: "*(a)* in very mild cases; *(b)* in very severe cases in which the reaction of the system is slight. The degree of leucocytosis in pneumonia is proportional, on the average, to the extent of the local lesion, but it follows much more exactly the grade of systemic reaction to the poison generated." The many researches published since, including the works of Stiénon,[10] Demoor,[11] and others have confirmed these observations. Stengel's[12] conclusion that the leucocytosis is of the active polymorphonuclear variety, the actively amœboid corpuscles being increased in greater proportion than the other forms, also summarizes the teachings of more recent observations. All this is applicable to the croupous pneumonia of children. Heim[13] for example, also found an increase of leucocytes in this disease. Hypoleucocytosis invariably proved to be a serious prognostic sign, though not necessarily fatal. All the cases studied—nineteen—showed a great increase of polynuclear neutrophiles, with a relative decrease of lymphocytes.

The febrile process remains about the same from five to nine days. The cough, at first short, becomes harder. In the beginning also, ropy, viscid mucus is expectorated, owing to concomitant bronchitis, but the sputum soon becomes red or reddish-brown, "rusty" or "prune-juice" like, and may contain fibrinous coagula. If gangrenous tissue be present, it may be very fœtid. Herpes on the lips and nose are commonly observed. The urine is scanty, high-colored, and sometimes contains albumin. A characteristic feature of pneumonia is that the chlorides are reduced or absent. There is also great thirst when the fever is high. Jaundice is an early symptom in some cases. The tongue may be dry and leathery—a fact which suggests absence of alkaline salts and fluids in the blood. In simple pneumonia constipation is usual, but diarrhœa is apt to occur in the more serious cases.

During the FIRST STAGE, which lasts no longer than twenty-four hours, the air penetrates to the alveoli; *palpation* elicits a

[8] *Cf.* vol. i, pp. 610 and 671 *et seq.*, in the first edition.
[9] Ewing: N. Y. Med. Jour., Dec. 16, 1893.
[10] Stiénon: Ann. de la Soc. des sci. méd. et nat. de Bruxelles, T. iv, p. 49, 1895.
[11] Demoor: Jour. de méd. de chir., et de pharm. de Brux., July 6, 1895.
[12] Stengel: Jour. Amer. Med. Assoc., Aug. 19, 1899.
[13] Heim: Arch. de méd. des enfants, vol. iv, p. 21, 1901.

slight increase of vocal fremitus; *percussion*, if anything, a slight increase of resonance soon replaced by dullness; and *auscultation*, a broncho-vesicular murmur, soon supplanted by the typical crepitant râle at the end of each inspiration.

The pathological changes are characteristic. The affected area becomes intensely congested, and the capillaries between the air-cells or alveoli, and which course in their direction, are greatly distended. They evidently pour their contents into the alveoli, for the latter and the terminal bronchioles are more or less filled with red and white corpuscles, epithelial cells, etc., and blood-plasma. The leucocytes found in large quantities at this time are transitional cells, *i.e.*, cells which are developing into adult granular leucocytes, and which, closely examined, are found to contain lymphocytes (broken-down leucocytes), red corpuscles, nuclear detritus, and bacteria.

We are dealing evidently with phagocytic cells which are antagonizing the intruder and ridding the air-cells and terminal bronchioles of detritus. Examined post-mortem at this stage, these cavities are found to contain a bloody or reddish exudation, containing, when the lung tissue is slightly compressed, air-bubbles. It is this exudate which, when voided by way of the bronchi, gives the viscid sputum its prune-juice, rusty aspect.

