Pulmonary consumption, pneumonia, and allied diseases of the lungs : their etiology, pathology and treatment, with a chapter on physical diagnosis / by Thomas J. Mays.

PULMONARY CONSUMPTION,

PNEUMONIA,

AND ALLIED DISEASES OF THE LUNGS;

THEIR ETIOLOGY, PATHOLOGY AND TREATMENT,
WITH A CHAPTER ON PHYSICAL DIAGNOSIS.

BY

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PREFACE.

In the present era of rapid bookmaking the author feels that no one is justified in imposing another volume on the medical public, unless he has good grounds for doing so; and this is especially true of a work, the subject of which has been so critically discussed and analyzed by many eminent writers. Then, too, it may at first sight appear startling that a book should be written, the animus of which is to show that a certain class of diseases, which have always been regarded as pure lung affections, are originated by causes lying almost entirely exterior to the respiratory organs; yet after one has devoted nearly thirty years to the conscientious study of a single line of thought it is natural that ideas and convictions should be developed and formed which are not in exact accord with orthodox teaching—and it is for the purpose of bringing these views more prominently before the medical profession that the author has decided to essay the present venture, feeling certain that it will receive thoughtful and respectful consideration from those to whom it is addressed.

The fundamental concepts of the work may be formulated into the following propositions:

1. That pulmonary phthisis in the large majority of cases is primarily a neurosis, and that the pulmonary disintegration is secondary;

2. That any agent, influence, or condition which undermines the integrity of the nervous system will engender pulmonary phthisis, or some other form of pulmonary disorder;

3. That the only remedies of value in the treatment of pulmonary phthisis are those which appeal to, and act through, the nervous system;

4. That of special value in the treatment of phthisis is the counter-irritant action of silver-nitrate introduced hypodermically over the vagi in the neck; and

5. That acute pneumonia, and other forms of acute pulmonary disease, are closely affiliated with disorder of the nervous system.

The sections on the treatment of the diseases which are included in this volume could have been swelled to much larger proportions, if a description of all the measures and agents that have been variously recommended from time to time had been incorporated; but feeling that the greatest degree of wisdom and good do not always emanate from a multitude of counsellors, the author has resolved to make a special effort to commend only those remedies that have proved useful in his own hands.

The many thanks of the author are due to Dr. J. H. Hamilton, Professor of Pathology, of Aberdeen University, Scotland, for permission to copy a number of figures from his work, "The Pathology of Bronchitis, Catarrhal Pneumonia, and Tubercle." Macmillan & Co., London, 1883.

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PHYSICAL DIAGNOSIS OF PULMONARY DISEASES.

For the sake of accuracy, the chest surface is mapped out into three general regions, the *anterior, lateral,* and *posterior* divisions. These are further subdivided as follows: The anterior into the supraclavicular, the clavicular, the infraclavicular, the mammary and the inframammary regions; the lateral into the upper and lower axillary regions; and the posterior into the supraspinous, the infraspinous, the infra and the interscapular regions. To be still more exact, we may refer to the first, second, third, fourth, etc., ribs, or intercostal spaces; to the vertebral spines, beginning at the vertebra prominens, which is the seventh cervical vertebra, and count downward; to the lower angle of the scapula, which usually overhangs the seventh rib; or to the nipple line, which is a vertical line drawn from the middle point of the clavicle downwards.

There are eight different methods which are employed in the art of diagnosticating diseases of the lungs. These are Inspection, Palpation, Mensuration, Percussion, Auscultation, Succussion, Alteration of Patient's Position, and Hypodermic exploration.

INSPECTION.

Inspection always takes precedence. It informs us concerning the shape, movements and surface-appearance of the chest.

In disease there are many departures from the standard shape—from the slightest depression or prominence of certain areas to the most pronounced deformities. Depression may be produced by pleuritic adhesions, pulmonary cavities, chronic infiltration, collapse and atrophy of the lungs; and bulging may be caused by emphysema, pleuritic and cardiac effusions.

When these abnormal conformations are very pronounced they are classified under the following names: The emphysematous or barrel-shaped chest, the paralytic chest, the asymmetrical chest, the rickety chest, the pigeon breast, the funnel-shaped chest, and the shoemaker's breast.

The *emphysematous* or *barrel-shaped* chest is almost circular in form. The ribs are generally strong, the lower intercostal spaces deepened, and the thorax is short. It is commonly associated with *emphysema, asthma,* and persistent dyspnœa of any kind.

The *paralytic* chest is the exact counterpart of the emphysematous form. It is flat and emaciated, and the ribs are prominent, the intercostal spaces are wide and deep, especially on the unaffected side. The affected side is contracted and the intercostal spaces may be narrow, and both scapulæ stand out like wings. The breathing is accelerated, and often so shallow that no thoracic movements are noticeable. This shape is also known as the phthisical thorax.

The *asymmetrical* chest implies that either side of the chest is expanded or contracted. Expansion is found in *pneumothorax,* in large *pleuritic effusions,* and sometimes in marked *functional impairment* of the opposite lung. In the pleuritic effusion of children the expansion occurs more readily than in adults, on account of the greater plasticity of the chest walls. *Circumscribed expansion* occurs in *hypertrophy of the heart,* in *pericardial effusion,* and in *isolated pleuritic effusions. Contraction* or *shrinking* is observed after *pulmonary gangrene* and *abscess,* in *phthisis,*

in persistent *contraction* of the lungs, and after recovery from chronic *pleuritic effusions* in consequence of *pleural adhesions.* A shrinkage of this kind is often so marked that the heart, mediastinum and diaphragm are displaced towards the affected side.

The *rickety* or *pigeon-breasted* chest is formed by thickening of the articulations between the cartilages and the ribs, and by a tendency to push the sternum forward. When the sternum becomes so prominent as to form the blunt apex of a cone, the base of which is formed by a line drawn through the middle of the chest transversely, the pigeon-breasted chest is developed. It is caused by any protracted interference with the free entrance of air into the upper air passages, such as *enlarged tonsils, pharyngeal* and *nasal disease, whooping cough, etc.*

The *funnel-shaped* chest consists of a pronounced retraction of the lower end of the sternum.

The *shoemaker's* breast is an acquired deformity, and resembles the funnnel-shaped chest somewhat, although it is not so deep, nor does it involve more than the ensiform cartilage. It is produced by pressing shoes against the chest while they are manufactured.

The veins of the thorax are scarcely noticeable in health except in mothers who suckle their children. In disease, however, they may not only become turgid, but be accompanied by a pronounced œdema, which is limited to the chest, arms, neck, and head. This swelling becomes enormous sometimes, and occurs when an aneurism of the ascending aorta, or some other tumor presses upon the descending vena cava, and interferes with the return flow of blood from the upper portion of the body.

There are two types of breathing-movements of the chest—the costal and the abdominal—the former being peculiar to the female, and the latter to the male sex. In *neuralgia,* or *paralysis* of the *diaphragm,* in *pleuritis* involv-

ing the *diaphragmatic portion* of the *pleura,* in *subphrenic* abscess, in *peritonitis* involving the *diaphragmatic* layer of the peritoneum, in pronounced *distension* of the *abdomen* by tumor, dropsical effusions, in the *collection* of a large amount of *gas* in the *abdominal cavity,* the abdominal breathing becomes impaired and the costal breathing is proportionately increased. On the other hand, in the *aged,* when the ribs and cartilages have lost their elasticity, and the thorax has become rigid, in *paralysis* of the *thoracic* muscles, on account of *injury* to or *disease* of the spinal nerves, the costal is displaced largely by the abdominal type of breathing.

Impaired frequency of breathing may be caused by *pressure* of tumor, or dilated blood-vessels on the brain, by *hemiplegia,* by *meningitis,* by *paralysis* of the vagi, and by poisoning with *opium, atropine, cocaine, veratrine, muscarine, digitalis, anemonin, gelsemin, physostigmin, ether, chloral,* and *chloroform.*

Increased frequency of breathing occurs in simple fever—especially in children and in nervous adults, in *pleurisy,* in *pneumonia,* in *asthma,* in *bronchitis,* and in *pulmonary* œdema and *congestion.*

Dyspnœa is generally applied to that form of difficult breathing which is caused by some obstruction in the air passages, or when there is some *interference* with the exchange of gases in the lungs, regardless of whether the breathing is accelerated or retarded. This occurs in *croup,* or *stenosis* of the *larynx,* when *foreign bodies* are lodged in the trachea or bronchi, in compression of the *trachea,* or *bronchi* by tumors, *enlarged glands* or *aneurisms;* or when the inferior *laryngeal* nerve is compressed either by aneurism or pleuritic adhesion, and paralysis of a vocal cord and narrowing of the glottis are thereby induced. Dyspnœa is also present in asthma, in feeble persons when they exert themselves, in abdominal *dropsy* and *tumors* when these

press against the diaphragm, in anæmia, leukæmia, in angina pectoris, and generally in all cardiac diseases.

Orthopnœa is that extreme form dyspnœa which compels the patient to maintain the upright or sitting posture. It occurs in pronounced interference with aeration of the blood, as in *asthma, hydrothorax, pleuritis, pericardial,* and *abdominal* effusions, and in serious affections of the cardiac valves.

Cyanosis depends on a superabundance of carbonic acid and a deficiency of oxygen in the blood, and is caused by any interference with the air entering the pulmonary air cells, or by any impairment of the efficiency of the cardio-pulmonary circulation. Thus it is seen in *œdema, tumors,* and *paralysis* of the *larynx;* in *croup;* in *obstruction* of the *trachea* or *bronchi;* in *asthma;* in *whooping-cough;* in *epilepsy;* in grave forms of *heart disease,* and in patency of the foramen ovale.

The Cheyne-Stoke's breathing consists of groups of respiratory movements, which are separated by short inter- or re-missions—each group beginning with a slight respiration, and each successive movement gradually increasing in depth until the crisis is reached, when a gradual decline takes place in these movements until the interval is reached. It is always a grave indication, and is found in aggravated cases of heart disease, especially in the dilated and fatty kind; in uræmic coma, in meningitis, in disease of the oblongata, and in poisoning by opium.

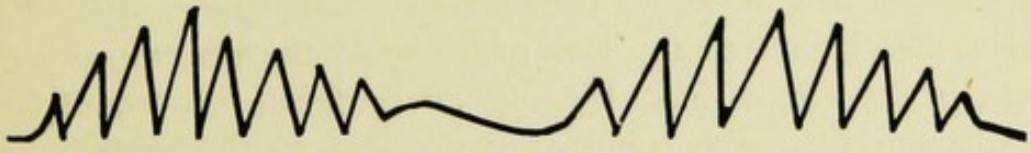

Fig. 1. Tracing of Cheyne-Stoke's breathing curve. The up strokes represent inspiration and the down strokes expiration.

Palpation.

This is the art of examining the chest through the sense of touch or feeling. By it we are often enabled to detect vibrations which are produced by friction, sonorous or sibilant râles, and sometimes by cardiac and aneurismal murmurs. By increasing the resistance to the sense of touch, areas of consolidation can be differentiated from normal lung tissue through palpation. By touch we may determine areas of soreness, fracture and caries of the ribs, prominences of the costo-cartilaginous articulations, count the ribs and spinal prominences, feel the depth and width of the intercostal spaces, detect fluctuations in the pleural and pericardial sacs, and locate the apex beat of the heart.

Vocal fremitus is the art of feeling symmetrical spots with the finger tips of both hands while the patient is counting loud *one, two, three* at the same time. If the vocal vibrations are felt more distinctly on one than on the other side it is probable that the lung tissue is in a state of consolidation at the former spot. This needs confirmation by other physical signs. If the vocal vibrations are absent a pleuritic effusion probably exists.

Mensuration.

This is the method of measuring the dimensions and the degree of expansion of the chest. The circumference of the chest is found by placing a tape-line around it, over the nipples. The length of the chest is ascertained by measuring from the clavicle to the lower border of the ribs on either side, in the nipple line. Its semi-circumference is found by measuring from the spine of a vertebra to the middle of the sternum, first on one and then on the other side. To find its contour lay a leaden hoop, or wire, around it, fitting it carefully to each depression and elevation, and then care-

fully remove it and trace its shape on paper. In making these measurements it must be borne in mind that the right side of the chest—especially in right-handed people—is about half an inch larger than the left side. This method indicates the presence of a pleural exudation, as well as an increase or decrease of the same, that of a bulging which may be produced by a tumor or aneurism, or that of depression brought about by retraction of a lung.

Percussion.

Percussion is the method of eliciting sounds from the chest by tapping or gently striking it. When the thorax is percussed directly, *i. e.*, with nothing intervening, it is called *direct* percussion. At present this is only employed in percussing the clavicular or scapular regions. When some materials like the finger, hard rubber, or ivory, which are called pleximeters, are laid on the chest and are percussed it is called *indirect* percussion. The instruments which are employed in striking are the fingers and a hammer, which are called *plessors*. The fingers are always to be preferred, for the reasons that they are always present, they can be readily adjusted to the intercostal spaces, they have a most delicate touch, and are able to perceive the resistance which may be present in the chest.

Percussion is a process of comparing sounds which are obtained from opposite and symmetrical spots of the chest, and hence the relationship between the patient's body and the percussor's ear must be disturbed as little as possible. It is important, therefore, that the percussor should stand directly in front of, or behind, the patient, as the case may be, and must not step from one side to the other as he percusses around the body. Only one finger of the left hand, and preferably the second, with the other fingers raised, should be firmly laid against the chest wall; and in such a direc-

tion that the distal end points outward, and the central end towards the sternum, if the front is percussed, and the latter end towards the spine if the back is percussed. As a plexor, the second finger of the right hand should be used. This should have its nail well trimmed, and it should be bent at a right angle at the second joint. The blows should be delivered gently but quickly, from the wrist, and always perpendicularly to the chest wall. The patient must not stand against the wall or lean against any object. His arms must not be folded, but hang loosely by the patient's side, with a slight forward inclination.

There are three typical sounds which proceed from the chest when it is percussed, viz., clearness or resonance, flatness and tympany—the first of which belongs to health, and the two last to disease. Examples of the first may be obtained by percussing over the healthy lungs, of the second by percussing over the head of the humerus, and of the third by percussing over the cheek when it is made tense by separating the jaws. All these sounds have the properties of pitch and intensity, or, in other words, they may be high or low, and loud or soft. Low-pitched sounds possess great intensity, while high-pitched sounds have little intensity.

Pitch and intensity of chest-percussion sounds vary with the density and volume of tissue which is thrown into vibration: the larger this volume is the lower the pitch and the greater the intensity, and the smaller and denser this is the higher the pitch and the less the intensity. For this reason dense bodily tissues like bone, liver, spleen, etc., give rise to high pitched, short sounds; and porous tissues like healthy lungs emit low-pitched sounds of long duration when percussed. All diseases of the lungs, like pneumonia, for example, which are characterized by a deposit in the air cells, are accompanied by a percussion-sound, the pitch of which is higher than normal. It is evident, however, that the degree of the elevated pitch depends on the degree of de-

posit, or infiltration, *i. e.*, a slight degree of infiltration produces a slight elevation, while intense or excessive consolidation calls forth a sound the pitch of which approaches that which is obtained by percussing over a bone, as the head of the humerus, for example, and which is called a *flat* sound. Between a slight departure from a normal percussion pitch and flatness we have, therefore, a number of shades of dulness, which may be classified as follows: (1) impaired percussion resonance, when the infiltration is slight; (2) dulness, when the infiltration is more marked; and (3) flatness when the infiltration is complete. The diseases with which these varieties of percussion sounds are associated are *croupous* and *catarrhal pneumonia, infiltration of phthisis, miliary tuberculosis, pulmonary œdema, pleuritic effusion, hydrothorax, thickening of the pleura, tumors, aneurisms, etc.*

A tympanitic percussion sound emanates from a cavity—the cavity being an excavation of lung tissue, or an enlarged and distended bronchial tube, or an accumulation of air in the pleural cavity. It also has pitch and intensity—both of which being determined by the size of the cavity and the tension of its walls. The larger the cavity and the lower the tension of its walls, the deeper the pitch of its sound, and vice versa.

When a cavity is surrounded by a thick layer of consolidated tissue it gives rise to a composite sound when percussed, which is called a *dull-tympanitic* sound. The *cracked-pot* sound or the bruit-de-pot-fêlé is a variety of tympanitic sound, and is in nature analogous to a sound which is derived from striking an empty vessel, the walls of which are cracked. It has somewhat of a metallic ring but also partakes of the nature of a short, high-pitched thud. It is generated in a cavity which has communication with the outside air, and which has sufficient firmness in its walls to deaden the vibration of its contained air. In order

to bring out this sound well the percussion blow should be strong, quick, and concentrated, while the mouth of the patient is wide open. It is heard over *lung cavities,* in *pneumothorax* with an external opening, above large *pleuritic exudations* at the point where the lung is retracted, and, as is taught by some authorities, over *pneumonic consolidation.* The metallic echo may be brought out over a large cavity, or over a pneumothorax, by tapping one silver coin on another over the affected area, either in front or on the back, while the auscultator listens opposite.

Auscultation.

Auscultation is the art of listening for sounds which are produced in the body. Those of the chest are of three kinds: first, those produced by the loud and whispering voice; second, those by breathing; and third, those by râles and friction.

When we place the ear directly over the skin and listen we practice *immediate* auscultation; and when we interpose an apparatus between the ear and the skin we practice *mediate* auscultation. For adaptability, cleanliness, and ability to concentrate effort, the latter method is preferable, although every auscultator should familiarize himself with both methods of observation. Both the single and double stethoscopes are employed in mediate auscultation—the single hard rubber instrument being preferred by the author. Of the double stethoscopes, that devised by Dr. Camman of New York, is probably the most useful.

In auscultating observe the following rules: Listen over the bare skin. Do not press the stethoscope too firmly against the chest, nor allow clothing, or the fingers to rub on the instrument while listening. Always compare the signs of one side with those found on the exact spot of the opposite side, and listen during both ordinary and forced

breathing. Remember that a hairy chest gives rise to crackling sounds under the stethoscope.

Vocal resonance is the mode of listening to the sounds which are produced in the lungs by the vibrations of the spoken voice. In health these sounds are vibrated throughout the trachea, bronchial tubes and lungs. Over the trachea, it is called the *tracheal sound;* over the bronchial tubes, or in the suprasternal region, the *bronchial voice,* or *bronchophony,* and over the pulmonary or vesicular tissue, *normal vesicular resonance.* While the voice sounds are reflected over the whole chest in health, they are best heard where the bronchial tubes are most superficial, and are almost entirely absent, or at least very much subdued, where the tubes are thickly covered with vesicular tissue. The reason for this is that air is a bad conductor of sound, and that the facility with which the latter is conducted depends entirely on how much or how little air intervenes between the bronchial tubes and the stethoscope. Hence, when the vesicular tissue becomes infiltrated, as in pneumonia, the vocal resonance becomes more perceptible, while if the same tissue becomes rarified, as in emphysema, it is scarcely heard at all. Vocal resonance, like palpation and percussion, is, therefore, largely dependent on the specific density of the pulmonary tissue. If this is moderate, as in slight consolidation, we say that the vocal resonance is increased; and if complete condensation exists, then the voice sound is no longer known as such, but goes under the name of bronchophony, all the air in the lung tissue being now displaced. When the patient whispers *one, two, three,* the sound which is produced is called whispering bronchophony, the indications of which are similar to those of increased vocal resonance and bronchophony.

Whispering pectoriloquy may be regarded as a variety of bronchophony, with the difference that in pectoriloquy the articulation of the whispered words are distinctly perceived,

while in bronchophony the sounds of the words are only heard. The existence of whispering pectoriloquy is generally regarded as pathognomonic of a cavity.

Since the air in the bronchial tubes conducts the voice-sounds throughout the lungs, it is obvious that impairment or suppression of vocal resonance may occur in one of two ways: (1) through the *plugging of a bronchial tube with mucus,* or *catarrhal material,* or through *compression* of the same by an *aneurism,* or *tumor* of *another kind;* or (2), when a lung, or a part of a lung, *has undergone complete solidification,* and is no longer capable of admitting or receiving air. Independent of the lungs this condition may also be brought about by *large pleural effusions, empyema, pneumothorax* and *pyopneumothorax.* The air, or fluid which insinuates itself between the lungs and the chest walls under these circumstances compresses the lungs, forces them to occupy a smaller space, and prevents the voice sounds from reaching the surface of the chest.

There are at least four different varieties of breathing sounds in health, viz., the tracheal, over the trachea; the bronchial, over the large bronchial tubes; the vesicular, over the air cells; and the broncho-vesicular, a mixture of sounds coming from the two last. The tracheal and bronchial breathing sounds are of a smoothly blowing character, both during inspiration and expiration, with the pitch of expiration slightly higher than that of inspiration. The vesicular sound is soft and breezy, while the broncho-vesicular sound is a mixture of the former and the latter.

Although no bronchial breathing sounds are heard in health over the chest where the bronchi are covered with vesicular tissue, it is quite certain that they are generated there, but fail to be propagated to the surface on account of the intervention of the air-containing tissue, which is a poor conductor of sound. Hence, in pulmonary infiltration, where the air in the cells is displaced by inflammatory

products—the conductivity of the lung tissue is enhanced and the sounds which are produced in the bronchi are transmitted to the surface of the chest and are heard there. The knowledge that conductivity is increased under these circumstances is of great importance to the diagnostician, for by hearing the bronchial breathing sounds over areas where they are not perceptible in health we know that the pulmonary tissue is infiltrated. Different degrees of infiltration, of course, produce different effects; for if everything else is equal, the expiratory sound being higher in pitch, is heard before the inspiratory sound. Hence, in slight consolidation, the expiratory and not the inspiratory sound is heard. This is called the *prolonged expiration*—although there is really no prolongation of the expiratory sound. In more marked consolidation both the inspiratory and the expiratory sounds are audible, but the pitch of the latter is always higher than that of the former. At this stage of consolidation the non-conducting property of healthy lung tissue is so thoroughly abolished that the sounds which are produced in a bronchial tube are heard as readily on the surface of the chest as if the stethoscope were resting directly on the tube. *Prolonged expiration* is heard in the beginning of *phthisis*, and most usually at the apices. *Bronchial respiration* is heard in *pneumonia*, is often the first audible sign in this disease, in *phthisis, pulmonary œdema, pulmonary apoplexy*, and sometimes in *effusion of the pleural cavity* at or slightly above the level of the effusion.

Roughened breathing occurs when there is increased friction between the surfaces of the bronchial tubes and the air which moves over them, as is found in the first, or *dry stage* of *acute bronchitis*, or in *chronic bronchitis*, where the bronchial surfaces are rough and uneven in consequence of persistent inflammatory action. This form of breathing must be distinguished from the smooth tubular blowing of prolonged expiration, and of bronchial respiration.

Like vocal fremitus and vocal resonance, the vesicular murmur may be exaggerated, or diminished, or even suppressed in disease. When it is increased in the adult it is called *puerile breathing*, because it resembles the vesicular respiration sound in children. In the adult, puerile breathing is compensatory in character, is indicative of greater activity in the part where it is heard, and is a sign of impairment in function of some other part of the respiratory organs, as is seen in *pneumonia, pleurisy, pleuritic effusion,* compression of one lung, or of a bronchus by a tumor, etc. Vesicular breathing may become impaired by the impaction of a foreign body in one of the air passages, obstructing the entrance of air into a lung, by effusion into a pleural cavity, by defective respiratory movements due to faulty habits, or may be due to impaired pulmonary innervation, to debility, to old age, to paralysis, etc. It is always necessary to compare the respiratory murmur of one lung with that of the other and to note any difference between them.

There is a kind of breathing called the jerking, interrupted, wavy, or cog-wheel respiration, which consists of an intermitting inspiration. It is rarely heard during expiration. It may be due to *want* of persistent *expansion* of the lung, as in the case of pleurisy, or intercostal neuralgia, when the patient dreads the pain which comes with full respiration, or it may be caused by some *obstruction* in a bronchus to the free entrance of air into the lung. When it is not of a purely nervous character, as seen in nervous or hysterical persons, and is not occasioned by pain, and is located in an apex it may be regarded as a *forerunner*, or a *concomitant of phthisis*, or at least of circumscribed bronchitis.

Cavity respiration consists of a blowing inspiration and expiration and has, therefore, some of the characteristics of bronchial respiration, but according to Flint, differs from the latter by reason of the pitch of inspiration being higher than

that of expiration. This is partly true, for in many cavities expiration is practically absent, but it is by no means universal. Then, again, not very infrequently, the relative pitch of inspiration and expiration coming from a cavity differ nowise from that of bronchial respiration. It seems that the expiratory pitch of a cavity depends very much on the manner of the connection between the cavity and the bronchial tube, which is its outlet. If this is valve-like and offers more resistance to the air coming out of than to the air going into the cavity, then the expiratory pitch will be higher, otherwise the reverse obtains. But a cavity respiration has other qualities which distinguish it from the bronchial respiration, for it is usually *tympanitic, hollow,* or *amphoric* in character—the *amphoric* sound representing that which is produced by blowing across the mouth of a bottle. If the cavity contains fluid, hollow, gurgling sounds are heard.