Ritchie[14] in a recent presidential address, said: "I think it will be well for us to keep a mind open for the possibility that part of the increased metabolism may be the expression of work done by cells actively engaged in operating on the invading bacteria. That this is all the more likely is indicated if we correlate the known facts regarding the *increased excretion of potassium and phosphorus* during fever with the increased activity which can be microscopically observed *in the colorless cells* of the blood which contain *these elements in abundance*." The correlation between leucocytosis and the prognosis of the disease referred to above, indicates the importance of this function. The presence of transitional cells was demonstrated by several investigators. J. Pratt,[15] for example, in fifty autopsies, found that in all cases dying within the first three days "the alveoli contained large numbers of cells closely resembling the so-called transitional cells." In accord with Ehrlich's view that all leucocytes, excepting lymphocytes, are transitional cells which ultimately become granular, Pratt's text shows that the cells referred to were becoming such, since "they were surrounded by a rim of but slightly granular protoplasm," while the nucleus was "nearly as irregular as that of the polymorphonuclear leucocyte"—the typical granular cells, which ingest bacteria. Councilman,[16] in fact, observed that they were "frequently phagocytic."

[14] Ritchie: Brit. Med. Jour., Sept. 10, 1904.
[15] J. Pratt: Johns Hopkins Hosp. Reports, vol. ix, p. 265, 1900.
[16] Councilman: Jour. Boston Soc. Med. Sci., vol. iii, p. 99, 1899.

The signs of the SECOND STAGE, that of consolidation, or red hepatization, are clearly defined: The chest on the affected side hardly expands, while the other side does so with unusual vigor; abdominal breathing is also increased. *Palpation* elicits a marked vocal fremitus; *percussion*, a woody dullness posteriorly, and a clearer though dull note anteriorly and tympany over the normal adjoining areas—thus affording a means of estimating the limits of the area involved. *Auscultation* affords information as to the degree of engorgement: if the bronchi are permeable, moist râles, bronchial and tubular breathing are heard; such is not the case with choked tubes, however—another differential test, since the permeable areas can thus be located. Bronchophony, pectoriloquy, or egophony may also be discerned immediately above the hepatized area.

The consolidated area contains cells and detritus, as it did in the first stage, but now the leucocytes are found, post-mortem, merged in with a copious network of fibrin-threads. This means that during life they were surrounded by a fluid containing the three constituents—phosphorus-laden nucleo-proteid, oxygen-laden adrenoxidase and trypsin—which jointly digest as auto-antitoxin not only the bacteria, but also their toxins.* Indeed, the typical polymorphonuclear granular leucocytes and their granules are found in large numbers often within the first forty-eight hours and thereafter. Death at this time is thought to be due to excessive accumulation of all the elements enumerated, since the fibrin is found to fill the air-cells, the small bronchi, etc., but as stated above, this is in reality a post-mortem change and the dense supply of fibrin only serves to prove that every available space is filled with the protective substances.* Many of these features are clearly illustrated in the annexed plate.

As I have shown in the first volume of this work, the material found in the tissues and which causes them to be termed "fatty" is not such: it is composed of the three constituents referred to above which during life are in the liquid state—all internal secretions. The presence of a ferment in the blood-serum has been demonstrated recently by Delézenne and Pozerski,[17] and their results have been confirmed by Hedin.[18] The latter investigator states, among other facts, that "the serum of the ox contains a weak *proteolytic* enzyme, which acts in an *alkaline* medium." He closes his paper with the statement: "As to the origin

* *Author's conclusion.*

[17] Delézenne and Pozerski: C. r. de la Soc. de biol., T. lv, pp. 327, 690, 693, 1903.

[18] Hedin: Jour. of Physiol., vol. xxx, p. 195, 1903.

of the enzyme, nothing can be stated at present. Yet it should be remembered that a similar enzyme has been found in the *leucocytes* of the spleen, and it therefore does not seem to be impossible that the serum protease should be derived from the leucocytes in the blood or in other organs, either by a destructive process, which might set the enzyme free or by an act of secretion," the latter referring doubtless to secretion by the leucocytes. That the cells which secrete the antitoxic and bactericidal bodies are present in this stage is shown by the statement of Pratt that in 50 autopsies the typical-cells, the polymorphonuclears "were the predominating cells in almost all the cases dying after the third day," and that "they often appeared in large numbers within the first forty-eight hours." The experiments of Rosenow[19] showed that "the higher the leucocytosis, the fewer the number of pneumococci in the circulating blood." In 7 cases with a leucocytosis ranging from 35,000 to 43,000, "the number of pneumococci which developed was very small indeed, varying from 0 to 25 per cubic centimeter of blood." As these germs are the source of the toxins, the importance of the protective process I describe is self-evident.

When the THIRD STAGE, that of gray hepatization, progresses favorably, it becomes the stage of resolution, for the abnormal physical signs enumerated gradually disappear. The râles in the bronchi become coarse and moist; this is followed by broncho-vesicular breathing which ultimately disappears. Considerable dullness over the affected area, however, may be elicited in some cases long after recovery.

The onset of this stage in the lung is attended by a still greater influx of leucocytes and a marked decrease of the red cells and plasma. Hence the gray—and in old subjects the granite-like—appearance at this stage which contributes a large share to the mortality of the disease, owing to the interference with the respiratory process and the increased labor imposed upon the heart.

When it marks the onset of resolution, however, it is because the accumulated leucocytes embody the elements necessary for their own liquefaction, *i.e.*, their nuclein and trypsin. With the aid of the adrenoxidase in the red cells and plasma these leucocytes undergo a process of digestion, *i.e.*, conversion into a purulent liquid which is either expectorated or carried to the blood by way of the lymphatics for final conversion into products of elimination.* At this time large phagocytes are also found in the pulmonary mass of detritus, which ingest in the main the identical polymorphonuclear leucocytes which now con-

* *Author's conclusion.*

[19] Rosenow: Jour. of Infectious Dis., Mar., 1904; Jour. Amer. Med. Assoc., Mar. 18, 1905.

stitute the only source of danger. These various steps are followed by the proliferation of new alveolar and bronchial epithelial cells, and finally by the reconstruction of the disintegrated areas.

The actual presence of a substance—such as trypsin—capable of digesting not only bacteria and their toxins, but also the cellular elements themselves, is well shown by the investigations of Flexner[20] in a large number of lungs obtained at autopsy. Salkowsky having demonstrated in 1882 that ferments played a very important rôle in physiological as well as pathological conditions, Flexner found that this was applicable to pneumonia in that autolysis occurred both in the stage of red hepatization, though imperfectly, and in that of gray hepatization, in which it took place rapidly and perfectly.

The presence of the large phagocytic cells referred to is well shown by the following statement of Pratt's: "They were found in nearly every case (50). *Late* in the disease they were often present in enormous numbers." As to their contents, he writes: "Red corpuscles, lymphocytes, and plasma-cells were occasionally seen, but the most common inclusion was the *polymorphonuclear leucocyte*. Often only nuclear fragments or partially digested cells were found. They also contained bacteria."

The *crisis* may occur any time between the third day and the end of the second week, but in most cases it occurs between the fifth and the ninth day. When it is near at hand the sputum becomes purulent and more abundant, and is eliminated with comparative ease. This sign, combined with the pronounced fall of the temperature, the relative comfort, a refreshing sleep, and sometimes free perspiration which characterize the crisis, points to the latter as being the true crisis, in contradistinction to the pseudo-crisis sometimes observed. These temporary falls of temperature may occur quite early, and recur several times. As previously stated also, the true crisis is usually preceded by a more or less sudden rise of one or more degrees. The gradual fall to normal or slightly below takes from eight to twelve hours.

The crisis marks the time when the bacteria and their toxins have been overcome by the body's auto-protective elements, the phagocytic cells and the blood's antibodies. Important in this connection, however, is the accumulation of toxic wastes and worn-out leucocytes in the blood. Welch[21] found that the blood of a person convalescing from pneumonia, three or four days after the crisis, was rapidly fatal to rabbits. Rosenow[22] also concludes from his observations that "patients with excessively high leucocyte counts are apparently more prone to the development of empyema and other complications." These features are closely related to the treatment adopted, since the use of appropriate measures prevents the accumulation of these worn-out cells by facilitating their removal, destruction in the blood-stream, and elimination.

[20] Flexner: Univ. of Penna. Med. Bull., vol. xvi, p. 185, 1903.
[21] Welch: Medical Record, May 14, 1898.
[22] Rosenow: Jour. Amer. Med. Assoc., Mar. 18, 1905.