Râles and Friction Sounds.

Under these headings are included all the new sounds which are produced in the lungs and between the pleural surfaces. They are also called adventitious sounds, because they are found nowhere in health. The râles are produced in the bronchial tubes and in the air-cells. Those râles which are produced in the *large bronchial* tubes are called *large mucous* râles, those produced in the *smaller bronchial tubes* are called *small mucous râles,* and those produced in the bronchioles, or the smallest bronchial tubes, are called *sub-crepitant râles;* whilst those which are produced in the *alveoli* are named *crepitant* râles. It must be remembered that the former râles are produced when the collection in the bronchial tubes is liquid or fluid, but when this accumulation is dry, or tough, especially in the bronchioles, there are produced râles of a

musical character which, according to their pitch, are either called *sibilant* or *sonorous râles*. The former are of a high-pitched, whistling character, while the latter are low and deep in sound.

So much, then, for the râles which are produced in the tubes. When the catarrhal process extends from the bronchi into the air vesicles the nature of the râles change entirely. In the bronchi they are either bubbling noises (mucous râles), or musical sounds (sibilant, or sonorous râles), and are heard during both inspiration and expiration; while those which are heard in the air cells are crackling or crepitant in character, and are heard only during inspiration. Moreover, the former are never accompanied by dulness, while the latter nearly always are.

Occasionally, before catarrh of the bronchi diffuses itself into the air cells, the smallest bronchial tubes, or bronchioles, may be almost exclusively involved. The vesicular walls become glutinous, and adhere together, and during inspiration the air separates them and this rupture produces one or more *clicking râles* at or towards the end of inspiration, This click is often the only abnormal sign to be discovered, and if it is heard in one apex only, as is usually the case, it is the forerunner of serious pulmonary changes, since it usually marks the beginning of phthisis.

At the point of communication between a bronchus and a large cavity there is occasionally produced a click or sound which has a metallic ring. The click is caused by the bursting of a film of fluid formed over the entrance of a bronchus into a cavity, and it receives its hollow metallic quality from the reflection of its sound into the cavity. It is heard only during inspiration, and since fluid is essential to its production it is quite evident that if the tube becomes dry, or if the film is removed by coughing, or by forced inspiration, the sound disappears.

When a large cavity lies in close proximity to the heart

the impaction of the cardiac impulse against the wall of the cavity sometimes produces a metallic tinkle with each cardiac pulsation. This sound may be imitated by letting a drop of water fall into a large empty bottle, from a dropper or from a pipette.

Pleural Friction Sounds.

During health the pleural surfaces are moist and smooth, and move noiselessly one on the other during respiration, but in disease they become dry and rough, and their surfaces rub on each other and give rise to a scraping or friction, or rubbing sound. Occasionally this is intense enough to set up vibrations in the chest wall, which can easily be felt by the hand. The sound is yielded to best advantage at the base and sides of the chest, where the respiratory movements are most pronounced. It is best heard when auscultating directly over the affected area, and if the disease exists in circumscribed spots, as it frequently does, it may be overlooked if this fact is not borne in mind. So soon as pleuritic exudation occurs this sound disappears, but reappears when absorption of the fluid occurs. When the pleural covering of a lung-cavity becomes thick and indurated it gives rise to creaking sounds during inspiration and expiration, which may be imitated by bending stiff sole-leather on itself. Besides the occurrence of these sounds in pleurisy, they also accompany *phthisis, pulmonary infarction* and *bronchiectasy.*

Succussion.

When air and fluid—either pus or serum—collect in the pleural cavity, as in pyopneumothorax, or hydropneumothorax, and the patient shakes his thorax from side to side, or is suddenly shaken, a peculiar tympanitic splashing sound is

produced, which is called Hippocratic succussion, because it was first described by Hippocrates. It is loud enough sometimes to be audible some distance from the patient. Care must be exercised so as not to confound with this sound the splashing which may be produced in the stomach or colon.

Change of Patient's Position.

A change of the patient's position is frequently an important aid in diagnosticating disease of the lungs. In suspected pleural effusion the level of the supposed fluid is ascertained by percussion while the patient is standing, or sitting, and the line of absolute dulness is marked with ink. The next step is to find out whether this line changes if the patient is placed on his back. If fluid is present in the pleural cavity, and if this is not circumscribed, the line of dulness will sink, or disappear altogether from the front of the chest, if the effusion is moderate. Then, again, the upper border of dulness should be ascertained in the back, and then it is to be found out whether this border shifts its position when the patient lies on his face. Moreover, pleural friction sounds also become more audible when the patient leans toward the auscultator, and allows the lung to gravitate more closely to the chest wall.

Exploration of the Pleural Cavity.

In any doubt as to the existence of pleural effusion it is always advisable to introduce a well disinfected hypodermic needle into the cavity. The needle should be about two inches long, and enter the cavity in a perpendicular direction to the chest wall, and in an intercostal space, where the fluid is supposed to be located. As a rule the puncture should not be made higher in front than the fourth intercostal space, nor higher than the sixth in the back. Care must be observed in avoiding the region of the heart.

A TABULAR VIEW OF WHAT IS TO BE LEARNED FROM THE SOUNDS WHICH ARE HEARD IN THE LUNGS, AND OF THE DISEASES WHICH THEY INDICATE.

1. VOICE SOUNDS.	CONDITIONS INDICATED.
1. Increased vocal resonance...	Condensation of lung tissue.
2. Bronchophony.....	
3. Whispering bronchophony ..	
4. Whispering pectoriloquy	Excavation.
5. Diminished or suppressed vocal resonance.....	Plugging, or compression of bronchial tube, or pleural effusion.

2. BREATHING SOUNDS.	CONDITIONS INDICATED.
1. Puerile breathing.....	Compensatory in adults.
2. Roughened breathing.....	Bronchitis.
3. Prolonged expiration.....	Various degrees of lung condensation.
4. Bronchial respiration.....	
5. Cavity respiration.....	Excavation.
6. Amphoric respiration.....	
7. Wavy respiration.....	Intercostal neuralgia. Nervous breathing. Phthisis.

3. RÂLES.	CONDITIONS INDICATED.
Large mucous râles.....	Affections of the bronchial tubes—bronchitis, asthma, etc.
Small mucous râles.....	
Subcrepitant râles.....	
Sibilant râles.....	
Sonorous râles.....	
Crepitant râles.....	Condensation of lung tissue—pneumonia.
Clicking sound.....	Incipient phthisis.
Metallic tinkling.....	Excavation.
Cardiac metallic tinkling	
Friction sounds.....	Pleurisy.
Succussion.....	Hydro- or pyopneumothorax.

Method of Recording Pulmonary Diseases.

For the purpose of making an intelligent and profitable study of diseases of the lungs it is absolutely indispensable that every case that comes under observation should not only be thoroughly investigated and studied, but that all the data pertaining to it should be methodically recorded in a case-book, and in the following order:

1. *Preliminary:* Number, date, name, address, age, occupation, married or single, nativity, number of children.

2. *Family History:* Father alive? Age? If dead, age and cause of death?

Mother alive? Age? If dead, age and cause of death?

No. of brothers alive? No. of brothers dead? Age and cause of death?

No. of sisters alive? No. of sisters dead? Age and cause of death?

Order of patient's birth? Ages of preceding and succeeding member?

Order of birth of sisters and brothers dead, or suffering from any pulmonary disease. Ages of father and mother at marriage? Any of grandparents, uncles, aunts, sisters or brothers suffering, or dead, from phthisis, asthma, insanity, epilepsy, alcoholism, gout, rheumatism, periodical headaches?

Personal History: Physiognomy? Complexion? Pitch of voice? Pulse? Respiration? Temperature? Weight? Stature? Circumference of chest during deep inspiration? Expiration? Length of spinal column from seventh cervical to end of spine? Duration of cough? Duration and quantity of expectoration? Color of latter? Presence or absence of tubercle bacilli? Pain in chest? Hemoptysis? Dyspnœa? If present, is it paroxysmal? Most likely to appear by day or night? Œdema? Loss of flesh? Chills? Fever? Night-sweats? Appetite? Condition of Tongue? Vomiting? Bowels? Menses? Cardiac pain or palpitation? Headache? Dizziness? Nervousness? Pain or twitching in arms or legs? Spinal pain? Neuralgia? Malaria? Rheumatism? Gout? Syphilis? Alcoholism?

Physical Examination:

Inspection.

Palpation.

Mensuration.

Percussion.

Auscultation.

The space of the case-book, whether one, two, or three pages, that is devoted to the history of each case, should contain at least two diagrammatic cuts showing the anterior and posterior aspects of the chest, on which may be inscribed the following characters, indicating the physical conditions, and which are copied from West. *How to examine the chest:*

(*a*) (*b*) (*c*) (*d*) (*e*) (*f*)

Fig. 2. Characters used to indicate the physical conditions as found on examination of the chest.

Crepitation, small dots (*a*).
Mucous râles, small circles (*b*).
Cavity, large circle (*c*).
Pulmonary dulness, perpendicular lines (*d*).
Pleural-effusion dulness, horizontal lines (*e*).
Pleuritic friction sounds, zigzag lines (*f*).
I.=inspiration.
E.=expiration.
R.=respiration.
E+.=prolonged expiration.
R+.=exaggerated respiration.
R—.=deficient respiration.
R.C.=cavernous respiration.
Rh.=rhonchus.
Sb.=sibilant râle.
Pect.=pectoriloquy.

CHAPTER II.

ETIOLOGY OF PULMONARY CONSUMPTION.

DEFINITION: A chronic wasting disease of the whole body, in which the lungs are actively involved.

SYNONYMS: Phthisis pulmonalis, Pulmonary Tuberculosis, Consumption, Decline, Tabes Pulmonalis, Lungenschwindsucht, Phthisie Pulmonaire, etc.

GENERAL CONSIDERATIONS: It may be stated as a truism that, according to current medical usage, much is denominated disease which is really only an effect of the same, and that many coexistent organic disturbances in the same body are looked upon as separate and independent disorders, which, on investigation, are found to form a group of superficial manifestations, and which owe their origin to a common underlying cause. This is no more nor less true in medicine, however, than it is in other lines of thought. The ordinary observer, in watching the fury of a storm at sea, attributes the final cause of commotion in the water to the action of the wind, and is unmindful that the wind and waves are mere surface indications of the imperative influence of the sun's heat, which gives life and being to every storm by sea or land. To him the wind is the ultimate cause of the disorder, and the waves, floods, wrecks, and changes in the coast-outline, are looked upon as its legitimate effect.

That which applies to diseases in general is specially true of diseases of the lungs in this respect, and particularly of pulmonary consumption. This disease, whether tubercular or not, is usually regarded as a local disease of the lungs, yet it is well known

that the larynx, stomach, heart, kidneys and other important organs are either functionally or organically involved at the same time. We have here, then, a number of co-existing diseases, which, like the wind, waves, floods, wreckage, and transformation in the coast-line, may be regarded as independent disorders, but are really the superficial effects of a deeper-rooted cause.

If pulmonary consumption is truly a superficial manifestation of some deeper-lying disorder, what is the nature of the latter? Where is its seat? What are the forces which bring it about and perpetuate it? And what is the link that binds it to the disturbances and conditions which accompany it? In order to make this clear it is necessary to briefly consider the development of the nervous system in so far as it stands related to this particular subject. In the lowest form of animal life there is no special nerve-tissue, although sensation, motion, digestion, circulation, respiration, secretion, reproduction, etc., are all carried on in the undifferentiated organism. But as life advances to a higher plane, the simple undifferentiated organism develops into a body of many separate organs, and, as is well known, it is for the purpose of co-ordinating the function of these organs with each other, and, with the body as a whole, that the nervous system and brain arise simultaneously.

The connection between the brain and nervous system and the other organs of the body may be compared to that which obtains between the officers and the privates of an army of soldiers. The former formulate and transmit orders while the latter execute them. Each lower officer, like a lower nerve center, although he may have a company of privates under his command, is, in turn, commanded by a higher officer, and he by a still higher one, and so on until finally the general and highest officer is reached, who, like the brain and the higher nerve centers, holds supreme command. Now in primitive warfare, military organization is

crude and imperfect, same as is physiologic organization in the lower stage of animal life. Here each individual soldier exercises his own choice more or less in the discharge of what he believes to be his duty. No strict line of demarcation is drawn between those who command and those who are commanded. Everything is in a comparatively homogeneous condition. On the other hand, in the modern army, where drill and discipline are forced to the highest state of efficiency, where the only duty is to obey, the individual-will of the private soldier sinks into the will of the higher commander, and hence a well organized army becomes a colossal animal which knows no other governing power than that of its commanding general, whose range of wisdom, intelligence and integrity decide not only the fate of a battle, but the destiny of nations.

The brain and nervous system, therefore, occupy the distinction of being the great centralizing and controlling agencies of the body, and in these pages the proposition will be maintained that serious impairment of the integrity of the central nervous system will eventually be followed by disease in many of the most important and vital organs of the body, and in which disorder of the lungs plays a very significant rôle.

Etiology.

Now the conditions and influences which produce pulmonary consumption may be divided into three classes: (1) those which vitiate the nervous system in general, and the pulmonary nerve supply in particular; (2) those which irritate the lungs directly through the inhalation of dusty air; and (3) those which act through infection.

In considering these different causes it is taken for granted that no sharp and insurmountable barrier exists between most of the diseases that invade the pulmonary organs, that various forms obtain at the same time, and that

one form is frequently transformed into another. That which is œdema at one time may gradually become bronchitis at another, bronchitis may evolve into catarrhal pneumonia, and the latter may develop into pulmonary phthisis.

Influences of the First Class.

That pulmonary phthisis and many other morbid manifestations of the lungs are due to disordered and diseased pulmonary innervation is by no means a theory which is now advocated for the first time. It has been observed long ago that diseases of the nervous system bear a causative relation to pulmonary consumption, and as far back as 1842 Cheneau* attributed this disease directly to disorder of the pneumogastric nerves. In 1850 Dr. J. C. Holland defined pulmonary consumption as a disordered condition of the nervous system† Dr. Copland regarded tuberculosis and scrofula as being dependent on abnormal conditions of the nervous system, and that the accompanying disturbances of digestion, of assimilation, of the circulation, and even the local determination of these diseases are traceable to the state of the nervous influence of these parts‡. Laycock in his "Clinical Lectures on Physiological Diagnosis of Disease"§ says "defective pneumogastric innervation consequent upon a loss of cerebrospinal power is a very common predisposing and exciting cause of phthisis." Dr. Clifford Albutt,‖ under the heading of "Phthisis as a Neurosis," discusses a variety of phthisis which, he holds, does not agree

* "De l'Influence de la huitième paire dans la production de la Phthisie." Paris, 1842.

† "On the Nature and Cure of Consumption," cited from Ancell's "Treatise on Tuberculosis," page 556.

‡ "Dictionary Practice of Medicine," part xv, p. 750.

§ *Medical Times and Gazette*, 1862, p. 205.

‖ *Medical Times and Gazette*, 1871, vol. ii, p. 613.

with the general meaning of the term "phthisis." He* says that individuals who belong to this type of the disease come from the neurotic class, and cites the following examples to bear out the correctness of his observations:

"About a year ago I attended a refined lady, *belonging to a highly neurotic family,* and whose children presented like characters. She had been a nervous, irritable, neuralgic woman all her life, but never actually ill in any serious way. She was worn down by nursing one who was very dear to her, and whose death, which followed, shocked and prostrated her still more. She took to her bed with consolidation of the apex of the right lung, then of the left; in both the mischief spread rapidly, hectic fever ran high, and in three weeks she was dead. Her age was forty-three. On the autopsy which was made in consequence of her death appearing to her family to be very sudden, we could not find a single tubercle in the body, but the apices of the lungs were almost destroyed, and less degrees of mischief were found below."

"I had seen another case, a few years before, in which such a galloping consumption had occurred after nervous depression, in a highly nervous subject. The son of nervous parents on both sides, the father being odd and eccentric, and the mother actually insane, he was himself one of those heady, impassionable, gay fellows who make our charming prodigals—and a very prodigal he was. Ruined in purse and character, and terribly depressed, though I do not think in any great degree worn out by actual vice, he arrived at home to meet with the reception such parents (or, at any rate, one of them) were likely to give him. A few days later he began to feel more and more exhausted; his pulse and temperature ran up; his right lung solidified and broke down; the left lung followed; and after five weeks of dis-

* See also by same author, "Neurosis of the Viscera." *Lancet,* 1884, vol. i, p. 604.

tressing illness he was dead. This case was the first which impressed me strongly with its probable neurotic origin. . . . Having, then, cases of phthisis presenting the exact features already described, and having also many cases of catarrhal bronchopneumonic phthisis, and, more strictly, tubercular phthisis, presenting like character intercurrently with others of their own, I felt that neither the catarrhal nor the tubercular theory accounted for all instances. . . . until I began to find in my own practice, and in the writings of alienists, how large a part phthisis plays in neurotic families. Even, then, however, it did not occur to me to associate any particular form of phthisis with neurotic disorder until a few striking cases of my unclassable variety occurred in neurotic families under circumstances which spoke too eloquently to be overlooked. . . . If we try to go a step further, and ask for a pathologic explanation of these facts, we approach a land of darkness. The more, however, I study the relations of the disease, the more I am satisfied that the lung mischief is also a neurosis—by which I mean that the lesion is one not originating in the local tissues, but in the nervous system."

The author's own belief in the neurotic character of phthisis dates back to 1885, although the possibility of such a state of things had been present in his mind for a number of years before. In June of that year he received under his care a case of phthisis which riveted his attention firmly to the subject, and it is not too much to say that all the study and consideration which he has been capable of giving to this subject since that time have served to arouse and intensify his interest in it, and have given him renewed confidence in its reliability and truthfulness. The history of the case referred to is as follows: Mrs. W., aged 26, was confined during the previous December, from which she made a good recovery, with the exception of a violent attack of pain in the region of the right ovary, as she described it, about ten

days after labor. This soon left her and she remained well until the following January, when she became chilly occasionally and began to have night-sweats. When first seen she had lost some flesh, a poor appetite, irrégular bowels, and a whitish expectoration, not at all profuse. Her menses reappeared one month before. She never had malaria, nor rheumatism, but complained of an ill-defined and sometimes a shooting pain throughout her body, which localized itself behind the right ear, right side of the neck, and right costal portion of the chest. She also perspired more profusely around the back part of the head and neck than anywhere else.

Physical examination showed her heart normal. Right lung: Diminished respiratory motion throughout, and a slight impaired percussion resonance in apex, associated with a few mucous râles in same area. Left lung: Want of proper expansion, otherwise normal. There is a family history of phthisis. She was also examined by an expert gynæcologist, who pronounced her generative organs sound. Under treatment her appetite, cough, and expectoration gradually improved, and the mucous râles had all disappeared by the following August, and, with the exception of several attacks of pain in the right ovarian region, which, during their greatest intensity, radiated upwards to the stomach and intestines, she apparently felt very much better. Her menses failed to appear, however, after the following September, and from this time on her case developed the most uncommon nervous symptoms. Her temperature, which did not vary much from the normal hitherto, now began to rise. The ovarian pain became very much aggravated, and tenderness began to show itself along the whole spine. The pain in the right side of the head, neck, and chest, of which she complained at her first visit, also became worse. In November there was dulness in the right apex and subcrepitation in the same area, which extended to the third rib in front

and to the angle of the scapula behind. Her evening temperature averaged about 101°F. The ovarian pain appeared now at more frequent intervals, and nothing but large doses of morphine would relieve it. In December her arms and lower limbs became so painfully stiff that she was only able to move them with the greatest difficulty. This attack lasted for four or five days, when it entirely disappeared. The pain in the ovarian region and that in the right side of the chest often came on alternately. She frequently said that when the pain appeared in her right lung the abdomen was free from it, and that the reverse was also true. By applying one pole of a constant current between the shoulders and the other on the sternum, the pain would disappear from the chest at once, but would reappear in the pelvic region. The application of a hot poultice to the chest would drive the pain to the parts below, and *vice versa*. When both poles of the battery were applied along the spine—one in the cervical and the other in the sacral region—the pain would recede altogether. In fact, this was the only measure which afforded her permanent relief, and this was decided and instantaneous. After the manifestation of these nervous symptoms she became rapidly worse, and died in a short time. The pulmonary disintegration was limited chiefly to the right lung until about six weeks before her death, when the other side also became invaded. At no time was there any œdema, or paralysis of the extremities in her case; and at no time, except when she was stiff, did she suffer from any well-marked pain in these parts of her body.

What was the precise nature of the relationship between the nervous affection and that of the lung, especially on the right side, in this case? Was the one dependent on the other, or not? If so, which was primary, and which was secondary? On account of the absence of important *post-mortem* data this is, of course, very difficult to determine, but it is very certain that the nervous symptoms antedated

the lung conditions, and that the former aggravated the latter. The points of special interest here are the confinement of the disease to one side of the body until up to within a short time of her death, and the close sympathy which existed between the pain in the right ovary and that in the right side of the chest. It seems as if the pain in the right side of the head, neck, and chest was reflex in character, and depended on the ovarian disturbance as its primary source of irritation, and that in time a reciprocal channel of communication was established between the two, through which they reacted the one on the other.

At this time the author did not know that others had been similarly impressed with the close relationship that exists between pulmonary disease and disease of the nervous system, and on searching for evidence on this question he has been greatly surprised at the richness of material in medical literature which directly supports the theory that phthisis and other pulmonary diseases are secondary to injury and disorder of the nerve supply of the lungs. This evidence is chiefly gathered from the domain of clinical medicine, but from its very nature it is more exact and weighty than if it had been worked out in the laboratory. Laboratory work only becomes really valuable when it is confirmed by the crucible of clinical experience and by the *post-mortem* table. In most of the instances which are given in this chapter, nature herself performed the experiment, as it were, and that, too, with the utmost precision. The conditions demanded by experiment were all that could be desired, and the products, too, were uniform. The terms of the experiment are these: Given a case in which there is pressure or disease of the pulmonary nerves, pulmonary disintegration in some form will follow. Abstracts of the histories of these cases will be related and discussed in the following order:

(1) *Those in which the pneumogastric nerves were injured or compressed by aneurisms, tumors, glands, etc.*

(2) *Those in which specific poisons, like those of alcohol, mercury, scarlatina, diphtheria, etc., produced disintegration of the pneumogastric nerves.*

(3) *Those in which the pneumogastric nerves became involved in diseases of the peripheral and cerebrospinal nervous system; and,*

(4) *Those in which the pneumogastric nerves became implicated in diseases of the highest nerve centers.*

(1) Injury of and Pressure on the Pneumogastric Nerves.

Case 1. After death Montault* found both inferior laryngeal nerves compressed by numerous swellings in an individual, who during life had asthmatic attacks approaching paroxysms of suffocation, and a cough simulating that of pertussis. Condition of lungs not stated.

Case 2. Andral† observed persistent dyspnœa in a patient without perceptible cause. Section showed that both vagi were surrounded and compressed by enlarged lymphatic glands.

Case 3. Robert,‡ in tying the carotid, also included the vagus on that side. The patient began to scream that he would suffocate and became hoarse at once and fainted. He recovered, but remained hoarse for six months.

Case 4. Stromeyer§ observed a case of contusion of the left vagus by a bullet. Respiratory sounds were suppressed in left lung, while those of the right were apparently undisturbed. Death occurred in three weeks, and it was found

* Cited by Longet. *Anatomie und Physiologie des Nerven-systems*, 1849, Bd. ii, S. 311.

† Cited by Longet. *Supra*, p. 312.

‡ *Gaz. des Hôp.*, 1853, No. 102, p. 413.

§ *Maximen der Kriegsheilkunde*, Zweite Auflage. Hanover, 1861, S. 116.

that the bullet had pressed the left vagus against the vertebral column and greatly injured it.

Case 5. H. Demme* contributes a case of vagus injury in which were found at first very slow, deep, moving, laborious respiration and laryngysmus, then marked dyspnœa and diminished respiratory sounds in the lung on the injured side.

Case 6. Dr. S. W. Gross,† while removing a large sarcoma which was incorporated with the large vessels and nerves of the left side of the neck of a female patient, aged about 55, excised about two inches of the left vagus. In the course of eight hours she died of œdema of both lungs, which was most marked on the left side.

Case 7. Swan‡ saw a patient who, in spite of apparent integrity of the lungs, suffered from persistent dyspnœa. Appetite became voracious without being able to satisfy it. The patient emaciated and died, and it was found that both vagi were atrophied.

Case 8. Bignardi§ observed in the body of a woman who died of phthisis, and who had a voracious appetite during the latter part of her life, that both vagi were affected with red-colored neuromatous swellings. (Vagotomy sometimes produces polyphagia in animals.)

Case 9 Longet‖ contributes a case of Johnson, in which he observed softening of the oblongata, compression of the left vagus root on account of aneurismal enlargement of the left vertebral arteries, and complete loss of hunger and thirst.

Case 10. Goodhart¶ saw a child eight months old suffer-

* *Allgemeine Chirurgie der Kriegswunden nach Erfahrungen in den Norditalienischen Hospitalern.*

† *Private communication.*

‡ "Treatise on diseases and injuries of the nerves." London, 1834, p. 170.

§ Longet. *Supra*, Bd. ii, S. 313.

‖ *Supra*, Bd. ii, S. 313.

¶ *Brit. Med. Jour.*, 1879, vol. i, p. 542.

ing from crowing respiration, and very prone to lose its breath. A bad cold, which aggravated this condition, was followed by convulsions which were especially liable to come on when the child was placed in the recumbent position. After death, which soon took place, it was found that the right vagus was compressed by an enlarged gland, and that the middle lobe of the right lung was in a state of collapse.

Case 11. Diebel* publishes the case of a forty-nine-year-old female, from whom Professor Langenbeck extirpated a carcinomatous tumor in the neck, and in doing so he excised the implicated portion of the vagus. Cyanosis and great dyspnœa supervened, and in eight hours the patient died of pulmonary œdema.

Case 12. Langenbeck† describes swelling in the region of the left tonsil in a male patient 65 years old, which he extirpated. Death occurred two days after the operation. It was then found that the left vagus was compressed and flattened by the tumor, and the lungs were œdematous and the bronchi contained a large amount of mucus.

Case 13. Dr. Busch‡ extirpated a glandular tumor from the right side of the neck of an adult patient and in doing this he divided the right vagus. The patient collapsed at once, the voice lost its sound, the pulse became exceedingly quick, respiration was impeded, later convulsive movements of the muscles of the neck, right arm and of the right leg came on, and death occurred in eighteen hours. Section showed œdema of the lungs.

Case 14. Stackler§ contributes the case of a female, aged 53, who was always nervous, and who, for the last five years of her life, suffered from violent palpitation, cardiac pain, œdema of lower extremities, enlarged liver, bloated abdo-

* *Centralblatt f. Chirurgie*, 1881, p. 748.
† *Archiv f. klinische Chirurgie*, Bd. 1, S. 77.
‡ *Med. Times and Gaz.*, 1861, vol. 2, p. 176.
§ *Centralblatt f. klin. Medicin*, 1883, p. 316.

men, frequent vomiting, poor appetite, a pulse rate of forty per minute, occasional epileptic seizures and spasms of right arm, marked cyanosis of the face, and oppression of the chest. The veins of the neck and upper portion of thorax more prominent on right than on left side. *Post-mortem* examination showed dilatation of the aorta from near the aortic valve to the bifurcation of the innominate artery; emphysema and hemorrhagic infarcts in both lungs, and the right vagus, in its whole course behind and along the trachea, was diseased.

Case 15. von Ziemssen* reports the case of a male patient, 69 years old, who was suffering from phthisis, aneurism, weakness of left vocal cord, and a pulse of 100. After death it was found that the left vagus was flattened by the pressure of the aneurism.

Case 16. Bernheim† and Simon describe a case of aneurism of the arch of the aorta, which was followed by pulmonary phthisis. Section showed compression of right vagus and of both recurrent nerves.

Case 17. Sir William Gull‡ reports the case of a male, aged 61; had been ill for four months when admitted, and was greatly emaciated. Symptoms began with sharp pains in the right side of his chest, without cough. The whole right side was flattened and the infraclavicular fossa was much depressed. Dulness pervaded the whole of this side, too. No bronchial sounds or respiratory murmur audible. Left side resonant throughout, but contained some râles in the larger tubes. After death universal adhesions of the right pleura were found. There were fibrous thickening and induration around the large bronchus, not narrowing or compressing it, but implicating the trunk of the right

* *v. Ziemssen's Handbuch*, Bd. xi, S. 2.

† Abstract in *Internationales Centralblatt für Laryngologie*, 1887–1888, p. 68.

‡ *Guy's Hospital Rep.*, 3d ser., vol. v, 1859, p. 312.

pneumogastric nerve and the branches of the pulmonary plexus. The trunk of the nerve was so entirely confounded with the new tissue that it could not be traced through it. The smaller divisions of the bronchial tubes were universally dilated up to the periphery of the lung, and they, as well as the larger tubes, were choked with muco-purulent secretion. The pulmonary tissue was consolidated and in the stage of gray and iron-gray hepatization. In the lower part of the upper lobe was a large sloughing cavity. In some parts the pulmonary pleura was an eighth of an inch thick. The left lung was healthy, as were the heart and the abdominal viscera.

Comments by Dr. Gull: "This case affords an excellent illustration of the effects which are referable to paralysis of the pulmonary plexus on one side—accumulation of muco-purulent secretion in the paralyzed bronchi, subsequent dilatation of the tubes at their peripheral distribution, concomitant exudation into the air cells (hepatization), and at length disintegration of the tissues. . . . It is one of the evils of a too-exclusive humoral pathology, that it leads us to overlook the minute anatomical relations of disease, which are in themselves often a key to the sequence of morbid changes. This and the two following cases illustrate this proposition, for the possible local effects on the lung of injury to the pneumogastric and pulmonary plexus being recognized, whenever cause for that injury exists we may anticipate its results, and are not wholly dependent upon physical examination as we are if we limit our pathologic view to the mere changes in the lung without considering how they are produced."

Case 18. Gull* describes the case of a male, aged 45, who was always well until he became 23 years old, when he was attacked with paralysis of the right side and aphasia. He gradually recovered, but was never able to read or write, and

* *Ibid.*, p. 307.

his face remained thin and haggard, his expression was anxious, his voice husky, respiration hurried, had dysphagia, tickling in the throat and cough, but no pain in the chest. Dulness over the whole of the left side of his chest. No vocal fremitus, nor vesicular, but bronchial breathing in the apex of this lung. Right lung normal except puerile breathing and some mucous râles in the larger tubes. Under the cartilage of the second rib on the left side is heard a soft, double murmur. After death the right lung was found to be healthy. Left lung in a state of consolidation, with irregular cavities in the upper lobe, which communicated with the bronchial tubes. The tubes themselves were filled with muco-purulent secretion. Heart normal. At the left side of the arch of the aorta there was an aneurism of the size of a large orange, which had extended downwards and backwards and compressed the left pneumogastric nerve and the adjacent branches of the pulmonary plexus.

Case 19. Gull* gives the history of a male, aged 45, who had difficult deglutition and pain under lower third of sternum, cough, and a peculiarly offensive muco-purulent expectoration streaked with blood. Dulness from the lower angle of the scapula to the base of the right lung, absence of respiratory sounds over seat of dulness, and subcrepitation and bronchophony above. *Post-mortem* examination showed the existence of cancer of the œsophagus, the ulceration of which extended down as low as the root of the lungs, but neither the lungs themselves nor the pleuræ were invaded. The right pneumogastric nerve was at this point of its course implicated in the disease so that it could not be traced to its distribution. The right lung was extensively consolidated, its lower lobe being infiltrated with a grayish or greenish sero-purulent fluid of an offensive odor. Left lung and heart and other viscera healthy.

Case 20. Dr. Stimson† gives the history of a male, aged

* *Ibid.*, p. 311.

† *Amer. Jour. of Medical Sciences*, 1881, p. 192.

46, who fell while carrying a heavy load on his shoulders. Soon after this his voice began to change and afterwards a pulsating tumor appeared just above the right side of the sternum. Admitted nine months after his fall, when it was found that he suffered from aneurism of the first portion of the left subclavian artery. Died of phthisis three years afterwards. Examination showed the heart small, but its valves were normal. Arch of aorta uniformly dilated; but there was an irregular enlargement occupying the position of the innominate and subclavian arteries in the walls of which the right vagus was lost.

Case 21. Whipham* details the case of a male, aged 55, who had paroxyzmal cough which was followed by a copious, watery expectoration. He also suffered from pain in the thorax, and death, due to difficult respiration, occurred a short time after admission. An aneurism in the posterior wall of the descending portion of the arch of the aorta was then found. The left recurrent and pneumogastric nerves were flattened and compressed by the tumor. Cavity in the apex of right lung.

Case 22. Murchison† gives the following history: Male, 20 years old, suffered from great dyspnœa, paroxysms of cough, hoarse and husky voice, and was unable to lie down. There was dulness in right infraclavicular region, and he died in about a month after his admission. Section: A cancerous tumor was found in the upper part of the right chest, which did not appear to invade the right lung. The right pneumogastric and recurrent nerves passed through the cancerous mass and there were several cavities in the right lung.

Case 23. Baumler‡ contributes the case of a male, aged 53, who complained of pain in the neck, occiput and right

* *Trans. Lond. Path. Society*, vol. 33, 1882, p. 82.
† *Trans. Lond. Path. Society*, vol. x, p. 240.
‡ *Trans. Lond. Path. Society*, vol. 23, 1872, p. 66.

shoulder, and of cough and expectoration. There was decided dulness over the upper part of the sternum, extending about an inch and a half towards the right. He became aphonic, had much dyspnœa and pain, and died two months after admission. Section: Phthisis in both lungs, an aneurism of the innominate artery compressed and flattened the right recurrent and pneumogastric nerves.

Case 24. Hanot* gives this case. Male, aged 38, had always been well until a short time ago he began to have pain in the head, left eye and ear, difficulty in swallowing, weak voice, accelerated respiration, and dyspnœa. Left vocal chord immovable on phonation. He became worse rapidly and died four months after admission. Section: Aneurism of arch of aorta. Left pneumogastric and recurrent nerves were so closely incorporated with the sac of the aneurism that they could not be separated. The whole left lung had undergone caseous degeneration.

Case 25. Grocco† gives the history of a patient who suffered from respiratory spasm and cardiac irregularity and who died of phthisis. After death it was found that the vagus and its cardiac branches were compressed by enlarged lymphatic glands in the posterior mediastinum.

Case 26. Wilks‡ admitted a female, aged 25, into Guy's Hospital, who was affected with general neuromatous swellings, and who finally died of phthisis. Nearly every nerve in the body was involved, and the pneumogastrics had a number of swellings on them. In his comments on this case, Dr. Wilks says: "It has been said that the patient died of phthisis, and that these tumors were found accidentally, but in all probability the affection of the nerves, that of the pneumogastrics, was the cause of the pulmonary disease, and, therefore, so far from neuroma being a harmless affection it was the cause of the girl's death. This idea was suggested

* *Archives gen. de méd.*, tome xxviii, 1876, p. 294.

† *Schmidt's Jahrbücher*, 1887, Bd. 214, S. 29.

‡ *Trans. Lond. Path. Society*, 1859, vol. x, p. 1.

by the observation of several other cases of pulmonary disorders, occurring in connection with disease of these nerves—particularly as witnessed in aneurism of the aorta and cancer of the œsophagus; in these diseases death is often brought about by pulmonary affections, and the pneumogastric nerves are found implicated in the disease, or pressed on by the tumor.

Case 27. Heusinger* describes the case of a male, 23, years old, who was weak, pale, emaciated, and had hollow supra- infraclavicular fossæ, and deep intercostal spaces. Neuromata over his whole body. Pulmonary phthisis of both lungs. Death one month after his admission. Section: Both apices infiltrated. Neuromatous swellings of all the peripheral nerves. The vagi, on both sides, were swollen to the size of a finger.

Case 28. Male,† 55 years old, suffered from right lateral homonymous hemianopsia and other symptoms indicating a serious condition of the brain. After death the lungs were found in a congested and hemorrhagic state, the brain was softened, membranes thickened, the vessels sclerosed and thick, and the oblongata congested.

Case 29. Eisenlohr‡ gives the account of a male, aged 33, who began to complain of hoarseness, irritable cough, dysphagia, etc., in 1881. At the time of his admission he suffered from marked aphonia, which was found to be due to left-sided recurrent paralysis. His pulse rate was 100, and he also showed loss of sensibility in the face, and had rolling of the eyeballs. In 1884 he began to develop an affection of the lungs, which was accompanied by fever, hemoptysis, and from which he died at the end of that year. Section: Phthisis pulmonalis, associated with pleurisy and marked degeneration of the nucleus and root of the pneumo-

* *Virchow's Archiv*, vol. 27, p. 206.

† *Trans. Lond. Path. Society*, 1890, p. 1.

‡ *Archiv f. Psychiatrie u. Nervenk.*, Bd. 19, S. 314.

gastric nerve. In his remarks on this case Dr. Eisenlohr says that without doubt the lung affection depended on the bulbar degeneration, for he was absolutely free from pulmonary disease when he first came under observation.

Case 30. Generisch* contributes the history of a male, 22 years old, who suffered from numerous large and small resistant fusiform tumors, which were scattered over his whole body. He began to have pain, fever, and died of pneumothorax. Section: Phthisis pulmonalis. The vagi were enlarged and knobbed, as were also the phrenic, and many other nerves.

Case 31. Habershon† describes the case of John D., aged 71, who had been under observation for two years before he entered the hospital. He had difficulty in swallowing and was markedly emaciated, and finally died of pneumonia. The œsophagus was contracted one inch above the bifurcation of the trachea, and its walls were thickened and contained cancerous deposits. The left pneumogastric nerve was involved in this diseased structure. The entire left lung was in a state of gray consolidation.

Case 32. The same authority (p. 97) details a similar history. Male, aged 50, had suffered for six months with dysphagia before his admission. He coughed and expectorated, and his lungs became involved. Death. There was cancerous ulceration of the œsophagus, extending from the point of the cricoid cartilage to the bifurcation of the trachea. This had destroyed the larger branches of the left pneumogastric nerve. The greater portion of the lower lobe of the left lung was in a state of gray hepatization, and in the apex of the same lung some iron-gray hepatization was found associated with white tubercles. The right lung was also somewhat condensed.

Case 33. Fearn‡ gives an account of a female, aged 68,

* *Virchow's Archiv*, Bd. 49, S. 15.

† "Diseases of the Abdomen," 3d ed., p. 96.

‡ *Amer. Jour. of Med. Sciences*, 1848, p. 266.

who was stabbed in the left side of the neck with a pruning knife, and who lived seventy-nine days after the attack. During the whole of this period she had a suffocative cough, a distressing difficulty in swallowing, an excited and a very weak circulation, and was in a state of extreme exhaustion. On section it was found that the left pneumogastric nerve had been divided and that both lungs were involved in purulent bronchitis.

Case 34. Riegel* gives the following history: Male, 53 years old, suffered from oppression of the chest, dyspnœa, cough, palpitation of the heart, and much emaciation. Pulse 164 per minute. Death. Section showed that the left vagus was imbedded in a degenerated lymph gland. Slight degree of bronchitis existed.

Case 35. Eccles† contributes the case of a male, aged 46, whose common carotid artery was ligated on account of a glandular tumor of the neck. Patient improved, but died in several months of bronchitis. Section showed the carotid obliterated, and that the vagus of the same side was compressed by the tumor.

Case 36. Quain‡ observed a female, aged 55, who began to suffer with wheezing in her throat and chest, which was followed by considerable expectoration. She soon began to have dysphagia, and rejected her food. In three months she had an attack of pneumonia, and gradually became worse, and in three months more she was dead. Section: Heart and left lung were apparently healthy. Left pleura slightly adherent, right completely so. Lower lobe of right lung was completely hepatized. Large, fibrous, nodulated tumor in posterior mediastinum, in which both vagi were imbedded.

Case 37. Maixner§ saw a male, 27 years old, who suffered

* *Berlin. klin. Wochenschft.*, 1875. No. 31.
† *Lancet.* 1844, vol. 1, p. 724.
‡ *Trans. Lond. Path. Society*, 1857, vol. 8, p. 45.
§ *Prager Vierteljahrschrift*, 1879, 1, S. 87.

from infiltration of the upper lobe of the left lung. He also had palpitation of the heart and præcordial distress, and a pulse rate of 192. Death. Section showed an enlarged and degenerated bronchial gland compressing the left vagus. Pneumonia and pleurisy on left side.

Case 38. Weil* observed a male patient, 33 years old, who had a number of mediastinal swellings, but who manifested no disturbance of the heart, excepting a pulse rate of 116. His respiration was normal and his voice was unchanged. Two weeks later he became feverish, his respiration rapid, and his pulse rate 200, but his heart sounds were pure and there was no palpitation. Death occurred from pneumonia, and it was then found that the vagus was compressed by a mediastinal tumor.

Case 39. Goodhart† saw an eight-year-old child who suffered from severe paroxysmal attacks of cough and dyspnœa. The child would suddenly start up in bed, his face becoming blue, his eyes staring and all the respiratory muscles were thrown into violent action. Death. Recent double pleurisy and bronchopneumonia at both bases. The right vagus was so firmly adherent to and compressed by an enlarged bronchial gland that it could not be isolated.

Case 40. Pelizaeus‡ relates the case of a male, aged 47, who complained of cough, hoarseness, and occasional marked dyspnœa. There was crepitation over the whole pulmonary surface, with dulness at the apices. Temperature was normal, pulse 96, and his voice was hoarse. He improved for some time, but afterwards began to suffer from fever, had a pulse from 100 to 124, and five months after coming under observation he was seized with violent dyspnœa, cyanosis, a pulse of 140 to 164, and died in one of these paroxysms. Right vagus was compressed by a degenerate lymph gland.

* *Archiv f. klin. Med.*, Bd. xiv, 1876.

† *British Med. Journal*, vol. i, 1879.

‡ "*Inaugural Dissertation*," Wurzburg, 1880.

Case 41. Hubrich* saw a male patient, aged 31, who suffered from vomiting, dysphagia, and weakness in the lower extremities, and who died of pulmonary disease in about a year after he was admitted. Section showed tumor in the cerebellum, which pushed forward and compressed the right vagus and glossopharyngeal nerves. The right lung was gangrenous.

Case 42. Lecorché† reports the case of an adult male who was afflicted with violent attacks of coughing, like those of whooping-cough, pain in the right side of the chest, a sense of suffocation, and an abundant muco-purulent expectoration. The posterior base of the right lung was consolidated. Death. Section showed an aneurismal tumor of the size of a mandarin, occupying the right innominate artery, which forced the right vagus forward and flattened it. There was also bronchopneumonia of the right lung, and the left lung was slightly affected.

Case 43. Fergusson‡ gives the history of a male patient, 56 years old, who was well until three weeks before his admission, when he began to suffer with severe pain in the right side of the head and neck, which extended to the shoulder and arm of the same side. He also began to cough and soon a small pulsating tumor appeared directly above the inner end of the clavicle, which gradually increased in size and in pulsation. The viscera of the chest seemed healthy. The common carotid was ligated, and five days after this he began to cough and expectorate, and to suffer pain in the chest. The next day evidence of bronchitis developed and on the following day he died. Section: Right lung healthy, with the exception of a large amount of mucus in the tubes. No pleural effusion. Left lung was congested and middle lobe was engorged with blood, and mucous patches of recent

* *Archiv f. Psychiatrie u. Nervenkrank.*, vol. v, p. 550.

† *Centralblatt f. klin. Med.*, vol. i, p. 282.

‡ *Amer. Jour. of Med. Sciences*, vol. 3, 1842, p. 221.

lymph, mingled with pus, were present. Right vagus was in close contact with aneurismal tumor, and both it and the recurrent laryngeal were compressed by the enlargement.

Case 44. Wilks* contributes the case of a male, 71 years old, who had cancer of the œsophagus and died of pneumonia. The cancerous process did not extend to the lungs, but it involved the left pneumogastric nerve and partly destroyed it. The pneumonia was on the left side.

Case 45. Eger† gives the history of an adult male who discovered a small, painless swelling in the right side of his neck which was accompanied by cough and hoarseness. His left vocal cord was motionless in the middle position during respiration and phonation, while the function of the right cord was undisturbed. Six months after the appearance of his trouble he had a severe attack of dyspnœa, for which tracheotomy was performed. Two days after this he died suddenly. Section showed that pneumonia and pulmonary œdema existed, and that a mediastinal tumor compressed the left vagus.

Case 46. Hanot‡ contributes the case of a male, 59 years old, who suffered from cancer of the œsophagus. Physical examination of his chest showed consolidation of right apex, and catarrh of the whole chest. Deglutition became more and more difficult and he gradually grew worse and died. There existed pulmonary phthisis, cancer of the œsophagus, and great enlargement of neighboring lymphatic glands. Both vagi passed through this ganglionic mass, and the right was thin, patulous and fibrous.

Case 47. Arnold§ describes the case of a female who was phthisical and who, during the last few weeks of her life, had a pulse of from 100-124. After her death the following

* *Trans. Lond. Path. Society*, vol. x, p. 159.
† *Archiv f. klin. Chirurgie*, vol. 18, p. 502.
‡ *Archives générales de médecine*, tome xxviii, 1876, p. 294.
§ *Deutsches Archiv f. klin. Med.*, vol. vi, p. 277.

condition was found. The right lung was congested and contained tubercular nodules. The left lung also had some softened nodules. The left vagus and recurrent were normal. Right vagus had undergone carcinomatous degeneration in the subclavian region, and was swollen here to a thickness of five mm. The left part of the thymus gland had undergone colloidal degeneration and there was serous effusion in the pericardium.

Case 48. Chapin* gives the account of a child nearly three years old who had a slight cough, but no dyspnœa, and in whom the physical signs of the chest indicated no disease. In a few days, however, violent dyspnœa set in and death took place. Post-mortem examination revealed an abscess which proceeded from the first and second dorsal vertebræ, and extended forward and pressed on the right vagus. Directly in front of the nerve, and in contact with the abscess, was a cheesy, bronchial gland. At its seat of contact with these masses the vagus was compressed and inflamed. Miliary tuberculosis existed in both lungs and in the trachea.

Case 49. Stackler† contributes the following case: Female, 53 years old, was nervous since her youth, and during the last five years of her life she complained of violent palpitation and pain in the cardiac region. She also suffered from oppression of the chest, slow pulse and respiration, former 40 per minute, and irregular, œdema of lower extremities, poor appetite and vomiting. At times she had epileptic seizures—spasms in the right arm, and marked cyanosis of the face. Dulness in the upper portion of left lung. Death. Autopsy: Both lungs permeated with hemorrhagic infarcts. Brain and spine and heart normal. Aorta much dilated, especially from the valves to the bifurcation of the innominate artery. The right vagus, in its whole course behind and along the trachea, was diseased.

* *N. Y. Med. Jour.*, 1884, p. 294.

† *Centralblatt f. klin. Med.*, 1883, p. 316.

Case 50. Riedel* relates a case in which, in order to extirpate a cervical gland that enveloped the left vagus, it was necessary to exsect 15 cm. of this nerve. The patient died in fourteen days, and a general purulent bronchitis of the left lung was found after death.

Case 51. Mercklen† gives the history or an eighteen-year-old male patient, who had cardiac palpitation. His pulse was small—160 per min.—but there was no cardiac disease. Shortly after the patient was seen the apex of his left lung became infiltrated and he died suddenly, while making an effort to rise from his bed. Post-mortem examination showed that his mediastinum was filled with enlarged tubercular glands, which enveloped and compressed the left vagus. There was also a tubercular affection of the lungs.

Case 52. Modrzejewski‡ details the case of a female, aged 37, whose whole body was covered with nodules of different sizes—3020 in all. After death from pneumonia it was found that nearly all the nerves of her body were affected with multiple fibromata. In the region of the neck there were two large swellings on the vagus.

Case 53. Gerhardt§ describes the case of a male 30 years old, who suffered from multiple neuromata and who died of pulmonary œdema and pleurisy. There were neuromatous swellings on the vagi and on the other cranial nerves.

Case 54. Pilz‖ gives the history of a male patient, 43 years old, who suffered from a vascular tumor, which extended from the frontal region to the parotid gland, and for which the carotid was tied. After the operation the patient became temporarily unconscious, his respiration became weak, dysphagia ensued, and death followed the operation

* *Fortschritte der Medicin*, 1883, p. 499.
† *Deutsche medizinal Zeitung*, 1887, p. 1108.
‡ *Berlin. klin. Woch.*, 1882, p. 627.
§ *Deutsche Archiv f. klin. med.*, vol. 21, p. 279.
‖ *Archiv f. klin. Chirurgie*, vol. 9, p. 336.

in ten days. Section showed phthisical infiltration of both apices. Vagus on side of tumor was red, swollen and imbedded in exudation.

Case 55. Eccles* contributes the case of a male, 46 years old, who was affiicted with glandular tumors of the neck, for which the common carotid artery was ligated. The patient died several months afterwards of bronchitis. Section showed the carotid obliterated, and that the tumor enveloped the vagus.

Case 56. Brown-Séquard† communicates four cases of disorder of the oblongata, in which diseases of the lungs were met. In one case a tumor pressed on the left side of the oblongata and vagus, and pleurisy and pulmonary tuberculosis were present. In two other cases pressure was produced on the left side of the oblongata and pneumonia was found in both instances. In a case of tumor pressing on the left protuberances the patient died of phthisis. In two other cases, where the vagus appeared not to have been affected, there were no pulmonary lesions.

* *Lancet*, 1884, vol. 1., p. 724.