Complications.—*Pleurisy* is probably present in all cases excepting those in which the central portion of the lung is alone consolidated. It is sufficiently intense sometimes to warrant the term pleuro-pneumonia. The onset of pleurisy is attended by a rise of temperature, a sharp local pain, and the friction sound; and if empyema follow, by a marked increase of the leucocytosis and obscuration of the typical auscultatory signs of pneumonia. *Endocarditis* is observed in about 1 per cent. of the cases and aggravates the prognosis, since it occurs usually in persons suffering from some valvular disorder. The pulse is usually rapid and weak, the fever irregular, and there is considerable weakness. Rough murmurs are usually discernible. *Embolism* may occur in various parts of the body: the right ventricle and the lungs especially, the brain (causing aphasia and sometimes hemiplegia), and, rarely, in the larger arteries peripheral venous thrombosis has also been observed. *Pericarditis* is not infrequently caused through extension, it is thought, of the pleuritic inflammation. Among other possible complications are *arthritis, parotitis, and peripheral neuritis, otitis media, metastatic ophthalmia, nephritis, stomatitis, hepatitis,* and *cholecystitis.*

When empyema develops, the patient's life depends upon its early discovery and appropriate treatment. In 860 cases of pneumonia studied by Hale White and Chaning Pearce at Guy's Hospital,[23] 26 developed pyemia, *i.e.*, 3 per cent. They attach great importance to a localized painful area and to œdema of the chest-wall. The temperature falls when crises should occur, but remains around 100° F. (37.8° C.) or thereabouts, and after three or four days rises again, there being an evening exacerbation. Endocarditis is ascribed by Preble[24] to the pneumococcus in almost all cases. It is oftener on the left than on the right side, but the tricuspid and pulmonary valves are affected four times oftener than in ordinary endocarditis. Emboli occur in one-half of the cases. On the continent of Europe cardiac complications are evidently rare: von Brach[25] only found in a total of 5738 cases of pneumonia 0.2 per cent. of endocarditis and 0.5 per cent. of pericarditis.

Etiology and Pathogenesis.—The primary cause of pneumonia is a deficiency in the body at large and in the air-cells of the lungs, of the auto-protective elements, *i.e.*, phagocytic leucocytes and auto-antitoxin. This deficiency, in turn, is due either to general adynamia, or to a temporary lowering through various external agencies of the temperature of the mucous membrane of the bronchi and alveoli.*

* *Author's conclusion.*
[23] Hale White and Chaning Pearce: Lancet, Nov. 10, 1900.
[24] Preble: Amer. Jour. Med. Sci., Nov., 1904.
[25] von Brach: Cited by Schatsky: Roussky Vratch, Oct. 4, 1903.

The first of these factors, *general adynamia,* is brought on by conditions now known to impair the so-called "vitality," *i.e.,* debilitating diseases, deficiency of food, alcoholism, overwork, confinement in overpopulated quarters, such as workhouses, prisons, tenements, etc., where aëration is defective and sunlight scarce.

Cities furnish the largest proportion of deaths. In a single workhouse at Middlesborough, England, Ballard[26] witnessed 43 cases. Rodman[27] observed 118 cases in a prison containing 735 inmates. Daly[28] treated successively four brothers, then their mother. The latter having succumbed, her mother, who had come to attend the burial, also acquired the disease and died. Equally suggestive examples of the contagiousness of the disease have been observed by Mosler,[29] A. Ross Matheson,[30] Hamilton,[31] Newsholme,[32] Kühn,[33] Zimmermann,[34] and Sokoloff.[35] The latter author concludes, after an analysis of 2360 cases, that pneumonia can be transmitted from patient to patient in hospital wards. By isolating the patients and disinfecting the wards previously occupied by them, Sokoloff obtained a marked reduction both of the number of cases and of the complications in those isolated. That infection can occur from contaminated quarters was further shown by Jaworski and Chrostowski,[36] who treated five cases in a house which had not been free from pneumonia since 1860.