† *Journal de Physiologie*, cited from Van der Kolk; "Atrophy of the Brain," *Syd. Pub.*, vol. xi, p. 170.

CHAPTER III.

ETIOLOGY CONTINUED; (2) THE PRODUCTION OF PULMONARY DISEASE THROUGH THE ACTION OF SPECIFIC POISONS ON THE PNEUMOGASTRIC NERVES.

In this section a number of cases will be given, which demonstrate that although pulmonary consumption is not, strictly speaking, a specific disease itself, it may be bred by a great many specific poisons. These poisons are well recognized in some instances, as in alcohol, mercury, and lead, for example; while, on the other hand, the specific nature of the poisons of syphilis, scarlatina, measles, etc., is a land of darkness to us, it must be admitted that such intoxicating agents exist, and that, like the better known poisons, they have the power of exercising a pernicious influence on the nervous system, and especially on the nervous supply of the lungs and of the heart.

Alcohol. Among the agents which are most potent in the production of pulmonary consumption the first place belongs to alcohol. Its action in this respect has been well studied, and it is pretty generally understood that it possesses a special affinity for the nervous system. Dr. James Jackson, in this country, and Dr. Wilks, in England, were, so far as the author knows, the first to point out this form of nerve-disease, and they called it alcoholic paralysis. It has, since then, received the more appropriate name of alcoholic neuritis, and it is characterized in its early stages by numbness, tingling, hyperæsthesia in the extremities, and later on by anæsthesia, paralysis of motion, loss of knee-

jerk, quickened pulse, shortness of breath, and frequently by pulmonary embarrassment. The brain and spinal cord remain comparatively normal. The morbid changes occurring in the peripheral nerves, under the influence of alcohol are parenchymatous and interstitial, or, in other words, they are confined to the nerve substance itself, or to its investing membrane. As a rule, these changes occur together, the latter in many instances depending on the former, but frequently one exists exclusively of the other, especially in the case of degenration of the nerve itself. Dr. Buzzard* gives a very interesting account of the common symptoms and conditions of alcoholic neuritis, viz.: Patient is unable to stand and is compelled to lie in bed. He is able to flex the thighs upon the pelvis fairly well, and possibly to bend the knees. The muscles of his legs are atrophied, and "wrist drop" may exist. Lightning pains, or gnawing, burning or pricking sensations are usually most pronounced in the lower extremities. There may be more or less cutaneous anæsthesia in the legs and feet. There is usually a remarkable loss of memory. The *post-mortem* findings are subacute inflammatory processes in nerve trunks, especially in the radial, tibial (anterior), or the peroneal. The nerves seldom show any particular change to the naked eye, but after being hardened evidence of parenchymatous degeneration is discovered in them, together with more or less interstitial neuritis. *In females the catamenia are almost always suppressed, and often for many months during the illness. Paralysis of respiration and of deglutition is common. Pneumonia, which occasionally terminates the case, is very likely due to lesions of the vagus.*

Case 57. Drs. Oppenheim† and Siemerling give the history of a male alcoholic, aged 26, who was received on account of delirium tremens. Had been addicted to alcoholic

* *British Med. Jour.*, June 21, 1890, p. 1419.

† *Archiv f. Psychiatrie u. Nervenkrankheiten*, Bd. 18, S. 114.

excess for three years. He improved and was dismissed, but during the two following years he had two attacks of the same kind and was again admitted. He now had marked disturbance of the nervous system. He suffered from lamiating pains, dizziness, and tenesmus of the rectum. Sensibility and motility of the lower extremities were much impaired. Five months after his last admission he began to have infiltration of both apices. His well-nourished body emaciated universally, and he gradually sank and died. On microscopic investigation it was found that degeneration was present in the oblongata, in the posterior columns, and in all the peripheral nerves which were examined. There was marked evidence of phthisis, but the state of the vagi was not inquired into.

Case 58. Thomsen* cites the case of a male, 47 years old, who was a chronic alcoholic. He was admitted with delirium tremens. His temperature was subnormal, pulse 86, his gait was unsteady, he had dizziness and suffered from dyspnœa, distress in chest and in throat. Died in about two weeks. Post-mortem examination showed atelectasy and bronchopneumonic foci; hyperæmia of oblongata, and a grayish color of the cranial nerves. The nucleus of the hypoglossus was degenerated to a high degree. The condition of the vagus could not be definitely ascertained.

Case 59. Vierordt† relates the history of a male, aged 30, who was much addicted to alcoholic excess. For a year before he died he had piercing, lightning pains, weakness, unsteadiness, and formication in the lower extremities. Three months before his death wasting began in the upper extremities and marked neuralgia and paresis developed in his legs, as well as tuberculosis of the lungs. After death the oblongata, the cervical and dorsal portion of the spine and the columns of Goll were degenerated. There was also extensive tuberculosis of the lungs.

* *Ibid.*, Bd. 19, S. 191.

† *Ibid.*, Bd. 18; *Neurolog. Centralblatt*, Bd. 5, p. 421.

Case 60. Schultze* reports the history of a male, aged 39, who developed diabetes insipidus in 1882, but had been feeble since childhood. He used alcohol to excess. Sometime after this date he began to suffer from nystagmus, trembling in the arms, perversion (paræsthesia) of sensation in the legs, and of constriction around the thorax. In 1886 he had severe attacks of dyspnœa, and death was caused by paralysis of respiration. Section: Degeneration of oblongata, spinal cord, and of the nuclei of the vagus and hypoglossus. No information is given concerning the physical state of his lungs.

Case 61. Strümpell† details the case of a male, aged 47, who was received November 25, 1881, and died February 13, 1882. Up to the time of his admission he used alcohol to excess, and on admission suffered with tearing pains in his arms and legs, and with a weakness and uncertainty in the latter. During the last six months he was confined to his room. Three weeks before he suddenly became restless and delirious, the paresis of the legs increased rapidly and the arms also became weak. He seemed to be a large, powerfully built man, with slight symptoms of psychical disturbance. Both arms hung helplessly at his side, and the back of the hands were œdematous. One month after admission, temperature 38.5° C., pulse 124, and deglutition and power of speech were unimpaired. Seven days before death his temperature rose to 39° C., and he became weak, and began to suffer from diarrhœa and œdema of both legs. The day before his death his respiration was suddenly disturbed, which became quickened and laborious. With each inspiration there was labored action of the muscles of the neck, while the diaphragmatic respiration remained quiet. There was slight cough and a bronchial murmur on both sides. Death came through respiratory disturbance and general ex-

* *Neurolog. Centralblatt*, Bd. 6, 1887, p. 271.

† *Archiv f. Psych. u. Nervenkr.*, Bd. 14, S. 339.

haustion. Section: Marked phthisis of the lungs; sections of the radial, median, crural, and sciatic nerves showed very decided degeneration. Spinal cord normal. It was inferred that the phrenic nerves were also degenerated. Commenting on this case Strümpell says that the paralysis of respiration, and especially of the diaphragm, is of great clinical importance. It is very probable that the cause of this, he thinks, was due to disease of the phrenic nerve, and the marked pulse frequency he attributed to an affection of the vagus. He further states: "Whether tuberculosis has any relationship to neuritis we do not know. It is important to notice, however, that in many of the recorded cases of multiple neuritis tuberculosis co-existed."

Case 62. Ross* reports the case of a male, 35 years old, artist, a chronic alcoholic, who was admitted December 30, 1889. About a month before he began to suffer from severe cramps in the calves of his legs, his feet and hands had become numb. His respiration was 28, pulse 132, and temperature 99° F. There was dulness in right apex and towards the base of same lung, and râles were heard over the whole of his right lung. Dyspnœa was marked; he rapidly grew worse and died on the following 14th of January. Phthisical degeneration of his right lung, and the vagi, phrenici, and anterior tibial nerves were degenerated.

Case 63. Déjerine† contributes the case of a female, 46 years old, who suffered from chronic alcoholism, and consequently from paralysis of upper and lower extremities. Pulse from 150-160. No fever. Heart sounds were pure. Death from pneumonia. Section showed parenchymatous neuritis of the cutaneous and muscular nerves, as well as of both vagi in the cervical region.

Case 64. Sharkey‡ relates the history of a female, aged

* *Medical Chronicle*, May, 1890, p. 91.

† *Deutsche med. Zeitung*, 1887, p. 711.

‡ *Trans. Land. Path. Society*, 1888, p. 27.

32, who was admitted August 29th and died September 25, 1887. She had been a hard drinker for three years, and had hemoptysis five months before her admission, from which time she gradually lost flesh and became weaker, vomited everything, and lived almost entirely on beer. Her temperature was normal, her pulse was weak, and 130 per minute, and her respiratory sounds were harsh. She had no albuminuria. On September 9th she had rigors, her face became livid, her temperature rose to 102.8° F.; great dyspnœa occurred, and her respiration rate increased to 58, and that of the pulse to 180. Paroxysms of dyspnœa, high fever, accelerated pulse and respiration continued, hemoptysis again returned and death was caused through failure of respiration. Section: Phthisical degeneration in both apices. Brain normal. The dorsal and cervical regions of the cord were softened. The vagus, phrenic and other nerves were degenerated.

Case 65. Eisenlohr* details the case of a male, 25 years old, who was a musician, and an inebriate. He was thoroughly drenched in a rain-storm on December 18, 1877, after which he began to complain of marked weakness in the lower extremities and trouble in his chest. The weakness increased to such an extent that he became unable to walk and was received into the hospital on the 27th of the same month, when it was found that he also suffered from right-sided pleurisy, and paralysis and atrophy of the muscles of the lower extremities. He became subject to violent pains in the lower limbs, but the upper limbs remained free from pain. He gradually developed peribronchitis in both lungs, and tubercular peritonitis, and died the following May. Section: Tuberculosis of both lungs, peritoneum, and pleurisy. No visible change in the brain or spinal cord. Marked muscular atrophy of the lower extremities. The muscle-nerves and the trunks of the sciatics were much degenerated. No other nerves were examined.

* *Centralblatt für Nervenheilk.*, vol. ii, 1879, p. 100.

SYPHILIS. Syphilitic phthisis has been recognized by most writers on pulmonary disease. Indeed it is a question with some authorities whether or not all that which goes under the name of pulmonary consumption and scrofula is originally due to syphilis. That this assumption is by far too sweeping to be true is shown by the fact that pulmonary phthisis exists with as great a frequency and fatality among monkeys as it does in the human race, yet syphilis has never been found among that species of animals. There can be no question, however, that consumption is very often generated through syphilitic infection; and that some pulmonary affections, which are regarded as examples of the ordinary non-specific kind, possess an element of congenital syphilis.

Dr. A. Sokolowsky* says that clinically there are two distinct forms of syphilitic phthisis: (1) There appears, without any apparent signs of syphilis in other organs, well defined syphilitic hyperplasia in the lungs, with signs of consolidation, etc., but with complete absence of the ordinary symptoms of phthisis, such as fever, sweats, diarrhœa, etc.; (2) materially more frequent is that form which manifests itself as syphilitic pneumonia, and in connection with syphilitic affections in other organs. In this form are present many of the characteristic signs of ordinary phthisis, such as wasting, cough, diarrhœa, albuminuria, etc., but fever and sweats are likewise absent. Dyspnœa is frequently a prominent symptom which may become asthmatic in character. At the autopsy he found, in one case, hard, cicatricial swellings, together with cavities in both apices, while in two other cases there was present, besides cavities in the upper lobes, a well-marked hyperplasia of connective tissue in other parts of the lungs. Dr. A. Hiller† tabulates the post-mortem results

* "Ueber die Luetische Phthisie," *Centralblatt f. die med. Wis.*, 1884, p. 122.

† "Ueber Lungensyphilis und syphilitische Phthisie," *Charité Annalen*, ix, 1884, S. 184.

of 58 cases of pulmonary syphilis. The typical changes which were found to be most abundant were interstitial connective-tissue proliferation, peribronchial fibrous induration, diffuse thickening of the lobular parenchyma, syphilitic gummata, and nodular induration or bronchopneumonia.

So much, then, concerning the changes which this virus produces in the lungs. What other influence has it? That syphilis has a special affinity for the nervous system, and exerts a poisonous action on it in its tertiary manifestations, there can be no question. It may attack any nerve tissue, but it seems to affect the cranial more frequently than the peripheral nerves. Like alcohol, its intoxicating effect shows itself in pain, hyperæsthesia, anæsthesia, neuralgia, spasm, and paralysis in the course of nerve tracts, on the one hand, and in infiltration and degeneration of the same on the other. Both analogy and the direct evidence which will be offered below seem to show that, like alcohol, it produces pulmonary disintegration through the instrumentality of the nervous system.

Case 66. Walshe* contributes the case of a male, about 27 years old, who had chancre and suppurating bubo. "Some months later a troublesome cough brought him under my notice; he had had no hemoptysis; the expectoration, mucopurulent, was destitute of special character; there was no pyrexia; little if any positive wasting; the aspect generally was not suggestive of tuberculosis or other disintegrating lung disease.

"The left side of the chest had all the physical attributes of health. On the right side high-pitched, toneless dulness, not the lest wooden in quality, suggestive of soft consolidation, reached from a shade below the clavicle to about the fourth rib, almost joining with the liver dulness; it was not perceptible above the clavicle, nor in the axilla, and was much less marked behind than in front. The respiration, high-

* "Diseases of the Lungs," 4th edition, p. 513.

pitched but weak, reached the ear unattended with dry or moist rhonchus. The patient went South, improved in general health; shortly after had epileptic seizures; came home, with palpable extra-cranial and inferrible intracranial nodes, and his lung unchanged. He was put on a course of bichloride of mercury, under which, among other changes, the lung so completely recovered that after awhile not a particle of difference could be detected in the percussion note on the two sides. Years afterward I saw this patient in perfect health."

Case 67. Naunyn* gives the case of a female, 19 years old, who was received January 15, 1882, on account of syphilitic infection, but had always been well until the previous May. Some time after her admission she developed a violent cough, dyspnœa, debility, night-sweats, weakness in the lower extremities and marked shakiness while walking. Temperature 38.8° C.; no œdema; sensorium and sensation unimpaired. Physical examination now revealed phthisical degeneration in her lungs. Death, July 18, 1882. Section: Phthisis in both lungs, and degeneration in the oblongata, and in the cervical and dorsal portions of the spinal cord.

Case 68. Penzoldt† contributes the case of a female suffering from tertiary syphilis, followed by paraplegia. This became complicated with inspiratory dyspnœa—the vocal cords being paralyzed, only allowed a small place between for the entrance of air. Expiration was good. Pneumonia occurred and tracheotomy was performed in order to relieve the impeded respiration, but the patient died, and it was then discovered that both vagi and accessorii were atrophied.

Case 69. Buss‡ reports the case of a female, aged 29, who was infected with syphilis, became hemiplegic, first on one, and in twelve days, on the other side. She had disorder of

* *Deutsches Arch. f. klin. Med.*, Bd. 34, S. 433.

† *Centralblatt f. d. med. Wissenschaften*, 1874, p. 474.

‡ *Ibid.*, 1888, p. 195.

speech, hearing, taste, and sight. She also suffered from dysphagia and marked ataxia. Death followed from pulmonary phthisis. Section: Sclerosis of both olivary bodies, degeneration of the fibres in the oblongata, and atrophy of its ganglia.

Case 70. Neumann* details the history of a syphilitic female, aged 42, who was attacked with left hemiplegia, in 1878. On admission she had great dyspnœa, dysphagia, and whistling breathing, but examination of the larynx showed no stenosis. The inspiratory muscles of the right side of the thorax, and also the right half of the diaphragm, were paralyzed. Breathing became greatly embarrassed more and more until death was produced by paralysis of respiration. Section: In the immediate vicinity of the foramen magnum, and pressing on the right half of the oblongata, was a tumor about the size of an apricot. The vagus fibres, the hypoglossus nucleus, the roots of the tenth, eleventh, and twelfth pair of nerves, as well as the right phrenic, were degenerated. The heart and lungs were apparently unaffected.

Case 71. Echeverria† contributes the two following cases: Female, aged 24, became infected with syphilis, which was followed by epilepsy. Three weeks after the accession of epilepsy she died of double pneumonia. Section: Red and gray hepatization of the lungs. Dura mater adherent to calvarium, arachnoid dense and thickened. Membranes over the oblongata also thickened.

Case 72. Ibid.‡, female, aged 26, acquired syphilis, which was followed by epileptic attacks. After this the apices of her lungs became infiltrated, and pleurisy developed in the right side of her chest. After death her lungs were found in a phthisical condition. Fatty degeneration of the oblongata chiefly on the left side, along the path and nucleus of the

* *Centralblatt f. klin. Med.*, vol. ii, p. 230.
† *Journal of Mental Science*, vol. 26, p. 165.
‡ *Ibid.*

hypoglossus. The root of the hypoglossus was remarkably disintegrated. The lesion also invaded many of the fibres of the right pneumogastric nerve.

Case 73. Eisenlohr* describes the case of a syphilitic male, aged 30, who first began to complain of pain in both legs. Half a year later there was complete paralysis in the lower, and lameness in the upper extremities. Gradually his legs became subject to spastic contractions, tremor, etc., and he finally died of phthisis. On section it was found that nearly the whole spinal cord, from the oblongata to the sacral region, was in a state of degeneration.

Case 74. Vierordt† contributes the history of a syphilitic female, 35 years old, who began to suffer with pain and atrophy in the lower extremities. Respiration was accelerated, and there was only partial contraction of the diaphragm. Pulse from 130-150, fever, and death from exhaustion. Section developed that she suffered from phthisis. There was also degeneration in the nerve fibres of the vagi and both sciatic nerves. The muscles of the extremities were atrophied.

Case 75. Kahler‡; male, aged 29, came under observation May 26, 1885. Two days before he was suddenly attacked, while sleeping, with a peculiar sensation in the right side of his body, disturbance of speech, and complete paralysis of left arm. On inquiry it was found that three months previously he had acquired a syphilitic sore. Examination showed that at this time the thoracic organs were normal. Motility became impaired in the whole left half of the body, although sensibility remained intact everywhere. He was placed under energetic anti-syphilitic treatment, although he grew perceptibly worse. In August he began to complain of pain in the neck, spine, and thorax. Diarrhœa and ano-

* *Centralblatt f. Nervenheilkunde*, 1887, vol. 10, p. 12.
† *Archiv f. Psychiatrie*, Bd. xiv, 1883, p. 678.
‡ *Prager Zeitschrift f. Heilkunde*, Bd. 8, 1887, p. 1.

rexia set in, and he died the following November. Autopsy showed cheesy nodules and small cavities in both apices, and in the lower lobe of the right lung there were foci of pneumonic infiltration. There was sclerosis of the motor oculi and facial nerves, and the pons, the oblongata, and the cranial nerves were in a state of degeneration.

Case 76. Lewtas* describes a case of progressive paralysis of the cranial nerves, due to syphilis: "A young man, aged 22, complained of intense pain in the right side of his head and cheek, and an inability to walk steadily. The right cheek was swollen, the corresponding angle of the lips lower, and the saliva dripped from his mouth. He had been using remedies for toothache, although his teeth were sound. He spoke thickly and indistinctly, as though his tongue was too large for his mouth. There was such a degree of paralysis of the right facial nerve that a 20-cell Smee battery produced but very slight contraction on that side of the face. He remained in this condition for twelve days after I first saw him. At the end of this time obstinate and incessant vomiting set in. He would retch violently for ten or fifteen minutes at a time, then fall back exhausted, only to begin again after a short interval. In five days this vomiting had ceased, and although no history of syphilis was obtained it was decided to give him potassium iodide.

"One week later no improvement had occurred, but he was rather worse. Vomiting had returned. He now began to complain of deafness on right side and of pain at the epigastrium. On the twenty-third day he was writhing and groaning with pain in the left side of his neck, shoulders and chest. Left arm and leg were in a rigid, semi-convulsed state. He breathed superficially and quickly, emitting a sort of grunt with each expiration. Respiration 32, pulse 92. Little or no air is heard to enter left lung. Crepitation and impaired percussion resonance all over right apex, also

* *Med. Times and Gazette*, 1875, vol. 2, p. 17.

abundant muco-purulent sputum. He now received $^1/_{16}$ grain of bichloride of mercury with each dose of potassium iodide, and in a few days there followed marked improvement. He became almost free from pain in the head, neck and shoulders. Crepitant râles no longer heard over right apex, but the respiratory sounds there are harsh and bronchial. Curiously enough, subcrepitant râles are audible now over the left apex, whereas a few days ago no moist sounds were detectable in that situation. One week later the report was: Steady improvement in nerve symptoms; pain in head quite gone; crepitation over the greater part of posterior surface of left lung, also in front, but less distinctly; no morbid phenomena in right lung. One week after this date he felt sufficiently well to resume his studies, nothing remaining but slight deformity of the mouth and a muco-purulent expectoration."

In conclusion Dr. Lewtas states his belief that in this case there was successive implication of the facial, hypoglossal auditory, and pneumogastric nerves in consequence of syphilitic infection, and that the disappearance of one and all of the signs and symptons under a single plan of treatment tends to show that they all had a common origin in the nervous system.

Case 77. Berger* relates the case of a male, aged 35, who acquired syphilis four years before his admission into the hospital. His neck was long and thin, and the cervical glands enlarged and became indolent. After death, which occurred in a short time, infiltration and caseation of both lungs were found. The left vagus was imbedded in the enlarged glands of the neck.

Case 78. Vierordt† cites the history of a female, 23 years old, who contracted syphilis in 1880, and on October 3, 1882, began to complain of weakness and impaired sensibility in

* *Deutsches Archiv f. klin. Med.*, Bd. 23, 1879.

† *Neurolog. Centralblatt*, Bd. 3, 1883, S. 180.

the lower extremities. After this the knee and plantar reflexes disappeared, and decided paresis of both legs occurred. She developed all the symptoms of multiple neuritis. At the end of November paralysis of the diaphragm set in, accompanied by a high pulse rate, and death occurred on the following 19th of December. Section: Pulmonary phthisis. Macroscopically the nerves were of normal appearance, and the muscles markedly atrophied. Microscopically the spinal cord, on the whole, was normal. The peripheral nerves had undergone intense degeneration. This degeneration was complete in a branch of the gastrocnemius muscle, relatively so in the phrenic and right sciatic. The right bronchial plexus, right vagus, and left sciatic were atrophied.

Case 79. Unverricht* attended a male, 38 years old—infected with syphilis twenty years before—who was found, on July 10, 1887, with paralysis of the face. Mercury inunctions were without effect. He became affected with ptosis, loss of sense of taste, and paralysis of the tongue and laryngeal muscles. Pulse rate from 88 to 140. Pneumonia, which was followed by death, on September 14, of the same year. Section: In posterior mediastinum there was found an elastic tumor of the size of an apple, in which the vagus was imbedded.

Mercury. It has long been known that mercury causes serious lesions of the nervous system, such as tremor, paralysis, etc., but the assertion that a great majority of those who suffer from the effects of chronic mercurial intoxication also fall victims to pulmonary consumption is indeed a startling proposition to most of us. That such is the case, however, may be learned from what is presented herewith, and for which the author is chiefly indebted to the elaborate and invaluable† treatise on this subject by Dr.

* *Neurolog. Centralblatt*, 1888, S. 164.

† "Untersuchungen über den Constitutionellen Mercurialismus," Würzburg, 1861.

Adolph Kussmaul. This work is principally based on the following data: The critical examination of a series of historical observations on industrial mercurialism; the personal experience of physicians who practised among people who were engaged in the mercury manufactures, as in Fürth, Erlangen, etc.; post-mortem investigations, and the personal experience of Dr. Kussmaul. From these the author shall take the liberty of making liberal quotations.

Walter Pope* states that the laborers in the mercury mines of Friaul all become paralytic and hectic, sooner or later, and that their suffering frequently terminates in pulmonary consumption.

Scopoli (1786) noticed that workers in the mercury mines of Idria are suddenly overcome with general weakness and tremor. He saw sufferers who were unable to stand up, or to feed themselves. Among other symptoms of mercury poisoning he observed a poor appetite, impaired voice-power, diarrhœa, rheumatism, cough, asthma, hemorrhages and phthisis. Guelin (1779) states that cattle which grazed near the mines of Idria also suffered from tremor, loss of teeth and general decline.

Ramazzini and the translators of his work on "Industrial Diseases" (1700-1783) state that mercury workers become affected with salivation, diarrhœa, tearing pains in the limbs, dizziness, stammering, convulsions, marked oppression and constriction of the chest, bronchial catarrh, dry cough, asthma, hemoptysis, and phthisis; and that few of these people become old.

Bartholdi (1783) names the following symptoms among those which are present in chronic mercurial poisoning: Ptyalism, diarrhœa, colliquative sweats, arthritic rheumatism, tremors, paralysis, aphonia, convulsions, cough, asthma, congestion and œdema of the lungs, rigors, hemoptysis, phthisis, and premature death.

* "Philosoph. Trans.," vol. i, p. 21, 1665.