There is a marked predisposition to pneumonia during the first five years of life. The large proportion of deaths in children under one year old is due to artificial feeding, the infant being thus deprived of the antitoxin which the mother's milk alone affords; in cow's milk, which is, of course, used some time after being drawn, the adrenoxidase is reduced by the nucleo-proteid, and the antitoxin is thus decomposed.* A period of fifteen years is then passed during which the body is less susceptible to the pathogenic elements of this disease, but after the age of twenty years there is a steady increase of vulnerability until old age is reached, when, with increasing years, the death-rate is very large.

This is illustrated in the table given below, prepared from the last two (United States) censuses published. It gives the proportion of deaths during each census year for the five periods of life mentioned therein per thousand cases of pneumonia:—

* *Author's conclusion.*
[26] Ballard: Lancet, June 23, 1888.
[27] Rodman: Amer. Jour. Med. Sci., Jan., 1876.
[28] Daly: Lancet, Nov. 12, 1881.
[29] Mosler: Deut. med. Woch., Bd. xv, S. 245, 274, 1889.
[30] A. Ross Matheson: Brooklyn Med. Jour., Apr., 1888.
[31] Hamilton: Brit. Med. Jour., May 20, 1899.
[32] Newsholme: Practitioner, Jan., 1900.
[33] Kühn: Berl. klin. Woch., Bd. xxv, S. 337, 1888.
[34] Zimmermann: Correspondenzblatt f. schweizer Aerzte, Bd. xxii, S. 537, 1892.
[35] Sokoloff: Bolnichnaja Gazeta Botkina, No. 29, 1890.
[36] Jaworski and Chrostowski: Jour. Amer. Med. Assoc., Dec. 1, 1888.

PER 1000 CASES OF PNEUMONIA.

Census Year.	First 5 Years.	5 to 19.	20 to 39.	40 to 59.	60 and above.
1890	304.7	70.2	195.8	203.9	225.4
1900	382.6	59.1	147.6	171.5	239.2

The highest death-rate is thus shown to be borne by the two extremes of life. The influence of maternal milk on the mortality of infants is a complex subject which cannot be treated in this work. An article on this question will appear in some medical journal at an early date.

During middle life (between twenty and fifty-nine years) pneumonia occurs more frequently among males than females, owing to the greater exposure and hardships to which the former are exposed. From the fifth to the twentieth year, however, *i.e.,* during childhood and adolescence, and during old age, the opposite is the case. This is accounted for by the greater vulnerability of the weaker sex.

During the census year 1890, the excess over females during this period of life was 22 per cent., and in 1900, 20 per cent.; but in early infancy, *i.e.,* up to 5 years, the difference between sexes is insignificant. Thus in 1890 it was 0.04 per cent. in favor of the males, and in 1900, 0.03 per cent. in favor of the females. From the fifth to the twentieth year, however, which includes the periods of childhood and adolesence, a noteworthy feature asserts itself: The females preponderate during both census years: 20 per cent. during 1890 and 10 per cent. during 1900. After the sixtieth year the preponderance of females over males is striking: in 1890 it was 25 per cent. and in 1900, 39 per cent.

Traumatism of the chest, a contusion, fractured ribs, etc., may lead to pneumonia even without giving rise to a solution of continuity of the pulmonary tissue. This is due to the disturbing effect of concussion on the pulmonary cellular elements, and to the consequent weakening of the local defensive processes.*

This form of pneumonia has been termed by Litten[37] "*contusions pneumonie.*" Souques[38] studied 49 cases due to blows upon the chest without solution of continuity of the pulmonary parenchyma. He found a pleuro-pneumonia in the majority of cases, but the course of the pneumonia in all instances did not differ from that of cases usually ascribed to infection. He found pneumococci in the sputum of his cases. Mandillon,[39] Gauthier,[40] and others have found the pneumococcus in abscesses developed under such conditions. In a case, immediately following a fall upon the pavement, witnessed by Schild[41] the typical lesions of croupous pneumonia were present, though the injury of the thorax had been insufficient to be recognizable.