In February, 1810, the English war-vessel *Triumph* anchored in the harbor of Cadiz, and one month later a Spanish ship, laden with a cargo of quicksilver was wrecked near by. By means of small boats about one hundred and thirty tons of this metal was saved and brought on board of the *Triumph.* The bags, in which the mercury was contained, became decayed, the metal flowed out, and distributed itself generaliy among the furniture and in the living apartments of the ship. Soon the crew was afflicted with salivation, and within three weeks two hundred of the men were suffering from ptyalism, tremor and intestinal disorders. The mercury was especially poisonous to those who had a predisposition to diseases of the lungs. Three of the men, who always had good health, died of phthisis in a short time. Two others, one of whom had pneumonia, and the other who had always been free from chest disease, developed phthisis, and were left behind in the hospital at Gibraltar. In his remarks on these cases Kussmaul says that the rapid evolution of phthisis in consequence of mercury poisoning is of great importance.

Sundelin (1820) observed the following manifestations in mercury poisoning: Anæmia, tremor, emaciation, even though the appetite remains intact; general weakness, weak pulse, which is often slow and sometimes small and frequent, voice feeble, appetite gradually declining, and, finally, hectic fever and pulmonary consumption.

Burdin (1821) states that individuals who are exposed to the fumes of mercury begin to complain, in the course of a few months, of pain in the joints, debility, irritability, tremor, anæmia, and dizziness. The intellect and memory wane, often an idiotic condition develops, and death from phthisis or apoplexy usually follows.

Mitchell (1831) gives the following characteristics of mercury poisoning: Impediment of speech, emaciation,

tremor, convulsion, constriction of chest, dyspnœa and cough.

Canstatt (1848) collects the histories of thirty-four cases of industrial mercurialism, in twenty-five of which disturbances of the nervous system were present. Among these were tremor, more frequent in the upper than in the lower extremities, paralysis, dizziness, headache, emaciation, rapid pulse in many cases, cough, stitches in the chest and bloody expectoration.

Aldinger (1861), in an inaugural dissertation on mercurialism among the mirror-gilders of Fürth, states that by far the greatest danger from mercury poisoning comes from the fumes, which incidentally arise when this metal is distilled. Another cause of mercurial salivation he attributes to the manufacture, and wearing of shoe soles, out of the leather bags in which the mercury is shipped. According to him, older persons are less liable to be poisoned than those who are younger. The female sex shows a greater susceptibility to it. Scrofula, rickets, and *pulmonary consumption* he finds a common inheritance in the children of those who are engaged in mercury manufacture.

The following personal observations were made by Drs. Mair, Fronmiller and Bäumler on persons who were engaged in the mercury manufactories of Fürth: Tremor, anæmia and accelerated pulse-rate were common conditions. Among seventy individuals it was found that eighty was the lowest pulse frequency. Mercury is believed to induce abortion, or still-births, among female employees and to greatly predispose the offspring of such women to scrofula, rickets, and *pulmonary consumption*. Among other cases Dr. Bäumler cites this remarkable family history: Male; was admitted in 1860, when 69 years old. He became a gilder at the age of 39, and worked at this for twenty-five years, when he was compelled to seek other employment on account of serious mercurial intoxication. After beginning to gild he

was free from all inconvenience for twelve years, but then began to complain of salivation, stuttering, tremor, loss of memory, shedding of teeth, etc. He was married three times, and all his wives followed the occupation of gilding. From the first union there were four children, of whom one died of gangrene of both feet, and the other three and the mother died of pulmonary consumption. From the second union there were two children, who, with their mother, died of pulmonary consumption. From the third union all the children who were born before the mother began the work of gilding were well; while one who was born after this period was a weakling, and died of a cause that is not given, but the mother died of pulmonary consumption.

Table I. shows the ages and sex of 56 persons who died of chronic mercurial intoxication in Erlangen:

AGES.	MALE.	FEMALE.
15—20	—	2
20—30	3	10
30—40	6	7
40—50	8	7
50—60	4	2
60—70	1	2
70—80	2	—
80—90	2	—

Table II. shows the causes of death, and the sex of the 56 persons who are included in table I.

CAUSES.	MALE.	FEMALE.	TOTAL.
Pulmonary tuberculosis	17	20	37
Incipient tuberculosis and pneumonia	—	1	1
Pneumonia. Tuberculosis?	—	1	1
Pneumonia	—	1	1
Pleuropneumonia	1	—	1
Pleuropneumonia nervosa	—	1	1
Pleurisy, serous	1	—	1
Empyema	—	1	1
Suffocative catarrh. Marasmus	—	1	1
Senile marasmus	2	—	2
Tuberculous meningitis	—	1	1
Apoplexy, cerebral	1	—	1
Apoplexy	2	2	4
Varioloid	1	1	2
Scirrhus hepatic	1	—	1
	26	30	56

These tables show (1) that women are more liable to the evil effects of mercury than men are; and (2) that mercurialism has a powerful tendency towards the production of pulmonary consumption and of other diseases of the lungs. Of the 56 persons whose causes of death are here recorded 71 per cent. died of pulmonary phthisis, and altogether 78.57 per cent. died of diseases of the lungs; while, with the exception of two, who died of varioloid, and one who died of cancer, the others died of some form of nervous or wasting disease. By reckoning the general death-rate from phthisis among adults at 18 per cent., this would give us a mortality of the same disease among sufferers of mercury poisoning four times greater.

Comparison of the Effects of Chronic Mercury Poisoning with the Symptoms of Pulmonary Phthisis.

In going over the history of this process of intoxication one cannot avoid being struck with the great similarity between its effects on the human system and the symptoms which are characteristic of pulmonary phthisis. This is so obvious that it cannot be mistaken, and it evidently made a marked impression on Dr. Kussmaul more than thirty years ago, for in his comments on this subject he states (p. 244) that mercurialism sometimes presents to us a complete picture of *phthisis consumata*, with fever, expectoration, sweating and diarrhœa. It is remarkable, indeed, that evidence coming from the many varied sources above quoted should be so direct and unanimous in this respect; and for the purpose of illustrating this still more clearly the author shall, on the basis of this testimony, briefly consider most of these toxic manifestations in their bearing on the main issue.

One of the earliest symptoms of chronic mercurial poisoning, barring tremor, is a sense of *debility* and *exhaustion*, which shows itself in general weakness, shortness of breath,

impaired voice-power, irritability, etc., conditions which are almost universally present in phthisis. *Cough, expectoration* and *oppression of the* chest are noted by most observers. *Emaciation,* even though the appetite remains intact, is referred to by Sundelin and others. The *rigors* and *fever* which are present emanate, in all probability, from the inflammatory disturbances which are set up in the lungs and other organs by the poison. *Colliquative sweats,* occurring independently of fever or of inflammation, have been frequently observed. *Dizziness* or *giddiness* is very common, and often comes on suddenly and unexpectedly. Those who suffer from it liken their feelings to those of alcoholic intoxication. It is generally accompanied by ringing and fulness in the ears, flashes of light and various colors before their eyes, nausea, and vomiting, and may be so severe as to throw them on the ground, or induce unconsciousness. Although the writer does not recollect of ever having seen dizziness described as a symptom of pulmonary consumption by any of the authors who have written on this subject, yet his experience convinces him that this sign is present in a large number of cases of this disease. He has under his care at the present time three patients who show it to a remarkable degree. One of them, the worst, assured him that on a number of occasions he was suddenly overcome with one of these attacks, while walking on the street, and would have fallen down if he had not fortunately grasped some object of support near by. He is not suffering from venereal, alcoholic, or any other form of intoxication, but there is a family history of phthisis and insanity in his case. After being under treatment for three weeks the symptom disappeared entirely. The two others are also free from it at present..

Pain in the chest and extremities. This is a frequent concomitant of chronic mercury poisoning, although without any apparent alteration in the bones, joints or muscles.

Active exercise seems to have no palliating influence on this symptom, and it often becomes aggravated while the patient is lying in bed. Frequently it is confined to one shoulder or to one arm in the beginning, and radiates from here over the whole body. As a rule the upper extremities are more liable to be affected than the lower. Mercury being a nerve poison there can be no doubt that this pain is neurotic in character. We find a similar pain in phthisis independent of that which comes from pleurisy or pneumothorax. Pain in the latter disease is not always confined to the chest, but may extend to the shoulder and arm. It is generally located on that side of the chest on which the lung disease is situated. There is also frequent pain in the lower extremities in this disease, affecting the sciatic and posterior tibial nerves—most often the latter—which in all probability is always a neuritis, although it may partake of a rheumatic nature sometimes. *Hemoptysis* has been found by a number of observers, among which were Scopoli, Ramazzini and Bartholdi. An *irritable* and *rapid pulse* is another accompaniment of this form of intoxication. Disturbance of the *mentsrual flow* is common. This shows itself in suppression of this function, or sometimes in menorrhagia. Women who suffer from mercurial tremor are predisposed to abort, and the offspring which survives is constitutionally feeble and liable to scrofula and phthisis.

Lead. This is another metal which has the power of deteriorating the nervous system, and of producing pulmonary phthisis. In fact it appears that metallic poisons, on account of being more slowly eliminated from the body, are more liable to exert a prolonged destructive action on the nervous system than vegetable poisons. And it is exceedingly interesting to observe in this connection that both mercury and lead are more slowly excreted than the other metals. This is a most plausible reason why these two poisons should be more commonly associated with chronic nervous and pul-

monary diseases than the other members of their class. A few examples of this form of phthisis will now be given, after which the liability of lead workers to this disease will be considered:

Case 80. Carmuset* relates the case of a male, aged 48, painter by occupation, who suffered from lead colic, tremor of tongue and hands, weakness of the muscles and unsteady gait. Tuberculosis of the lungs supervened, which finally caused his death, after which it was found that his brain contained thirty-six mgr. of lead.

Case 81. Oppenheim† gives the following case: Male, aged 33, type-founder, who had for twenty years been working in lead and in lead dust. He frequently suffered from colic, and in 1879 became subject to weakness in the lower and upper extremities. Examination at this time showed typical saturnine extensor paralysis and atrophic paralysis in the lower extremities. He improved under electrical treatment, but became worse in 1884. Stomatitis and brain disturbance set in, and all the changes of chronic lead poisoning manifested themselves. After death, which occurred four days after admission, it was found that he had granular atrophy of the kidney, catarhhal pneumonia and hypertrophy of the heart. Marked degeneration of the posttibial nerves and of the muscles of the lower extremities. The anterior gray horns, and probably the whole spine, was diseased.

In the *British Medical Journal* (August 19, 1893, p. 415) Dr. Robinson gives an interesting account of the liability of the lead miners of Weardale, in England, to pulmonary consumption. It appears that lead mining has been carried on in this region since the year 1401. In the Stanhope division of the region there occurred twenty-three deaths, from 1885 to 1892, among the lead miners, five of which were ascribed

* *Centralblatt f. d. Nervenheilkunde*, Bd. 7, 1884, S. 69.

† *Archiv f. Psych.*, xvi, S. 376.

to consumption, seven to bronchitis, two to injuries, and one each to pneumonia, pleurisy, general tuberculosis, cystitis, chronic gastric catarrh, apoplexy and hemoptysis—fifteen out of the whole number (23) being due to chest affections, or about 65 per cent. Among nine retired old lead-miners, who died in this same division during the last eleven years, eight had diseases of the chest.

Not all the inhabitants of the Weardale region, which is divided into districts—named Bookhope, Eastgate, Stanhope, Fosterly, etc.—are employed in the mining of lead. Some are engaged in quarrying limestone and others in farming, and from the following table we clearly see the enormous difference in the liability to phthisis of these several occupations, the death-rate from this disease in England and Wales, of 1.5 per 1000 living inhabitants, being taken as a standard of comparison. The first three and part of the fourth of the

TABLE SHOWING THE DIFFERENCE IN THE LIABILITY TO PHTHISIS OF PERSONS WHO ARE OCCUPIED IN LEAD MINING, STONE QUARRYING, AND FARMING.

Name of Districts of Weardale.	Average population for seven years.	Average death-rate from pulmonary consumption for seven years per 1000 living.	Chief occupation of the working classes.
Bookhope.	500	4.6	Chiefly lead mining. A few also follow farming.
Eastgate.	220	0.6	Farming.
Stanhope, outside Local Board District.	750	1.4	Farming and limestone quarrying.
Fosterly.	1300	1.3	Limestone quarrying chiefly. Some farming, lead mining, and lead smelting.

districts which are included in this table are almost identical as regards the condition of the houses, the character of the soil and the climate, including rainfall, exposure to winds, range of temperature, etc., hence the chief and perhaps the only factor which yields such a marked variety in the mortality figures of this locality is the mode of employment which these people follow.

In the other lead mining districts of Weardale, viz., in St. John and Deverent, the phthisis death-rate is also excessive. In St. John it was 3.8 in 1891, and 1.99 in 1892; in Deverent it was 3.8 in 1891, and the same in 1892. In the adjoining lead mining district of Middleton, in Teesdale, the death-rate is also high, being 2.8 for 1892. In the adjoining districts of Stanhope, the chief occupation of the inhabitants of which are farming and limestone quarrying, the death-rate from phthisis for the last seven years is slightly below 2.0; while in Wolsingham, in which steel-workers and farmers principally live, it was 0.8 for 1891, and 1.5 for 1892. In Towlaw with its exposed situation, and with a coal-mining population of 4,364, the consumption-rate was 0.9 for 1891, and 1.7 for 1892.

These statistics show, therefore, that pulmonary consumption is from two to three times more prevalent among lead workers than it is among farmers living in the same localities, or among the general population of England and Wales; and that coal mining, a fact to which reference has been made on a previous page, is practically exempt from any such deleterious influences. Moreover, lead is not only capable of greatly increasing the liability to phthisis, but the experience of the author leads him to say that it produces a form of this disease which is perhaps peculiar, because the dyspnœa and other nervous symptoms are more clearly emphasized from the beginning, and because it seems to be less amenable to curative measures than when it is induced by other causes.

Comparison of Alcohol, Syphilis, Mercury and Lead as Factors in the Production of Phthisis.

In these agents we have an interesting group of poisons which possess many properties in common. Excepting the virus of syphilis, their nature is known and understood. Their process of poisoning is chronic in character, they all have a special affinity for and are capable of inducing disturbance in the nervous system, and they greatly predispose those who are suffering from their poisonous effects to pulmonary phthisis. Moreover, when the deterioration which is thus produced does not develop phthisis, it induces many of the symptoms which accompany this disease, as, for example, cough, dyspnœa, rapid pulse, pain in the chest and extremities, weakness, emaciation, dizziness, colliquative sweats, amenorrhœa, etc.

In this respect their action is alike, but as has just been stated, the disintegration which they evoke is of a chronic nature and this they bring about by persistent action, and in quantities which do not bring life into immediate danger. A great deal depends, therefore, (1) on the virulency of the poison, (2) on the amount and the frequency with which it is introduced, and (3) on the facility, or the difficulty, with which it is excreted by the body. In these particulars it will be seen that they vary greatly. Alcohol is eliminated rapidly through the lungs and the kidneys, and would not be attended by such serious danger to the economy were it not for the fact that the chronic "tippler" takes it in frequently repeated quantities, and for a protracted period. In the case of syphilis a single infection is capable of saturating the whole body for a long time. Mercury and lead enter the body gradually, either by being inhaled or ingested, and are eliminated exceedingly slowly. A comparative small quantity of these poisons suffices, therefore, to work grave and irreparable injury in the nervous system and in other vital parts of the human economy.

CHAPTER IV.

ETIOLOGY CONTINUED: THE ACUTE EXANTHEMATOUS AND CONTAGIOUS FEVERS, URICACIDÆMIA, BERIBERI, DIABETES, AND LEPROSY.

THAT most of the exanthemata bear a definite relationship to diseases of the respiratory organs has long been clearly recognized, but whether this is maintained by the direct irritation of the specific virus of these diseases on the lungs, or whether this is brought about indirectly through the instrumentality of the nervous system, is not so definitely established. Evidence will be submitted, however, in these pages which makes it exceedingly probable that the nervous factor which is present in these diseases does not play an insignificant rôle in the evolution of the pulmonary disorders which accompany them, and, as in the instances already noted, it may be the link which binds the one to the other. The diseases which sustain such a relation are typhoid fever, diphtheria, scarlatina, measles, whooping-cough, parotitis, influenza, and cerebrospinal meningitis.

TYPHOID FEVER is a disease which is characterized by certain inflammatory and ulcerative lesions of the solitary and agminated glands of the intestinal canal. It is the product of a specific poison acting on the human system, which, besides affecting the lymphatic glands of the small intestines, also has a deteriorating influence on the nervous system, as will be seen from a history of its symptoms and pathology. James* states that the fibrillar contraction which occurs on tapping the muscle of a weak and debilitated subject is indicative of great nervous and muscular debility, and

* *Pulmonary Phthisis*, p. 50.

that the best examples of this he has met in protracted cases of typhoid fever, although he has seen it pronounced in pulmonary phthisis. Ross* says that numerous nervous disturbances, such as insanity, aphasia, temporary hemiplegia, hyperæsthesia involving a considerable portion of the limbs and trunk, neuralgic pains in the muscles of the limbs, neck, thorax and abdomen, paralysis and degeneration of the tibial and peroneal nerves, paralysis of the abductors of the glottis, and of the external rectus muscle, paraplegia, acute ascending paralysis, progressive muscular atrophy, ataxia, multiple sclerosis and other chronic affections may be established during an attack of or convalescence of typhoid fever. This points out, therefore, that the central, as well as the peripheral nervous system, may become deeply implicated throughout an attack of typhoid fever; and we furthermore learn from the investigations of Dr. A. Lewin† that in twenty-six cases of typhoid fever which he examined he found degeneration of the vagus in every instance.

Now phthisis and other pulmonary diseases are very commonly associated with typhoid fever. Louis made the observation that inflammation of the lungs is more frequent in typhoid fever than in any other acute disease, and that young persons who make an imperfect convalescence from this fever frequently fall into an acute course of pulmonary consumption. Murchison says that an attack of typhoid fever is often followed by destructive disease in the lungs.

Diphtheria. Of all the acute infectious diseases diphtheria seems to be the one which is most frequently followed by sequelæ of the nervous system. These manifest themselves primarily in loss of motion and sensation in the pharynx and larynx, in impairment of taste, and in defective vision due to lameness of the ocular muscles. Later come

* "Diseases of the Nervous System," p. 975.

† *Beiträge zur Pathologie der N. Vagus*, 1888.

numbness and tingling in the fingers and toes, which gradually spread over both the upper and lower extremities. In some cases there is complete loss of power in the legs, which seem to be more prone to become implicated than the arms. Paralysis of the bladder and constipation from paralysis of the abdominal muscles have occurred. Trousseau noticed impotence in young men, and describes a case in which delirium and convulsions existed. The *heart* and *lungs* also become seriously embarrassed through the intoxicating action of the diphtheritic poison on the circulatory and pulmonary nerve supply. Retardation of the pulse and paralysis of respiration seem to be the result, as Ross says (*op. cit.*, p. 977) of a primary irritation followed by paralysis of the vagus. Squire* states that "loss of power and of sensibility in the parts supplied by the par vagum occur at a somewhat earlier period than the paralytic affections of other parts of the body." . . . "Dr. Gull reports the case of a boy, aged 11, who, five weeks from the commencement of diphtheria, was unable to prevent the head falling forward, or to either side, owing to paralysis of the muscles of the neck; he suffered dysphagia, aphonia, and paroxysmal dyspnœa; a few days afterwards the breathing became entirely thoracic. The diaphragm was unmoved in inspiration and depressed in expiration, indicating a loss of power in the phrenic nerves. Death approached rapidly by apnœa."

The two following cases give a clear history of lung mischief being wrought by the poisonous action of the diphtheritic virus on the pneumogastric nerves:

Case 82. Mendel†, male, aged 8, was taken ill with diphtheria, which was followed by paralysis of the muscles of the throat, eye and face, and by hyperæsthesia and coldness of the lower extremities. The pulse was 100, and the temperature 38.5. Physical signs of bronchitis developed, and albu-

* *Reynolds' System*, vol. 1, p. 125.

† Mendel, *Centralblatt f. Nervenheilkunde*, Bd. 8, 1885, S. 102.

min was found in the urine. Death was caused by embarrassed respiration. On section the brain and oblongata appeared normal. There were hyperæmia and extravasation along the vagus and abducens, and degeneration of their fibres.

Case 83. Schech*; a child 7 years old, one month after having passed through an attack of diphtheria, began to suffer from dysphagia and hoarseness, and expelled all forms of nourishment through the mouth and nose in fits of violent coughing. There was complete loss of motion and of sensation in the larynx. Indications of œdema and pneumonia appeared, and death ensued. It was found that the bases of both lungs were œdematous, and in the upper lobe of the left lung there was infiltration. Larynx and trachea were pale. Both inferior laryngeal and vagi nerves had undergone fatty degeneration.

Case 84. (Own experience.) Male, aged 42, who was first seen by author four years ago, when he complained of cough and expectoration, which he had since a severe attack of diphtheria twenty-nine years before. Immediately after this he began to suffer from paroxysms of dyspnœa, which he believed to have been asthmatic, although these disappeared after moving from the country to the city. A year before he came under observation he had a severe attack of diarrhœa, which prostrated him a great deal, and since that time he was more or less feverish, had lost considerable flesh, had a poor appetite, and was very nervous and a poor sleeper. At the first visit his pulse was 96, his evening temperature 100° F., and his urine normal. There was no asthma or lung disease in his family, excepting a paternal aunt, who died from consumption. There was marked consolidation in the right apex, with some crepitation in the same area. Treatment seemed to be of no avail. His fever gradually rose, although never to a very high point; he became thinner,

* *Archiv f. klin Med.*, xxiii, p. 2.

cough and expectoration became aggravated, marked dyspnœa set in, pain in the extremities supervened, and the lung disintegration slowly progressed. Towards the end, which took place in eight months, he became so sleepless and nervous that he could not be controlled, and after exhausting the action of all the hypnotics and sedatives, inhalation of chloroform was resorted to, which kept him quiet. No *post-mortem* investigation was made.

In a paper* of very great scientific interest, Dr. John Jenks Thomas, of the Boston City Hospital, records the histories of seventeen cases of diphtheria, five of diphtheria with scarlet fever, one of diphtheria with scarlet fever and measles, and two of pure uncomplicated pneumonia—25 in all—in which the pneumogastric nerves were examined. In eighteen of these cases, besides the cardiac lesions, the pulmonary conditions are noted as follows: In eleven there was acute pneumonia, in four pulmonary congestion, and in one each there was bronchitis, pulmonary œdema and pleuritis. A short abstract of each case in which complications of a pulmonary character existed is here given.

Case 85. M., æt. (about) 10. Ill three days before entrance. On fifteenth day vomited, became pale, with rapid and very feeble pulse. Respiration rose to 70. No note of paralysis. Death. Heart: Weight 180 gm., enlarged and dilated cavities, aortic regurgitation; congestion of the lungs, liver, kidneys and spleen. No cultures. Pneumogastric nerve intensely degenerated. It was impossible to find a fibre which was not affected. The amount of fat in the nerve sheath and axis cylinder was large.

Case 86. F., æt. 61. Admitted with laryngeal and faucial diphtheria, and bronchopneumonia. Ill two days. On third day she had a sudden collapse, rapid respiration and cyano-

* "Acute Degeneration of the Nervous System in Diphtheria," *Boston Medical and Surgical Journal*, 1898, vol. 138, pp. 76, 97, and 123.

sis. Death. Autopsy. Beginning near the tip of the epiglottis, a thick, dirty gray membrane extended through the larynx and trachea into the bronchi. There were bronchitis and pulmonary œdema. Heart: Weight 330 gm. Valves normal. Pneumogastric nerve: Showed marked degeneration, scarcely any of the fibres being free from changes.

Case 87. F., aged about two and one-half years. Died after an illness of six days. Autopsy: Heart weight 75 gm. Valves and cavities normal. Bronchopneumonia of right lung. Pneumogastric nerve: Considerable amount of degeneration and the presence of a moderate amount of fat in the nerve fibres. The axis cylinders were much beaded and broken. Fully one-half of the nerve fibres showed changes more or less marked.

Case 88. M., aged 2 years. Admitted with laryngeal diphtheria, and lived two days. Cause of death, bronchopneumonia. Autopsy. Heart weighed 80 gm. Valves and cavities normal. Acute bronchopneumonia of upper and lower lobes of right lung. Pneumogastric nerve: A considerable number of fibres were swollen and granular in appearance, and in the greater part of them fat globules could be seen in some part of their course.

Case 89. M., 3 years old. Died in collapse on the thirteenth day of the disease. Autopsy. Heart: Weight 120 gm. Valves and cavities normal. Bronchopneumonia of upper lobe of left lung. Pneumogastric nerve: A considerable number of fibres were stained gray, and of which the myelin sheath was irregular, swollen, granular and broken.

Case 90. Male, nine months old. Lived six days. No paralysis. Autopsy. Heart: Weight, 50 gm. Valves and cavities normal. Bronchopneumonia. Pneumogastric nerve showed marked degeneration. Most of the fibres contained fat drops, some of them in large numbers.