* *Author's conclusion.*
[37] Litten: Zeit. f. klin. Med., Bd. v, S. 26, 1882.
[38] Souques: La presse médicale, T. vii, p. 109, 1900.
[39] Mandillon: Jour. de méd. de Bordeaux, vol. xxvi, p. 260, 1896.
[40] Gauthier: Lyon médical, T. xcv, p. 329, 1900.
[41] Schild: Münch. med. Woch., Bd. xlix, S. 1569, 1902.

Exposure to cold and dry air—not damp air as is generally taught—predisposes to the disease, but only where the pathogenic organisms are present in the respiratory tract.

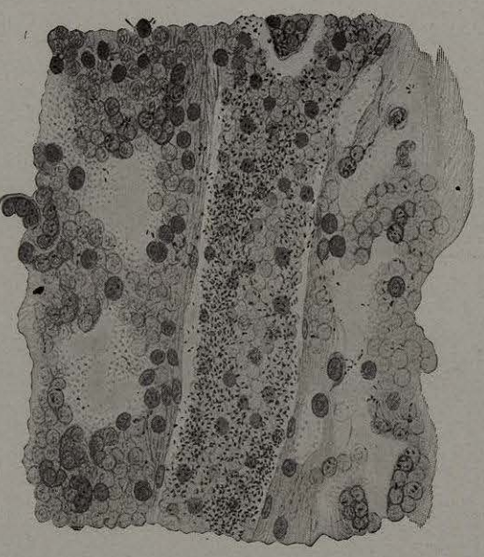
Pneumonia is less frequently met with in rural districts than in cities. It is only exceptionally met with among sailors. Sallard[42] states that it was extremely rare among Napoleon's troops during the retreat from Moscow notwithstanding the extreme hardships experienced. In large centers, Paris, for instance, similar conditions give rise to dissimilar results, for hackmen contribute largely to the yearly contingent of victims. Under such conditions exposure to cold is a prominent factor. In 79 cases recorded by Chomel, cold is incriminated 14 times, while Grisolle found this cause to prevail in 45 of 205 cases. Dampness is thought to increase the morbid effect of cold air, but H. B. Baker[43] has shown conclusively that this belief is based upon an erroneous interpretation of the actual condition of the air when it is said to be cold and damp. Cold air can hold relatively little moisture because its molecules are close together; warm air, on the other hand, can accommodate considerable since its molecules are far apart. Indeed Guyot's tables[44] based on Regnault's experiments show that air at zero F. contains per cubic foot (absolute humidity) when saturated with pure vapor, ½ grain Troy; at 32 degrees it contains 2 grains; at 70 degrees, 8 grains; at 98 degrees, 18.69 grains. It is cold, dry air, therefore, which lowers the resistance of the body to infection.

Ether-pneumonia is due to a similar condition.* The anæsthetic, owing to its rapid evaporation from the bronchi and alveoli, lowers the temperature of the broncho-alveolar epithelium, and of their contents. As ferments are activated by heat, this lowering of the temperature inhibits the activity of the proteolytic ferment in the leucocytes and the auto-antitoxin in the juices of the air-cells and bronchioles, which, under normal conditions, destroy the pathogenic bacteria and their toxins.* The germs are thus allowed to increase and to cause the disease.

This exemplifies the action of inhaled, cold, dry air as well.

General adynamia proves pathogenic in the same way, though indirectly. Starvation, squalor, fatigue, etc., entail inadequate nutrition not only of the body at large, but also of the organs that constitute the adrenal system, including the pituitary body.* As a result less adrenoxidase and trypsin are formed, and fewer leucocytes are present in the blood.* Again, the epithelial lining of the alveoli and the fluids bathing them are inadequately supplied with its protective constituents, and infection occurs if the pathogenic bacteria present are those of

* *Author's conclusion.*
[42] Sallard: "Manuel de médecine," Paris, 1896.
[43] H. B. Baker: Ann. Report of Mich. State Board of Health, 1886.
[44] Guyot: Smithsonian Meteor. and Phys. Tables, p. 39 B., 1893.



PNEUMOCOCCI AND PHAGOCYTOSIS IN LOBAR PNEUMONIA. *[Mme. N. Schultz.]*