Case 91. M., 6½ years old. Albuminuria. Death on tenth day. Autopsy. Heart: Weight, 105 gm. Fibrinous

pleurisy and pulmonary emphysema. Pneumogastric nerve showed a moderately extensive degeneration. A majority of the nerve fibres had swollen and broken myelin sheaths, with a considerable amount of fat present.

Case 92. M., aged 6 years. Albuminuria. Death on forty-first day, at which time paralysis of the palate was noted. Autopsy. Heart weighed 115 gm., and was normal. Pulmonary œdema. Pneumogastric nerve showed slight degeneration. A few fibres were much swollen, irregular in outline, beaded and stained grayish.

Case 93. F., aged 64. The total duration of the case was about three weeks. Death was due to extension of the membrane. Autopsy. Heart weighed 210 gm. Valves normal. Bronchopneumonia. Lung culture, Klebs-Löffler bacillus and pneumococcus. Pneumogastric nerve: A large majority of the fibres grayish in color, granular, and some intensely swollen. In the affected fibres the axis cylinders could not usually be made out.

Case 94. M., aged five years. Duration of disease, fifteen days. Four days before death the pulse was irregular and weak. Autopsy. Heart: Weight, 120 gm. All cavities much dilated. No endocardial changes. Auriculo-ventricular valves were dilated. Slight increase in pericardial fluid. Congestion and œdema of lungs. Pneumogastric nerve: A large proportion of the nerve fibres affected to a greater or less extent. Most of the fibres were swollen and grayish, and in many of these considerable amounts of fat were present, both in the nerve sheath and in the axis cylinders.

The next five cases were of diphtheria, accompanied by scarlatina or measles.

Case 95. F., aged 7. Admitted on second day of the disease, with diphtheria. On the ninth day the rash of scarlet fever appeared. Cultures from throat positive. Pulse rapid. Temperature 103-104°. Acute nephritis. Death. Autopsy: Pericardial effusion. Pulmonary congestion

and œdema. Pneumogastric nerve: An occasional degenerated nerve fibre, with replacement by fat drops, and disappearance or beading of axis cylinders; but the sections were in the main normal.

Case 96. M., aged 3. Entered the hospital with measles and diphtheria. On the eighth day the child became cyanotic and died. Heart: Weight, 80 gm. Valves and cavities normal. Bronchopneumonia. Pneumogastric nerve markedly degenerated. About one-half of all the fibres showed the presence of fat globules in the myelin sheath, which was broken, irregular and swollen. The axis cylinders were swollen, beaded, broken and occasionally could not be made out.

Case 97. F., aged 14 months. A case of measles and diphtheria, which on the fourth day became complicated with scarlet fever. Death on the tenth day. Autopsy. Heart: Weight, 33 gm. Valves and cavities normal. Croupous pneumonia and pleurisy of left lung and bronchopneumonia of right lung. Cultures showed no pneumococci. Pneumogastric nerve was markedly degenerated. Many of the fibres contained fat globules, and the sheaths were swollen and irregular. The axis cylinders of these fibres were absent or beaded.

Case 98. M., aged 3 years. Admitted with diphtheria, and on the second day eruption of scarlet fever appeared. On the twentieth day general clonic convulsions supervened and death followed. Autopsy. Heart: Weight, 55 gm. Left ventricle dilated. Valves normal, except the mitral, which was incompetent. Pulmonary congestion and œdema. Pneumogastric nerve was markedly and extensively degenerated. Scarcely any fibre could be found which did not show the presence of fat globules. The myelin sheaths were swollen and broken, as were also the axis cylinders.

Case 99. Male, aged 10 months. Child had measles and was admitted with diphtheria, still having the measles erup-

tion. In four days pneumonia developed, and he died on the following day. Autopsy. Heart: Weight, 50 gm. Valves and cavities normal. Double bronchopneumonia. Cultures showed nothing but Klebs-Löffler bacilli, streptococci and staphylococci. Pneumogastric nerve showed extensive degenerative changes, though not exceedingly intense. Almost all the nerve fibres showed the presence of fat, but in rather moderate amount, and confined chiefly to the medullary sheath, which was fragmented and swollen. The axis cylinders were grayish in many instances, and swollen and beaded.

Case 100. M., aged 2 years. On admission diphtheria bacilli were found in his nose. No scarlet fever, but he developed measles, bronchopneumonia and gangrene of the face and jaw, and died on the thirtieth day of the disease. Autopsy. Heart: Weight, 50 gm. Valves and cavities normal. Double bronchopneumonia. Necrosis of soft tissues of upper and lower jaw and cheek, of inferior and superior maxillary bones, and of the soft tissues of the nose, palate, pharynx, uvula and tonsils. Culture showed streptococci and staphylococcus albus. Colon bacillus and a few Klebs-Löffler bacilli in the soft tissues of upper and lower jaw; few streptococci and Klebs-Löffler bacilli in lower lobe of right lung, many streptococci and few Klebs-Löffler bacilli in middle lobe of right lung, and the pus from the lungs showed pus cells, streptococci, and a few short bacilli. Pneumogastric nerve: Marked and extensive degenerative changes. The nerve fibres were much swollen and beaded. Very few fibres seemed normal except for a short distance. The axis cylinders were beaded, broken and replaced by fat drops.

Case 101. M., aged 11 months. Was admitted with pneumonia. Was reported to have had eleven convulsions the day of entrance. There were no diphtheria, scarlet fever or measles present. Sudden death on the ninth day. Autopsy.

Heart: Weight, 65 gm. Valves and cavities normal. Hepatization of lower lobe of left lung. Cultures; heart blood, lower lobe of left lung, liver, spleen and kidneys sterile. Pneumogastric nerve. In some nerve bundles scarcely a single nerve fibre could be seen. In others the degenerated fibres were approximately one-fourth of all the fibres. The degeneration was marked. There was much fat present in the medullary sheaths and in the axis cylinders.

MEASLES is another acute eruptive disease which is very liable to be followed by disease of the pulmonary organs. According to Copland there is a special tendency to consumption in those who suffer from its attack after having passed the period of puberty. Ringer* asserts that "acute tuberculosis, or chronic phthisis, may occur during the course of the disease, but it usually first gives evidence of its existence after the fever has declined. Acute tuberculosis follows measles more frequently than any other of the acute specific diseases, whooping-cough being, perhaps, excepted." Some attribute the lung changes to an extension of the inflammatory condition along the bronchial tract to the pulmonary tissue, while others, among whom is Ruehle, incline to the belief that they are due to infiltration and caseation of the bronchial glands which are involved in the process. The two following cases appear to show that compression of the vagi, by enlarged bronchial glands incidental to measles, are the cause of pulmonary phthisis.

Case 102. Basevi† reports the following case: Female child; was well until the spring of 1876, when she had an attack of measles, since which time she was subject to cough and bronchial catarrh. In the fall of 1876 she was admitted into the hospital, when she had infiltration of the right apex behind, and there were mucous räles distributed over the remainder of her chest. Death ten days after admission,

* *Reynolds' System*, vol. 1, p. 198.

† *Jahrbücher f. Kinderheilkunde*, 1878, S. 414.

when a cavity was found in the upper part of her right lung. There were enlarged lymphatic glands in the mediastinum, which compressed the œsophagus. The right vagus was situated between the hypertrophied glands and the œsophagus.

Case 103. Barlow* saw a male, 7 months old, who was suffering from lung disease since he had measles. After death it was found that his right lung contained a large cavity and that the right vagus was very much compressed by a bronchial gland of the size of a Spanish chestnut. The left lung was also somewhat affected.

WHOOPING-COUGH. Most authorities are agreed that whooping-cough is essentially a nervous affection, that its virus has a special affinity for that part of the nervous system which co-ordinates the function of the respiratory organs, and that of all the acute contagious diseases it is the one which is most liable to be followed by disorder of the lungs. Dr. Copland writes: "I believe that the disease is chiefly nervous in simple cases; that it preserves this character more or less throughout, even when inflammatory complications ensue, and that in the uncomplicated state the nervous affection never proceeds beyond irritation. . . . The inflammatory appearances in the oblongata and base of the brain may be owing to the functional relation of these parts to the respiratory order of nerves which receive the first impression of disease." Hufeland, Hoffmann, Wendt, Walshe and Puldame ascribe the principal seat of whooping-cough to irritation of the pneumogastric nerves, and Guibert to that of the general nervous system. Dr. Edward Smith,† in his article on whooping-cough, says: "Without denying the existence of a specific poison, and without admitting that the supposed poison is eliminated by the mucous membrane of the bronchi, we do not doubt that that feature which gives character and importance to the disease is the nervous or

* *Trans. London Pathological Soc.*, vol. 30, p. 254.

† *Reynolds' System*, vol. i, p. 55.

spasmodic one, and that in any uncomplicated case, when this has been abated, the disease is shorn of its specific characters and dangers."

The pulmonary pathology and morbid anatomy of whooping-cough is, therefore, to be sought with the greatest interest, inasmuch as it shows the manner in and the extent to which pulmonary changes may follow in the wake of an influence operating solely through the nerve supply of the lungs. Here, if anywhere, should be found the strongest evidence in favor of the neurotic theory of the origin of pulmonary disease, for it must be clear that whatever the nature of the lung disintegration may be in any case of whooping-cough it must be a pure sequence of disordered innervation.

The evidence goes to show that in all severe cases of this disease there is congestion of the pharyngeal, laryngeal and bronchial mucous membrane, as well as of the lungs, together with dyspnœa and feebleness of the respiratory sounds. There may also be a shade of dulness in some parts of the lungs. The heart's action is weak, the pulse is rapid, and emaciation and exhaustion are constant symptoms. Epistaxis and hemoptysis occur generally. Emphysema, chronic bronchitis, bronchopneumonia and phthisis are frequent complications, especially in the offspring of those who bear a history of chest disease.

MUMPS, as a rule, is a mild specific disease, but when complicated the author is convinced that in some way it is a forerunner of pulmonary consumption. One example of this especially impressed itself on his mind. It was that of a boy, 16 years old, who, while suffering from this disease, was caught in a rain storm. Directly after this he experienced a severe pain in his left chest and in his left testicle. The latter organ did not swell, but its pain, as well as the pain in the chest, was almost wholly relieved by hemoptysis. From that time on he began to cough and expectorate, and to show physical evidence of disintegration of the left lung,

and died from phthisis before he was quite twenty years old. His mother died of the same disease, and he was the youngest of a family of five children,

Bartholow* says that "during the late war the cases of mumps were accompanied by high fever, often delirium, and by great depression of the vital powers; pneumonia was a not infrequent complication, and those who recovered had a tedious convalescence, the blood being much impoverished and the body emaciated. . . . In some persons, the subjects of a dyscrasia, the morbid condition is awakened from its dormant state by an attack of mumps. The tubercular diathesis is the most common of these."

How is the awakening of the dormant phthisical dyscrasia, to which Dr. Bartholow refers here, brought about? Is it by the action of the poison of this directly on the lung, or through some other avenue—possibly through intoxication of the pneumogastric nerve?

Case 104. The following case, which is reported in *The Medical and Surgical History of the War of the Rebellion, Part Third, Med. Volume, p. 675*, would seem to have some bearing on this question: "Sergeant B., aged 26, was admitted March 21, 1863, with slight diarrhœa and pain, redness, heat and swelling in the region of the parotid. A poultice of arnica leaves and flaxseed was applied, and in a day or two the abscess communicated with the external auditory canal. On the 27th a free incision gave exit to six ounces of pus. On April 2d he was restless, but became quiet after the administration of hyoscyamus and opium. He was found dead in bed on the morning of the 3d. *Post-mortem* examination: The mastoid portion of the temporal bone was denuded and carious, and its cells filled with pus. The deep vessels and *nerves*† were completely dissected by the progress of suppuration. The right ventricle of the heart

* *Practice of Medicine*, 1st ed., p. 773.

† Italics mine: author.

contained a fibrinous clot. The left lung, its apex especially, was studded with miliary tubercle. The liver was large, but healthy; the gall bladder full; the bowels filled with flatus."

INFLUENZA. Whatever the precise etiology of influenza may be it must be recognized that essentially it is a disease of the nervous system, and that all its varied outward manifestations are but an expression of the disturbance which exists in the nervous system below. The organs which are most commonly involved in this manner are the lungs, the heart and the intestinal canal; and the synonyms *peripneumonia notha, peripneumonia catarrhalis, pleuritis humida, febris catarrhalis, catarrh pulmonaire, defluxio catarrhalis*, etc., which have been used by the older writers, while failing to define its specific nature, are clearly descriptive and expressive of these phases of this disease.

Among the early symptoms of influenza are severe and often excruciating headache, drowsiness, delirium, vertigo, bluntness of the senses, cold feeling along the spine, rigors, fever, sometimes profuse sweating, pain in the neck, shoulders, arms, back and legs, great lassitude and extreme prostration. Then comes a paroxysmal cough, which is sometimes dry, but frequently attended by stringy, tenacious and bloody expectoration, hoarseness, dyspnœa, and threatened suffocation. The pulse may be strong at first, but soon becomes soft and feeble, and may be very slow; nausea and vomiting may be present at the beginning, but diarrhœa usually sets in later. In some cases jaundice supervenes, and sugar or albumin may appear in the urine.

The morbid anatomy of influenza is principally seen in the meninges of the brain, in the spinal cord, peripheral nerves and in the lungs and heart. The brain and spinal cord are congested, hemorrhagic spots occur in the latter, and in the lateral ventricle, and degenerative changes take place in the axis cylinders and nerve fibres. Pulmonary œdema, bronchopneumonia, capillary bronchitis and pleurisy are its frequent

accompaniments. Frequently collapse of a lung, or a part of a lung, supervenes. The muscle of the heart wall is soft, and easily ruptured.

The pulmonary disease was believed by Graves to be due to paralysis of the vagi. Walshe* says that "this poison seems specially to exercise its influence on parts supplied by the pneumogastric nerve; and it is worthy of remark that whooping-cough, an affection in which that trunk is indubitably concerned, has often been noticed to prevail concurrently with influenza."

Influenza may, therefore, be regarded as a disease which, on account of its power to undermine the vitality of the nervous system, and especially that of the pneumogastric nerves, engenders various bodily derangements, among which lesions of the lungs, heart and intestines are met most frequently.

Cerebrospinal Meningitis is, as its name implies, a disease which involves the higher nerve centers, and manifests a strong tendency to concentrate its poisonous action on the base of the brain and the oblongata. Its pathologic anatomy, in so far as these regions are concerned, chiefly consists in congestion and in exudation of lymph in the cerebellum, pons and oblongata, and in their investing membranes, and in paralysis of the cranial nerves. Of the 105 cases of this disease reported in *The Medical and Surgical History of the Rebellion*† it is stated that "the *post-mortem* appearances of the cerebrospinal axis were very similar in all of them. . . . The parts most frequently and extensively affected by the deposit were the base of the brain, the pons varolii, cerebellum, oblongata, the spinal cord and the sulci between the convolutions of the cerebral hemispheres."

That which is of the greatest concern to us in connection with these morbid appearances is the fact that the essential difficulty in cerebrospinal meningitis resides in that special

* "Diseases of the Lungs," p. 563.

† Part iii, p. 592.

part of the nervous system which is directly related to the structure and function of the respiratory organs. Here, as in the case of whooping-cough, we should find evidence of pulmonary embarrassment if there is any truth in the proposition that lesions of the lungs are dependent on disease of their nerve supply. How, then, do the pulmonary organs fare during the progress of this disease? Radcliffe* states "that death chiefly occurs from asphyxia, caused by damage to the respiratory nerve centers." In the same volume (p. 507) he makes the following quotation from Dr. Burdon-Sanderson, who observed an outbreak of this disease on the Lower Vistula: "In all severe cases, whether of children or adults, the breathing was embarrassed in proportion to the general gravity of the symptoms. This embarrassment was marked by a slow, labored inspiration, followed by quick respiration and a long pause. In all the fatal cases which came under my notice the most prominent symptoms which preceded death were those which indicate impairment and perversion of the respiratory function. As the breathing became more hurried and difficult, the general depression became more intense, the pulse became weaker and quicker, and the temperature of the skin more elevated." In *The Medical and Surgical History of the War of the Rebellion*† we further learn from the *post-mortem* appearances of the recorded cases that "the lungs were more frequently the seat of morbid changes than any other organ or tissue, excepting the cerebral membranes. Reference is made to their condition in seventy-four of the ninety fatal cases the records of which have been preserved. In twenty of these they were healthy, in fifty-four altered. Generally only one lung was affected, but more frequently both were implicated in the congestion, extravasation and inflammatory changes that constituted the morbid condition.

* *Reynolds' System of Medicine*, vol. i, p. 511.

† Part iii, p. 599.

Congestion characterized the rapidly fatal cases; pneumonitic processes were more frequent in those that ran a protracted course."

From this description it is obvious, as has been intimated already, that the element of time determines very largely the degree of pulmonary perturbation which obtains in cerebrospinal meningitis. In swiftly fatal cases this may not amount to more than a passing congestion, or a functional depression; but in protracted cases it may end in deep-seated inflammation and disintegration. It is highly probable, therefore, that in all severe cases of this disease, as Dr. Burdon-Sanderson has found in those which he observed, the lungs are embarrassed, but the process of disease may be so transient and ephemeral in some cases as not to have time to impress any morbid, visible changes on these organs, which would account for the absence of *post-mortem* appearances in some of the bodies which have been examined.

Another very interesting relation which goes far in itself towards establishing the close causative affiliation between cerebrospinal meningitis and pneumonia must not be overlooked in this connection. This is the frequent coexistence of these two diseases, and the remarkable similarity which exists between their nervous symptoms and morbid conditions, as will be learned from the following observations: In the discussion of the pathology of cerebrospinal meningitis in *The Medical and Surgical History of the War of the Rebellion** it is stated that "the occurrence of cerebrospinal, or spotted fever, may be referred to the fulminant operation of that miasm which produced pneumonia under ordinary conditions. Viewing pneumonia as a specific constitutional disease, with a local lesion in the lungs, it becomes connected causatively with cerebrospinal meningitis. . . . This connection will be found to be no mere theoretical idea, for in the instances in which the medical records of the war show an

* P. 608, part iii.

epidemic prevalence of pneumonia, with a corresponding intensity of the febrile poison, the so-called cerebrospinal meningitis was present at the same time. Surgeon Russell, who reported fifty cases of cerebrospinal meningitis, in January and February, 1864, reported also the great prevalence and fatality of pneumonia—784 cases—of which 156 proved fatal, having been received into hospital during the months from January 1st to April 30th of the same year." In a foot-note on the same page it is remarked that "Juergenssen refers to the frequency of the association of an epidemic meningitis with pneumonia, and cites Immerman and Heller as having recently called the attention of physicians to this point. Out of thirty autopsies in cases of pneumonia they found nine in which meningitis was also present. It was ascertained beyond question that an epidemic of cerebrospinal meningitis was prevailing at the same time."*

Résumé. From the above considerations it appears that typhoid fever, diphtheria, scarlatina, measles, whooping-cough, mumps, influenza and cerebrospinal meningitis have the following characteristics in common: (1) They owe their existence to the operation of specific poisons on the human organism; (2) these poisons exert a decided destructive influence on the nervous system; (3) that over and above this general influence on the nervous system they show a well-defined affinity for the special nerve tract which controls the respiratory organs; and (4) that pulmonary diseases, and especially phthisis, are some of their most constant sequelæ.

This tendency to provoke pulmonary disorder is strongly pronounced in whooping-cough, influenza and cerebrospinal meningitis, which are all professedly regarded as diseases which specially implicate the respiratory nervous centers. It is true that the pulmonary embarrassment which is engend-

*Cited from "Ziemssens, Cyclopædia," American edition, vol. v, p. 115.

ered by these processes is not always that condition which is known as pulmonary consumption, but the position which will be maintained in this work, and which will be made more clear when the pathology of this disease is considered, is that one form of pulmonary disease develops into another, and that if the exciting causes continue it is the inevitable tendency of all acute pulmonary derangement to terminate in phthisis. That which is capable of producing pulmonary irritation has, therefore, the power to set up a possible phthisical condition.

The general trend of the action of these poisons, as has already been shown in the case of alcohol, syphilis, mercury and lead is to undermine the integrity of the nervous system and to produce pulmonary disease in an indirect way. Owing to their manner of action, however, these two groups of poisons differ from each other, the former—as has been stated—bringing about a comparatively rapid intoxication of the nervous system, and induce a crop of pulmonary diseases which are more or less acute in character; while the latter, owing to their slower action, are more prone to bring about a crop of pulmonary disorders which are more or less chronic in character.

URICACIDÆMIA. Notwithstanding the opinion which one hears expressed sometimes that rheumatism and gout are antagonists to pulmonary consumption, it seems that the uric acid diathesis is in some way closely connected with, and is probably indirectly responsible for, the latter disease in many instances. No reference is here made to those cases of phthisis which are more or less dependent on valvular lesions of the heart originally caused by rheumatism, but to those in which there is an active and painful manifestation of the rheumatic poison in the joints, more or less constantly present thoughout the course of the disease. An affinity between asthma and bronchitis, on the one hand, and gout and rheumatism on the other, is well recognized by eminent authori-

ties* who have written on the gouty and rheumatic aspects of these diseases, and it is quite probable that the relationship between pulmonary consumption and gout and rheumatism is equally well assured. Indeed there is strong evidence for believing that the close alliance that exists between the above-named diseases is brought about through the toxic action of uric acid on the nervous system. For, according to Dr. Alexander Haig,† of London, whose studies on uric acid as a cause of disease have opened a most fertile field in practical medicine, a number of nervous diseases, like migraine, epilepsy, hysteria, convulsions, etc., are frequently produced by uric acid poisoning, and in view of such an action it is not very difficult to conceive that uric acid may play an important part in the causation of pulmonary consumption.

Beriberi, Diabetes and Leprosy.

Although differing somewhat from the two groups of diseases which have just been considered, there is another group which bears an analogy to them, the leading characteristics of which will now be discussed. The members of this group are beriberi, diabetes and leprosy, diseases in every one of which the nervous system is compromised and in which disorders of the thoracic organs play a prominent rôle.

Beriberi is an endemic disease of Asia, Africa and America, also known under the names of kak-ke, hydrops asthmaticus, paraplegia mephitica, sero-phthisis perniciosa endemica, neuritis multiplex endemicâ, and pan-neuritis endemica. Scheube, who has written extensively on this subject, believes that it is an infectious, but not a contagious, disease.

* See following works: "On Chronic Bronchitis especially connected with Gout," etc., by E. H. Greenhow, M.D., 1869. "Chronic Bronchitis, Its forms and Treatment," by J. Milner Fothergill, M.D., 1882.

† "Uric Acid in Causation of Disease," Alexander Haig, M.D., London, 1896.

It is claimed that its characteristic micro-organism has been discovered, and that the principal features of the disease have been generated through its inoculation. Wernich holds it to be a disorder of general nutrition, calls it sero-phthisis perniciosa, and thinks it depends on food which is deficient in fat and albumin, or on inherited weakness of the vascular system and on climate. It is most prevalent in damp weather and variable temperature, and between the ages of sixteen and twenty-five. Females enjoy a relative immunity. The Japanese in large cities are specially liable to it, and particularly teachers, students, priests, clerks, merchants, or those who follow indoor and sedentary occupations. Among the predisposing causes are taking cold, getting wet, exhaustion of body and mind, emotional excitement, excesses, etc.

The disease begins to manifest itself by a heaviness and lameness in the lower extremities, which are often preceded by fever and catarrh of the bronchi, stomach and intestinal canal. These symptoms are followed by anæsthesia and pain, as well as by œdema of the legs, by cardiac palpitation, dyspnœa, gastric oppression, indigestion, paralysis, etc. The paralysis generally confines itself to the lower extremities, although it may extend to the face, tongue, pharynx and larynx. Effusion into the large serous cavities frequently takes place, which is associated with diminished excretion of urine.

Looking at the morbid anatomy of beriberi a little more closely it will be found that the brain and spinal cord are almost wholly exempt, while the peripheral nerves, the heart, and the lungs and the serous sacs are principally involved in this disease. According to Scheube, and other authors, the peripheral nerves, and especially those of the lower extremities, become diseased first and undergo very marked degenerative changes. The vagus, with its cardiac, pulmonary, laryngeal and pharyngeal branches, also become implicated. The heart dilates and hypertrophies, and its muscular fibres

undergo fatty degeneration. Endarteritis follows in the aorta and large blood-vessels. Generally the bases of the lungs are hyperemic and œdematous, and the bronchial tubes are filled with a frothy fluid. Emphysema of the apices is also present. The acute distention of the lungs, which has been observed by Scheube, is ascribed by him to degenerative changes in the vagus. This author further states that "the morbid changes in the vagus and its branches are never so pronounced as they are in the peripheral nerves, which is not surprising when we consider the vital importance of this nerve and that death must ensue before degeneration of a similar degree could occur in this as in other nerves." Pekelharing* and Winkler state that the peroneal nerves are attacked the earliest in this disease, and that lesions of the vagi and laryngeal nerves immediately threaten life. Baelz also found deterioration in the fibres of the pneumogastric nerves.

Dr. W. Gilmore Ellis, Medical Superintendent Government Lunatic Asylum, in Singapore, where beriberi has been endemic for years, examined a large number of nerves which were removed from patients who had died from this disease† and found the following markedly degenerated: The phrenic, branches of the cardiac and pulmonary plexuses, the splanchnics, branches of the solar and renal plexuses, branches of the mesentery, and vasomotor branches of the aorta, renal, splenic and tibial arteries. In no case of death from this disease did he fail to find degeneration of either the phrenic or pneumogastric nerves showing that death invariably occurs in this disease from implication of some of these nerves. The phrenic nerve is most frequently involved.

In conclusion he says it seems that beriberi is a most recoverable disease so long as the pneumogastric, phrenic, and branches from the sympathetic ganglia in the neck, are unaffected, but that when these nerves, all or any of them, once become attacked death is imminent.

* *Deutsche med. Wochenschrift*, 1887, No. 39.

† *Lancet*, Oct. 15, 1898, p. 98.

What, then, is the termination of beriberi? From all accounts it appears that in recent years its death-rate has been greatly reduced, and that at present this reaches only about four or five per cent. According to Scheube, if death occurs in acute cases it comes through paralysis of the heart or diaphragm, and in chronic cases, which are complicated with typhus, dysentery or pulmonary consumption, death comes from general exhaustion. It is frequently associated with intermittent fever, acute articular rheumatism, pleurisy and pulmonary and laryngeal tuberculosis. In reference to phthisis, Scheube states that "consumptive patients contract beriberi, or in the course of beriberi the patient becomes consumptive, I have observed not only once but in a whole series of cases." On this same point Baelz says that his experience in Tokio leads him to believe that phthisis pulmonalis is one of the most frequent complications of beriberi.

From the evidence which has been submitted it is clear that the same principle underlies the genesis of pulmonary disorder in beriberi as in those diseases which have been previously considered, *viz.*, that of an embarrassed pulmonary innervation. Indeed this is not only shown to be true of the lungs, but also of the heart. The nature and trend of beriberi in serious cases being thus established, it is important in connection with one of the main issues of this work to inquire briefly into the means which have been resorted to in alleviating this disease from a preventive standpoint. This is of especial importance in view of the fact that the disease is accompanied by a specific microbe to which, it is believed by bacteriologists, to owe its existence. On the other hand, Wernich contends that it is a constitutional nutrition-disease, and that it depends on poor or defective food; and the following crucial experiment, which has been instituted by the Japanese Government, an account of which is given in *The Boston Medical and Surgical Journal* for September 15, 1892, p. 271, entirely corroborates this opinion:

"The Japanese Minister of State for the Navy has recently published a short review of the preventive measures taken against beriberi in the Japanese Navy. These were so eminently successful, and resulted so completely not only in eliminating the disease from the navy but also in improving the general health of the service that Japanese naval officers are justly proud of it. Director-General Takaki, in 1880, noticing the great difference between the number of beriberi cases on board of ship and in barracks, came to the conclusion that the food supply was responsible for a large amount of the disease. A few years later, by personal application to the Emperor, he obtained the appointment of a special committee. As a result, in the year 1884, the dietary of the vessels of the navy was entirely remodeled, the principal changes being in the substitution of nitrogenous for non-nitrogenous food; the amount of animal food was much increased, barley and other grain, condensed milk, and other more or less nitrogenous foods substituted for rice. A war vessel had just arrived from a long cruise in South America, and had, during 271 days, 160 cases of beriberi in a crew of about 350. Another vessel with the new dietary was despatched for the same cruise the next year, and on this voyage the conditions, with the exception of the food being practically the same, the number of cases fell to 16. In the navy, as a whole, the number of the cases of the disease dropped suddenly after the improvement in the scale of the diet. In 1882 there were 1900 cases in 4700 men; in 1883, 1200 cases; in 1884, 100 cases; in 1885, 41 cases, and since then the disease has become practically extinct. The improvement in the general health of the men has also been very noticeable. . . . Surely no better argument is needed to show that beriberi is essentially a disease of malnutrition."

DIABETES.

Diabetes, or glycosuria, is a disease which is accompanied by the presence of sugar in the urine. There is always a certain amount of sugar in the blood, but, according to Bernard, if this does not exceed one-third of one per cent. it is not excreted by the kidneys. Too much sugar in the blood depends principally on increased production and partly on diminished oxidation of the same. When we look for the experimental influences which bring about an excessive production of sugar we find that first and foremost comes division of the vasomotor nerves between the oblongata and the liver. This has the effect of dilating the hepatic blood-vessels and probably of interfering with the normal metabolism of the hepatic cells. Injuries of the head, disease of the cerebellum and oblongata, puncture of the fourth ventricle, irritation of the central end of the vagus, an epileptic or a hysterical fit, division of the anterior column of the spinal cord between the oblongata and the fourth cervical vertebra, irritation of the liver or intestines by needles, electrodes, etc., injection of alcohol into the portal vein, irritation of the sciatic nerve, inhalation of ether, chloroform, amyl nitrite, nitro-benzol, etc., either by direct or reflex action on the vasomotor center in the oblongata, cause a dilatation of the hepatic blood-vessels and an increase of sugar in the blood similar to that which occurs when the vasomotor nerves, which supply the hepatic blood-vessels, are divided. This means that any abnormal impulse which is transmitted to the vasomotor center, either through the cerebrospinal nervous system or through the pulmonary, hepatic, gastric or intestinal branches of the pneumogastric nerves, has the power of inducing diabetes.

That which is of special concern here is (1) that the vasomotor center is profoundly involved in the process of sugar production; (2) that this center has very close anatomical

relations to the respiratory center; and (3) that of all the lower nerve centers the respiratory center is more impressible to and seriously affected by nervous impulses than any other. There are good physiologic grounds for believing, therefore, that any disease which implicates the vasomotor center would also be likely to affect pulmonary innervation and in consequence of this produce disease of the lungs.

Let us see what clinical medicine has to say on this point. Brunton* states that "pneumonia, gangrene of the lungs, and phthisis often occur, the latter being the usual termination of diabetes." Dr. Copland† asserts that he scarcely ever met a case of diabetes that was entirely uncomplicated with pulmonary disease. Bouchardat‡ says that in nineteen *post-mortem* examinations of diabetic patients he found tubercles in every instance. Ancell§ states that "tuberculosis and diabetes are by no means infrequently associated together, and the greater number of cases of diabetes become complicated with tuberculosis pulmonalis before death. In a discussion on the morbid anatomy of diabetes before the London‖ Pathological Society Dr. Findlay said that he and his colleague, Dr. Copland, had searched the *post-mortem* records of the Middlesex Hospital for a period of twenty-six years, and found particulars concerning twenty cases of diabetes, all of which, except two, had marked pulmonary and pleuritic lesions. Eleven of these were phthisical. The lesions of phthisis were often obviously of such old standing as almost to compel the conclusion that the diabetes arose in the subjects of tuberculosis, and not that the latter was secondary to the former.

In the same discussion (p. 355) Dr. Stephen Mackenzie

* Article "Diabetes Mellitus," *Reynolds' System*, vol. v, p. 390.
† Dictionary of Medicine, vol. i, p. 508.
‡ *Etiologie de la Tuberculisation pulmonaire*, 186, p. 4.
§ "Treatise on Tuberculosis," p. 602.
‖ See *Trans. Lond. Path. Soc.*, vol. xxxiv, p. 336.

reports thirty-seven cases of diabetes from the death records of the London Hospital during a period of nine years (from 1874 to 1883), twenty of which gave evidence of pulmonary phthisis. Dr. Mackenzie said: "From the above table it will be apparent that in many cases there were other conditions present, besides the diabetes that may have had an influence in bringing about the death of the patient. In some instances it is difficult to determine the share such conditions took in causing or being caused by diabetes. Some, as phthisis pulmonalis, and renalis, and gangrene of the foot, are no doubt the outcome of diabetes. . . . From a study of this series of fatal diabetic cases two facts stand out prominently: The tendency of this disease to bring about phthisis, and to terminate in coma.

"As to the dependence of the phthisis on the diabetes it seems that no doubt can be well maintained. I know of no other general disease which is so frequently succeeded or complicated by phthisis. It would seem, indeed, that the natural mode of termination of diabetes is by inducing phthisis, but that in a large proportion of cases this stage is only partly reached, or not reached at all, owing to the patient being prematurely cut off by coma."

At the same meeting of the Pathological Society Dr. Frederic Taylor (p. 371) reported fifty-three cases of diabetes which occurred in Guy's Hospital during a period of nine years, fourteen of which were phthisical and one pneumonic.

Frerichs* says "one of the most frequent complications of diabetes is pulmonary phthisis, with its usual characteristics of peribronchitis, cheesy infiltration, excavation and pneumothorax."

Dr. R. T. Williamson† gives an abstract of 23 cases of diabetes which had the following terminations: Phthisis 8, coma 6, syncope 1, not given 8.

* "On Diabetes," p. 79.

† *Medical Chronicle*, May, 1897.

The morbid anatomy of diabetes is not always well marked, but next in importance to the changes which occur in the liver are those of the nervous system and especially of the oblongata, the vagi, and the various branches of the sympathetic. Brunton (*op. cit.*, p. 392) states that "the oblongata has been found to be affected in a number of instances, sometimes by degeneration, sometimes by the pressure of a tumor, sometimes by clots or by inflammatory softening. Dickinson has noticed dilatation of the anterior and of the perivascular spaces. . . . The sympathetic in the abdomen has been observed to be much thicker than normal, and the semilunar ganglion and splanchnics have been found thick and cartilaginous. Atrophy of the cells in the solar plexus has also been noticed. The vagi have been observed to be thickened in some instances and in others atrophied from the pressure of concretion. The meninges of the brain have been found congested, adherent, or œdematous, and tuberculous deposits have also been discovered in them. Induration, hemorrhagic infarcts, softening, and tumors have been observed in various parts of the brain, in the cerebellum and in the pons."

The two following histories are typical examples of implication of the oblongata and vagus in diabetes:

Case 105. Weichselbaum* relates the following case of a male, 34 years old, admitted May 14, 1880, and who reports that he had been weak and sickly for two years. Increased excretion of urine for six months, which contained 3.4 p. c. sugar. Died October 31st, and examination showed chronic tuberculosis of both lungs, with cavities, and sclerosis of the vagus and glossopharyngeal nuclei.

Case 106. De Jonge† reports the history of a male, 37 years old, who was admitted with symptoms of pulmonary tuberculosis and anasarca. He was markedly emaciated, had

* *Wien. med. Wochenschrift*, 1881, S. 914.

† *Arch. f. Psychiatrie*, S. 658.

increased thirst, and his urine contained 6 p. c. of sugar and a trace of albumin. He died shortly after admission, and it was then found that besides pulmonary phthisis there existed a tumor about the size of a bean, reaching from the left olivary body to the first cervical nerve, and which pressed on the parts beneath.

The points of special interest here concerning diabetes are (1) that it is characterized by an excessive amount of sugar in the blood; (2) that such an overproduction of sugar occurs in disease, or injury of the vasomotor center, or of the vasomotor nerves which supply the hepatic blood-vessels; (3) that the vasomotor center is in close anatomical relationship to the respiratory center; (4) that, therefore, disease of the one may readily lead to disease of the other; and (5) that phthisis frequently follows or accompanies diabetes is well substantiated by clinical experience.

Leprosy.

Leprosy is regarded as a cutaneous disease, although its ravages are not confined to the tissues of this structure. It is generally divided into the *anæsthetic* and the *tubercular* forms; yet both forms are merely different stages of the same process, varying only in the intensity of certain of its manifestations and frequently become mixed in the same individual. The premonitory symptoms of both forms are general *malaise*, debility, mental depression and drowsiness, which are often associated with rheumatic-like pains in the extremities, hyperæsthesia, chills, and fever. These are followed by pain along the course of the peripheral nerves, numbness of the skin, maculæ, bullæ, muscular atrophy, and later by ulceration and necrosis of the skin, mucous membranes, subcutaneous connective tissue, periosteum, bones, and of some of the internal organs.

The principal difference between the two varieties is in the

onset of the attack and in the mode of eruption in each. In the *anæsthetic* variety, after the above-described symptoms may have lasted for nearly a year, spots of a pale-yellow color, of the size of an inch or two in diameter, and not raised above the surface, are noticed. These spots usually correspond to the distribution of a nerve, and evidence of nerve disease exists sometimes before the eruption appears* Then the nerve swells and can often be felt through the skin. The most frequent sites of these eruptions are on the back, shoulders, posterior parts of the arms, nates, thighs, around the knees, elbows, and the face. In from two to three years after the beginning of the disease the edges of these spots become raised, and then enlarged. The hands and feet now become devoid of feeling, bullæ, and ulcers, which are conical in shape, form, with blackened edges, and exude a thin, unhealthy pus. The ulcer's apex extends down to the bone, which becomes necrotic, and can eventually be removed with the forceps. These ulcers are very prone to occur on the soles of the feet of people who walk barefooted. When the bone is near the surface, as in the ankles and wrists, or in the hands and feet, the destructive process reaches the periosteum, the bone is laid bare, caries begins, and a large portion of the affected bone may be destroyed. Sometimes the ulceration enters articular cavities, and gives rise to inflammation, continued suppuration and to spontaneous amputation. The phalanges, and then the meta-carpal and metatarsal bones are most liable. This form may last from eighteen to twenty years.

The *tuberculated* variety is characterized by an eruption of tubercles, and, after the above-described premonitory stage,

* For the information which I have obtained of this disease, I am chiefly indebted to the following works:

Hillis, "Leprosy in British Guiana."

Bidenkap, "Lectures on Leprosy."

Carter, "Report on Leprosy in Norway."

Danielssen and Boeck, "Traite de la Spedalshed." (*Author.*)

erythematous, reddish-brown patches, in size from a shilling to a saucer, appear on the skin. After the eruption subsides, tubercles of the size of a pea form on the site of these patches. There is now also diminished cutaneous sensibility in the patch, although before this there is hyperæsthesia. These patches become elevated and œdematous. Successive crops of tubercles break out, which are preceded by pain and aching in the limbs, and accompanied by febrile attacks. After a tubercular eruption the fever and malaise usually disappear, but often one eruption, with fever, succeeds another for a very long time before a remission appears. The tubercles soften, ulcerate, and abscesses form in the skin, which destroy the deeper tissues—bone and cartilage—in a manner similar to those which occur in the anæsthetic variety. The mucous membrane of the throat, mouth and nose is attacked, and eventually that of the larynx. The tubercular infiltration in these parts is reddish, and develops slowly. The vocal cords usually have a tuberculated and uneven appearance, and the voice is hoarse.

From this survey of the manifestations of leprosy it is seen that the peripheral nervous system plays an essential part in the production of this disease. In reference to this Ross* states that" the early occurrence and severe degree of anæsthesia in the tubercular variety of leprosy, as well as the manner in which it progresses from the periphery towards the central parts of the affected limbs, would alone indicate that disease of some part of the nervous system is a prominent part of the affection. Characteristic anatomical changes have also been found, both in the nerve trunks and in the spinal cord and brain, although it is probable that the alterations in the nerve centers are of secondary importance."

Hillis states that the anæsthetic form essentially consists in a disease of the peripheral nerves, which become swollen at intervals. These swellings usually occur in those parts of

* "Diseases of the Nervous System," vol. i, p. 213.

the nerves which are most exposed to mechanical influences, changes of temperature, etc. The nerve changes from its normal white color to a grayish semi-translucent appearance, and occasionaly assumes a smoky tint. There is a deposit of inflammatory or granular cells around the nerves. Danielssen believes that the inflammation in the sheath of the nerve gives rise to the deposit, Hillis believes the reverse, and Charcot that it is a neuritis. Danielssen found that the smaller cutaneous branches of nerves running to the affected spots in the skin were red and swollen, especially the neurilemma. Later in the disease these nerves had a more browinsh color, especially the perineurium, which became tumefied and compressed the nerve fibres. In old cases swelling of the nerves could be traced as far as the brachial plexus and the sciatic nerve. At last the nerves lost their swelling, became yellowish and atrophied, and the axis cylinder disappeared. Virchow confirms this view of the pathology of the disease. Bidenkap says that leprosy is an effection of the peripheral nerves (partial perineuritis), which manifests itself by numness of the skin, maculæ, bullæ and atrophy of the muscles. Charcot* states that "it is above all in anæsthetic leprosy that we encounter in their full development the trophic disorders which we have studied in connection with traumatic nerve lesions. The first of which includes eruptions of various forms, but chiefly those characterized by vesicles and bullæ, the second pemphigoid eruptions. . . . Here we see the pemphigoid bullæ developing with great rapidity, and reappearing from time to time on different parts of the tegumentary system supplied by the wounded nerve. Brown-Séquard's experiments on guinea-pigs, etc., showed that ulceration of the toes, and loss of nails, etc., took place after section of the sciatic nerve."

Leprosy being, therefore, fundamentally an affection of

* "Lectures on Diseases of the Nervous System," New Syd. Soc. p. 26.

the nervous system, what relation does it bear, if any, to pulmonary consumption? Hillis* states that "lung complications occurred in seventeen per cent. of his cases of tuberculated leprosy, and with regard to phthisis as a complication it would be interesting to study how far, if at all, tuberculosis in general may be related to lepra. May the one disease be, as it were, lit up by the other? I confess I am not in a position to discuss this matter, and that I can only state, in the cases referred to in the table, it is believed there was a predisposition to phthisis." Bidenkap says that "lepers are frequently attacked by common tuberculosis, which then becomes the cause of death. The relation is remarkable and not easy to explain if we, as is universally believed, consider tuberculosis a specific infectious disease."

The points of interest in leprosy are: (1) that although it is regarded as a cutaneous disease it is essentially an affection of the peripheral nerves; (2) the fact that nerve disorder is capable of giving rise to erythema, bullæ, maculæ, tubercular eruptions, sloughing and ulceration of the skin and subjacent tissues, and (3) that its subjects are very liable to pulmonary consumption.

* *Op. cit.*, p. 39.

CHAPTER V.

ETIOLOGY CONTINUED: IMPLICATION OF THE PNEUMOGASTRIC NERVES IN DISEASES OF THE SPINAL CORD AND CEREBROSPINAL NERVES.

THIS section comprises the histories of cases which illustrate the genesis of various forms of pulmonary disorder through implication of the pneumogastric nerves in disease of the cerebrospinal nerves.

MULTIPLE NEURITIS. Cases of phthisis will be related under this heading which in their course become complicated with multiple neuritis, especially of the extremities, and also others in which the phthisical affection showed itself secondarily to the inflammatory condition of the peripheral nerves.

Case 107. Vierordt* describes the case of a male, 42 years old, who died January 14, 1883, of phthisis. No syphilitic infection. Eight days before he died he compained of tearing pains and coldness in the legs, while they were quite warm. Examination showed emaciation, paresis and partial anæsthesia of lower extremities, and knee reflex wanting. Section, beside the pulmonary disintegration, showed typical degeneration of the fibres of the right tibial and vagus nerves. No other nerves were examined. Spinal cord was normal. Vierordt, in commenting on this case and on some others, remarks that "on the whole nothing decided can yet be said as to the association between pulmonary phthisis and peripheral nerve degeneration, but the indication of such a possi-

* *Archiv f. Psych. u. Nervenkrankheiten*, vol. xiv, 1883, p. 678.

bility is so apparent that a further investigation is demanded."

Case 108. Pitres et Villard* relates the history of a female, 28 years old, who suffered from paræsthesia and laminating pains in both legs for half a year before she died of phthisis pulmonalis. Section showed degeneration of the vagi, as well as of the peripheral nerves.

Case 109. Remak† saw a male, aged 37, iron worker, suffering from laryngeal and pulmonary tuberculosis. There was a high degree of stiffness in the muscles of both shoulders and arms, without any marked affection of the shoulder-joint. This rigidity had been gradually developed in a year. No difference in the electric irritability of the nerves or muscles. Trousseau's phenomena was missing. The tendon reflexes in the upper extremities, and in the muscles of the neck, were very much exaggerated, but less so in the jaw reflex. A frank paralysis of the muscles was not present. An organic affection of the nervous system must be excluded in this case. Remak states that muscular rigidity and exaggerated tendon reflexes exist in certain nervous diseases, in articular rheumatism, in traumatic and idiopathic joint affections, and in hysteria, and accepts the view that the exaggerated muscle tonus and tendon reflexes in this case are to be classified with the various peripheral affections of this sort and belong, perhaps, to such a peripheral neuritis as is found in pulmonary tuberculosis.

Case 110. Rosenheim‡ contributes the case of a male, aged 35, coachman, always well, excepting an intestinal catarrh the previous summer, who found himself unable to stand on the morning of November 11, 1885, on account of weakness and sleepiness in his legs. This subsided, but re-

* *Revue de Med.*, 1886, p. 193.

† *Neurologisches Centralblatt*, 1890, p. 338.

‡ *Archiv f. Psychiatrie u. Nervenkrankh.*, vol. xviii, 1887, p. 782.

turned on the following day. On the fourteenth day an examination showed impaired vesicular breathing over his whole chest and mucous râles in left interscapular region. Heart sounds dull and somewhat distant. No fever. Pulse 100. No albuminuria, expectoration profuse and puriform. Both legs of a blue-red color, and feel somewhat cool below the knee. Pressure or pinching of the skin in lower extremities produces pain. Plantar and cremaster reflexes present. Pressure over both peroneal nerves produces pain. On the 15th lameness occurred in the arms; 17th, tubercle bacilli in sputum. After this there was dyspnœa, right pupil larger than left, irritation of the larynx on swallowing, emaciation, inability to expectorate, pulse 116-120, morning temperature 38°C, evening temperature 39°C, marked attack of dyspnœa, profuse perspiration, respiration 45, orthopnœa, râles over the whole chest, and death on the 27th of the same month. Section: Large and small cavities in both lobes of left lung. Gray-white miliary nodules, and cheesy nodules in upper lobe of same lung. Lower lobe of right lung hepatized. Bronchial mucous membrane red, and bronchi filled with mucous secretion. Brain and spinal cord normal. There was marked degeneration in both sciatic and vagi nerves and brachial plexuses.

Case III. Senator* admitted a male, aged 27, May 31, 1886, whose previous health was good with the exception of an attack of gonorrhœa two and a half years before. In the winter of 1885 and 1886 he was attacked with pain in the back, and felt slightly weak on walking. At this time he also began having some cough and dyspnœa, but was able to follow his occupation until ten days before his admission, when he was too weak to stand. His lungs became implicated, his legs were paralyzed, and his temperature rose to 39.2° C., his pulse to 130-140, and he died of dyspnœa June 28th, following. Section: Pulmonary phthisis, spinal cord

* *Zeitschrift f. k. Med.*, vol. xv, p. 61.

slightly abnormal. Nuclear degeneration and complete disappearance of axis cylinder of sciatic nerve in points, other muscles and nerves also degenerated. The anterior and posterior spinal roots of the sacral nerves were normal. The vagi were not examined.

Case 112. Freud admitted a male, aged 18, a baker, October 3, 1884, who had been in good health until a week before, at which time he was attacked with drawing pain and persistent coldness in both legs, oppression of the chest, dyspnœa, headache, pronounced prostration, and some œdema of the lower extremities. Examination showed a powerfully built and well-nourished frame. Patient was pale, temperature normal, but no albuminuria or diabetes existed. There was a systolic cardiac murmur. His condition gradually became worse, and he died December 17, 1884. Section: Pneumonia of both lungs, and a large amount of pus in the bronchial tubes. Left trigeminus and vagus were gray, red and inflamed, and their fibres were degenerated.

Case 113. Stewart† relates the following case: Male, aged 31, hotel keeper, admitted November 8, 1880, complaining of general weakness, pain, stiffness, and loss of power in hands and legs, and confused vision. These symptoms began to develop early in the previous August. There was no girdle pain, nor nystagmus, but sensibility to heat, tickling, and pain was very much impaired in the legs. He became drowsy and his memory imperfect, although on the 17th of November he was reported to be improved somewhat, but a few days later he was seized with croupous pneumonia and died December 4th. On *post-mortem* examination both lungs were found to be much congested, and the lower lobes in a state of red hepatization. The vessels of the dura mater contained a medium amount of blood. The arachnoid space at the vertex was very œdematous. The oblongata, in the

* *Wiener med. Wochenschrift*, 1886, p. 168.

† *Ed. Med. Journal*, 1881, p. 868.

neighborhood of the corpora olivaria, was slightly indurated There was no disease of the brain, but the spinal cord in the cervical and lumbar enlargements were apparently secondarily degenerated. The median, ulnar and tibial nerves were notably diseased. After some extended comments on this case, as well as on two others of the same kind which recovered, Dr. Stewart adds: "The rapidly fatal result of the pneumonia in this case shows that the superaddition of any acute disease must be regarded as most formidable where this disease exists. Should I meet with another case of this kind *I* should investigate most carefully the condition of the nerves supplying the inflamed part.*"

Case 114. Kast† relates the history of a female, aged 13, who suffered from multiple neuritis, and death from pneumonia occurred nine months after she was first attacked. On *post-mortem* investigation it was found that the vagi, the other cranial and the peripheral nerves were degenerated.

In an interesting and original research Dr. Jappa‡ investigated the pathologic changes of the peripheral nerves of the bodies of fifteen persons who died of pulmonary phthisis. The nerves examined were the sciatic, post tibial, internal plantar, crural, internal saphenous, superficial peroneus, median, ulnar, radial, cutaneous med. (in upper third of arm), r. interosseous and radials. There were no manifestations of any nervous symptoms during life other than those which are usually found, such as some undefined neuralgias and muscle pains, general hyperæsthesia, etc. The results of this investigation are as follows: In every case most of the above-named excised nerves showed marked degenerative changes, which existed in the axis cylinder and perineum, as well as in the sheath of Schwann. The intensity of the morbid changes was greater in the peripheral endings than

* Italics mine: author.

† *Deutsches Archiv f. k. Med.*, xi, 1886, p. 41.

‡ *Neurolog. Centralblatt*, Bd. 7, 1888, S. 425.

in the trunks of the nerves. The spinal cord was examined in twelve cases and found to be normal.

The points to be especially borne in mind concerning multiple neuritis are:

1. That the brain and spinal cord are not markedly implicated, although some form of pulmonary disorganization is very prone to develop in this disease.

2. That either the vagi or some other part of the respiratory nervous apparatus is involved in most, if not in all, cases.

3. That the lower extremities are more profoundly affected than the upper in this disease, which is also the case in pulmonary phthisis.

4. That multiple neuritis follows in the wake of pulmonary phthisis, but that the reverse is also shown to be true.

5. That some of the leading features of case Number 110 remind one very strongly of a case of acute miliary tuberculosis of the lungs.

6. That an intimate relationship is believed to exist between the nervous affection and the pulmonary disintegration of this disease is evident from the above-quoted remarks made by Drs. Vierordt and Stewart.

Locomotor Ataxia.

While this is principally an affection of the posterior columns of the spinal cord, and so impairs and abolishes the general co-ordinating power of the body, it also has a tendency to invade the region of the respiratory center and nerves, and to bring on and to terminate in some form of pulmonary disease. This is well illustrated by the laryngeal and bronchial "crises," which are known to occur so frequently during its course. On this point Ross* says "very frequently some intercurrent affection, such as typhus, pneumonia, diphtheria and phthisis causes a fatal termination"; and Bartholow†

* *Op. cit.*, vol. ii, p. 237.

† "Practice of Medicine," p. 569.

states that "the most frequent intercurrent malady is phthisis, for we find that in a collection of forty-three cases thirteen were terminated by consumption, four by broncho-pneumonic inflammation, two by enteritis, three by typhoid fever, etc. The following histories confirm this proclivity of locomotor ataxia.

Case 115. Sakaky* admitted a female, aged 38, July 15, 1878, who was well until three years before, when she was attacked with vomiting and violent headache. She became subject to tremor, weakness in the lower extremities, insomnia, poor appetite and great irritability. Death occurred in July, 1883, and on section phthisical cavities were found in the right lung, but the left lung was normal. There was degeneration of the posterior columns of the spinal cord.

The vagi were not examined, but all the other peripheral nerves which were investigated showed marked degeneration.

Case 116. Oppenheim und Siemerling† admitted a male, aged 41, September 27, 1877. In 1873 he began to suffer with tearing pains in the feet and back, and weakness in the legs, etc. He was under observation until his death, which occurred December, 1884. Section: Cheesy degeneration and adhesion of both apices, atrophy and degeneration of the fibres of the right vagus.

Case 117. *Ibid.*,‡ a male, 38 years old, was admitted August 6, 1881, and died June 7, 1885. He suffered from all the characteristic symptoms of locomotor ataxia and pulmonary phthisis. On *post-mortem* investigation the right vagus was found to have been atrophied.

Case 118. Oppenheim§ saw a female adult afflicted with

* *Archiv f. Psychiatrie u. Nervenkrankheiten*, Bd. 15, 1884, p. 584.

† *Op. cit.*, Bd. 18, 1887, S. 125.

‡ *Ibid.* p. 145.

§ *Ibid.*, Bd. 20, S. 1.

tabes dorsalis, who also suffered from accelerated pulse, spasmodic vomiting, laryngeal paresis, dyspnœa, dysphagia, etc. After death extensive changes were found in the oblongata and in the vagus and glossopharyngeal roots. There was also phthisical degeneration of the lungs.

Laryngeal Disease in Locomotor Ataxia.

In order to demonstrate the intimacy which exists between the phthisical process and the nervous system still further it is very interesting in this connection to dwell on the disturbances of the larynx, which often precede and accompany locomotor ataxia, since the same disorders are frequently associated with the phthisical process. In doing this the author has taken the liberty of drawing quite extensively on a critical review of this subject, contributed by Dr. Burger to the *Manchester Medical Chronicle* for May, 1894 (p. 144). In this paper the author collected all the cases of laryngeal manifestations in locomotor ataxia which were published up to the time of its appearance, which he classifies as follows: (1) Disturbance of co-ordination in the respiratory function of phonation—a true ataxy of the vocal cords; (2) spasmodic affections, the so-called "laryngeal crises"; (3) motor paralysis of laryngeal muscles; (4) parasthesia, or anæsthesia of the laryngeal mucous membrane.

These symptoms may occur at any stage of locomotor ataxia, but are often present at its very beginning. The apparent rarity of cases of laryngeal lesions in this disease is due to a frequent neglect of an examination of the larynx, and probably also to the fact that in many cases of paralytic affections of the larynx the possibly accompanying symptoms of tabes, such as absence of the tendon reflexes, Argyll Robertson's phenomena, etc., are overlooked.

(1) *Laryngeal Ataxia.* Of all the laryngeal symptoms

which accompany this disease an ataxic condition of the vocal cords was first recognized. As described by Fournier, in 1885, it shows itself as follows: "Suddenly and unexpectedly the patient loses his voice or at least loses the power of articulating, and the voice becomes thick, dull and discordant, as though the vocal cords did not vibrate in unison." In other cases the voice is jerky or interrupted by vocal intervals, and Burger points out the analogy of this disturbance with the ataxia of the extremities, in which an intended movement is often interrupted and only accomplished after many attempts.

Krause* was the first to observe this interrupted movement of the vocal cords during adduction and abduction. He describes the cords as being suddenly approximated, then remaining still in a semi-adducted position, and then approximated in the median position. During abduction the cords, after being driven together with great force, recoiled into the most extreme inspiratory position.

(2) *Laryngeal Crises.* These were first described by Féréol, in 1869, and, according to Gowers, they are of almost as frequent occurrence as the "gastric crises" in this disease. Burger thus describes an attack: "Suddenly the patient feels a peculiar sensation of tickling or irritation in his larynx, or an intense burning, or a feeling of dryness or of a foreign body in his throat. This sensation then changes to one of stricture of the glottis, and the patient gets fearfully anxious, the respiration becomes labored and painful, with noisy inspiration. Suddenly a severe attack of coughing occurs, closely resembling an attack of whooping-cough, and the patient becomes excited from fear of suffocation. The face becomes cyanotic, the cervical and facial veins are swollen and the conjunctivæ are injected. Then, after a period varying from a few seconds to five or ten minutes, the cough ceases without any, or only a slight, expectoration

* *Neurolog. Centralblatt*, 1885.

and the danger is over." Sometimes the inspiratory spasms is the most prominent symptom, the cough being less troublesome, the respiration being noisy and laborious, and inspiration unduly prolonged. In another case an attack of sneezing precedes the crises, or salivation or singultus accompany it. Occasionally the attacks are attended by vertigo, profuse perspiration, lightning pains in the extremities, pain in the back, chest, etc. Fournier describes the case of a young man who had a cough of this nature for more than a year before the diagnosis of locomotor ataxia was made. Another patient had been suspected of suffering from whooping-cough, and had been isolated from the rest of her family before the real nature of her disease was recognized.

(3) *Paralytic Affections of the Larynx.* In a total of 84 cases of laryngeal paralysis in locomotor ataxia there were 57 cases of abductor paralysis—46 bilateral and 11 unilateral—showing that this form is the paralysis *par excellence* of this disease.

Pathologic Anatomy. Post-mortem examination of locomotor ataxia with laryngeal symptoms are recorded in nine cases. In all these lesions were found in the oblongata. The alterations were a continuation of the well known sclerotic changes of the posterior columns of the spinal cord, foci of degeneration in the nuclei of the spinal accessory and vagus, in the posterior pyramidal tracts, corpora restiformia, etc., and on the floor of the fourth ventricle. Besides the central lesions there was found degeneration in the peripheral laryngeal nerves, in the vagus, and in the recurrent nerve—in the latter usually more advanced than in the former.

Résumé. The important bearing of the subject of locomotor ataxia on the main question is as follows:

1. That this disease is an affection of the spinal cord.
2. That it implicates the oblongata and the pneumogastric nerves.
3. That it frequently terminates in pulmonary disease.

4. That the laryngeal disturbances, which are common in its course, are the sequences of the extension of the disease to the laryngeal branches of the pneumogastric nerves; and,

5. That aside of the fact that locomotor ataxia frequently terminates in disease of the lungs, its laryngeal manifestations bear a striking resemblance to the symptoms which accompany phthisis pulmonalis without locomotor ataxia.

CHAPTER VI.

ETIOLOGY CONTINUED: IMPLICATION OF THE VAGI IN MULTIPLE SCLEROSIS, BULBAR PARALYSIS, AND TUMOR OF THE PONS AND OBLONGATA.

As in the case of locomotor ataxia, so in the diseases which are named in the heading of this section, the strong tendency is towards implication of the respiratory centers and the production of pulmonary disintegration. This is illustrated by the following examples:

Case 119. Koppen* describes the case of a male, aged nearly 50, who was received February, 1876, suffering from multiple sclerosis. In the following April he was attacked with right-sided pneumonia, complicated with bloody expectoration, delirium, incontinence of urine and fæces, bed sores, and died April 9, 1883. Section: Multiple cerebrospinal sclerosis. Old phthisical degeneration in left and pneumonia in right lung. The oblongata, especially at the seat of the pyramidal decussation, as well as the cervical and dorsal nerves, were degenerated.

Case 120. *Ibid.*†, male, aged 40, had paresis of the lower extremities, and all the symptoms of cerebrospinal sclerosis since 1879. He died in April, 1884, in consequence of pulmonary inflammation. Section showed multiple cerebrospinal sclerosis, croupous pneumonia and old phthisical degeneration in the lungs and the beginning of parenchymatous nephritis. The foci of disease were in the pons, oblongata, and cranial and dorsal nerves.

* *Arch. f. Psych. und Nervenkrank.*, Bd. 17, 1886, p. 63.

† *Ibid.*

Case 121. *Ibid.*,* female, aged 42, suffering from multiple sclerosis, was received April 18, 1883. She had formication in her feet for seven years before, had been confined to her chair and bed for three years, and had insufficiency of sphincter ani for one year. Death. Section: Phthisical degeneration in both lungs, cerebrospinal sclerosis, with foci of disease in oblongata, especially at the root of the vagus and in the spinal cord.

Case 122. Guttmann† records the case of an adult female, who received an injury on her head, which was followed by total anæsthesia of the right side of the body, and by a loss of the sense of smell, taste and hearing on the left side. She finally died of phthisis pulmonalis, and it was then found that she also had multiple sclerosis. Spinal cord, oblongata, corpora quadrigemina and vagal nuclei were degenerated.

Case 123. Adamkiewicz‡ saw a female, aged 30, who suffered from bulbar paralysis and died from an attack of dyspnœa and cyanosis. Section showed bronchitis and degeneration of vagi, glossopharyngeus and accessories.

Case 124. Bleuler§ gives the history of a male, 11 years old, who was received April 28, 1881. He had pleurisy in the spring of 1880, from which he recovered, but since then complained of stitches in the chest, palpitation of the heart, headache and diarrhœa. He also became subject to pain in the face, twitching of the eyes and partial facial paresis. Father died of phthisis. No history of syphilis. He gradually became worse and suffered from marked motor and sensory disturbances and from pulmonary disease, and died August 6th of the same year. *Section:* Phthisis of both lungs. Tumor in pons, measuring 2½ by 1 cm., which

* *Ibid.*

† *Centralblatt f. k. Med.*, Bd. 1, p. 393.

‡ *Centralblatt f. d. Nervenheilkünde*, Bd. 3, 1880, p. 168.

§ *Deutsch. Archiv f. k. Med.*, Bd. 37, p. 527.

pressed the olivary bodies forward and downwards. The nuclei of the right vagus and glossopharyngeal were diseased.

Case 125. Freund* first saw a male, aged 47, on December 11, 1882, when he was emaciated, pale, and suffered from all the characteristic symptoms of bulbar paralysis. At that time his temperature was normal, respiration 24, and his pulse 100, but regular. In February, 1883, he became worse; he had fever, his respiration rose to 40 per minute, his pulse ranged from 92 to 150, and death followed on the 19th of the same month. *Section* showed bronchopneumonia, and degeneration of vagus and hypoglossus roots.

Case 126. Maier† describes the case of a female, aged 48, who was well previous to March, 1868, but after that time she had difficulty in chewing and swallowing. She was admitted June, 1869, when she complained of pain in the occiput and legs. She died a year after her admission. Section: Bronchopneumonia and disease of the vagus, hypoglossus and accessories.

Case 127. Bulz‡ gives the history of the case of a female, aged 58, who died from bulbar paralysis. After death it was found that she had suffered from pneumonia and that there was degeneration of the left vagus, glossopharyngeal and hypoglossal nerves.

Case 128. Joseph§ describes the case of a male, aged 22, who had for six years intermittent headache, vomiting and loss of consciousness. There was also slight paresis of the glossopharyngeal and abducens nerves. After death a tumor was found in the fourth ventricle, which compressed the oblongata. Pneumonia also existed.

* *Ibid.*, p. 470.
† *Virchow's Archiv*, Bd. p. 1.
‡ *Archiv d. Heilkunde*, 1872, p. 192.
§ *Zeitschrift f. klin. Med.*, Bd. 16, p. 349.

Case 129. *Ibid.**, male, aged 30, was affected with pain in the neck, swelling of both hands, constriction of the chest, difficulty in speaking and in swallowing, and paralysis of the vesical sphincter and of the left upper extremity. Pneumonia developed, and death occurred. A sarcomatous tumor was found in the fourth ventricle, which compressed the oblongata.

Case 130. Glynn† admitted a male, aged 31, March 18, 1887, complaining of loss of power in his legs and arm, and of difficulty in breathing. He was temperate in all things, had no history of family taint or violence or exposure. He was a well-formed and healthy looking young man. His earliest troubles appeared to have been slight vomiting, about six weeks before admission. Fourteen days before he entered the hospital he noticed some numbness of the fingers of the right hand, and a few days afterwards there was numbness of the calves and weakness of legs and arms. The tongue suffered with the arms, which also felt swollen and numb. On the day of his admission he could not stand alone.

Examination showed no disturbance of intellect, no loss of memory, but there was cutaneous anæsthesia on right side of body; sight tolerably good, pupils somewhat dilated, hearing and smell normal, voice was weak and thick, and there was some difficulty in swallowing. Patellar reflex was absent, cremaster normal, epigastric and hypogastric slightly exaggerated. Respiration was purely thoracic and twenty-eight per minute. He also had a weak, coarse cough. On March 19 he coughed continually, had profuse expectoration of clear mucus, great difficulty in breathing, a tendency to the formation of tracheal râles, cramp-like pains in the legs and twitching of facial muscles. His respiration was 32 and pulse 100. There was neither albuminuria nor glycosuria. March 20th, tracheal râles and moist sounds over his

* *Ibid.*

† *Liverpool Medico-Chirurgical Jour.*, vol. 7, 1887, p. 428.

whole chest, and he seemed to be dying of suffocation. Small doses of atropine ($^1/_{100}$ gr.) had the effect of abating this temporarily. On the 24th there was sugar in his urine, his respiration increased to 56, he became very cyanotic, and died. After death a small gliomatous tumor was found in the middle line of the oblongata, at the level of the calamus scriptorius, which the author thought implicated the nuclei of the vagi, the accessories and the hypoglossals.

Case 131. Ewald* had charge of a male, aged 51, who suffered from weakness of the left side of his body, which was associated with marked swinging of the left arm and leg. Head was turned towards the right. Nystagmus and dizziness existed, and he finally died from phthisis pulmonalis. It was then found that he also had a tumor of the size of a pea in the pons, under the tubercula quadrigemina.

Case 132. Ross† admitted a male, aged 8, February 17, 1880. He was well nourished and in full control of his mental faculties. He complained of headache, and the occipital region was tender. Both eyeballs were inverted. Right pupil larger than left. Slight facial paralysis on right side extends to the eyelid. No loss of taste, deafness, paralysis of the tongue or of the extremities. The head was turned towards the left when the child stood up. He had a staggering gait, and manifested a constant tendency to fall towards the right. On admission the heart and lungs were normal. The urine was also healthy, and so were the reflexes. On the 9th the left half of the body became paralyzed, the power of articulation impaired, and there was difficulty of swallowing. He gradually became worse and died on the 20th from arrest of respiration. *Post-mortem* examination exposed a tumor projecting from the floor of the fourth ventricle on a level with the striæ medullares. It pressed forward into the

* *Virch. u. Hirsch Jahresbericht*, Bd. 12, 2, p. 91.

† "Diseases of the Nervous System," vol. ii, p. 376.

substance of the oblongata and pons. A few scattered miliary tubercles were found in the apices of the lungs. Excepting the changes in the oblongata and pons there was nothing abnormal in the nervous system.

Résumé. The important points to be borne in mind in connection with this part of the subject are (1) that the oblongata comprises within its sphere the center which controls respiration, and (2) that any disease of the nervous system which tends to implicate this center, or which attacks it primarily, as in the above-related cases of bulbar paralysis and of disease and compression of the pons and oblongata, disintegration of the pulmonary organs almost certainly supervene.

Respiratory Paralysis.

It is generally held that the heart is more vulnerable to disease and injury than the lungs, but on the whole it may be said that if these two organs are exposed to the same prejudicial condition, the lungs show less resistance than and will be paralyzed before the heart. This is frequently demonstrated in cases of chloroform and ether narcosis and in diseases of the peripheral nerves. Many patients who suffer from peripheral nerve disorders get well, but a certain number die, and usually through failure of the respiratory function. Thus Drs. Sawkins and Wallace* report six interesting cases of this kind, which were observed in Prince Alfred Hospital, Sydney, of which the following are brief abstracts:

Case 133. Female, aged 54, admitted in a dazed condition and complained of pain in the occipital region, but there was present neither paralysis nor paresis and her heart and lungs were normal. Seven hours afterwards she was suddenly seized with respiratory embarrassment, which was followed

* *Lancet*, August 31, 1895, p. 517.

a few minutes later by total cessation of respiration, but the heart continued to beat for ten minutes longer. Autopsy. Blood clot in fourth ventricle, extending through the iter into the third ventricle.

Case 134. Male, aged 27, complained of occipital and cervical pain. Five years ago had sunstroke, which rendered him unconscious for three weeks. On admission his temperature was 102F, but there was no impairment of sensation or of motion. Ten days afterwards he became cyanosed and respiration ceased, but the heart continued to beat. Artificial respiration was resorted to and the patient recovered, and after several more of such experiences he died, in eight days from the first attack. Autopsy. Old and recent cervical and basal meningitis. Hydrops of the fourth ventricle, iter, third, and both lateral ventricles.

Case 135. Male, aged 19, had been blind from optic atrophy following neuritis, and had paralysis of right arm. His breathing suddenly became stertorous, and finally ceased. Under the influence of artificial respiration his heart continued to beat for forty-five minutes. Autopsy: Four hydatid cysts were found in the posterior part of the left hemisphere. The largest cyst bulged into the cavity of the lateral ventricle.

Case 136. Female, aged 24, had intermittent pain for four years, commencing at the occiput and radiated to the right eye. Five months before admission the pain became so intense that she became unable to read on closing the left eye, and soon after this she had difficulty in breathing through the right nostril on account of a mass in the nasal passage, which was taken for a polypus and removed. Hemorrhage followed the operation. Her breathing became slow and stertorous, and finally ceased, but under artificial respiration the pulse continued to beat for two hours. Autopsy: At the base of the skull a soft, reddish tumor pressed on the right tempero-sphenoidal lobe. The right crus was flattened by pressure of the tumor. The latter had destroyed the sphe-

noidal and ethmoidal air cells on the right side and had invaded the upper part of the right nostril. The right side of the Gasserian ganglon and the optic nerve were involved in the growth .

Case 137. Male, aged 37, and a syphilitic for seven years, had giddiness and headache and poor eyesight for two or three years. Three weeks after admission his right arm became paralyzed. He gradually failed mentally, and seven weeks after admission his respiration suddenly became slower and stopped in a short time. The pulse continued to beat for an hour and a half. Autopsy: Endarteritis of branches of the basilar artery and also basal meningitis, closing up the foramen of Magendie. A gumma as large as a hen's egg was found in the left frontal lobe, surrounded by an area of white softening.

Case 138. Female, aged 10, nine months before admission had tubercular peritonitis. Six weeks previously had pain in the head and back, with convulsions; twelve days after admission had another attack of convulsions, with strabismus. Nine days later had another fit and respiration ceased, but under artificial respiration the heart continued to beat for over two hours. Autopsy: A tubercular mass about the size of a hen's egg was found in the roof of the fourth ventricle, protruding into that cavity and distending it and at the same time invading the right lobe of the cerebellum. The iter was greatly distended.

Case 139. Armstrong reports* the case of a male, aged 45, who, fourteen days after a fall, became suddenly affected with headache, dizziness, deafness and paralysis of right arm and leg and left side of face, difficulty of swallowing and articulation. In a few days more respiration slowed and finally ceased altogether, while the heart continued beating for a long time. Section: Hemorrhage with secondary soft-

* "Apoplexy of the Oblongata," *Med. Record*, Nov., 1892.

ening in the right side of the oblongata, between the pyramid and the olivary bodies.

Sir Dyce Duckworth relates* the histories of four instructive cases in which life and the cardiac function perisited for some hours after respiration ceased.

LANDRY'S PARALYSIS is another disease which begins by invading the peripheral nerves, and which, by gradually extending upwards, implicates the respiratory center, and if recovery does not ensue terminates in paralysis of respiration in the vast majority of instances. In a collection† of 93 cases of this disease, by the late Dr. George Ross, 33 recovered, 41 died of asphyxia, 1 each of bronchitis, pneumonia, phthisis and cancer, and 2 of pulmonary œdema, and in 13 the causes of death are not given. It is seen, therefore, that after deducting the 33 cases which ended in recovery, and without excluding the cases in which no cause of death is given, 76 per cent. died either of respiratory failure or of pulmonary disease.

* *Edinburgh Medical Journal*, Feb. 18, 1898.

† "Mode of Termination and Causes of 93 Cases of Landry's Paralysis," *Medical Chronicle*, 1889, vol. 10, p. 358.

CHAPTER VII.

ETIOLOGY CONTINUED: IMPLICATION OF THE VAGI IN DISEASES OF THE HIGHEST NERVE CENTERS.

Epilepsy.

Epilepsy is a paroxysmal disease and is characterized by sudden loss of consciousness and by partial or general convulsions. Whether its unconsciousness is due to the effects of a discharging lesion of the cortex, or whether it is brought about by sudden spasm of the cranial blood-vessels and consequently of cerebral anæmia, there is no doubt concerning the part which the convulsive center in the oblongata plays in the genesis of the excessive muscular movements which belong to this disease. It is, therefore, in the oblongata and in its important connections that we will have to seek the principal explanation for the intimate and long-recognized association between epilepsy and pulmonary consumption.

Echeverria in his work on *Epilepsy* (p. 313) observes: "I have most closely investigated the relations of pulmonary tuberculosis and epilepsy, and undoubtedly the genesis of tubercles in the lungs is favored by the lesion in the oblongata proper to epilepsy. I have traced the pulmonary trouble from its inception, and feel convinced that the association is more than a casual coincidence of both morbid conditions. I have been no less struck with the frequency of tubercles, or other pulmonary lesions, I have met with on post-mortem examination of epileptics." Again, on page 191, he remarks: "To class phthisis among the diseases which are

capable of superinducing epilepsy in the offspring may, at first sight, appear an altogether untenable opinion. However, a little consideration will make it manifest that it is perfectly consistent with the tendency of lesions of the oblongata to induce pulmonary tuberculosis, which has been pointed out in one of the preceding pages."

Not only is it true that phthisis develops epilepsy, but it is equally true that the latter is frequently translated into the former between parent and child. This is clearly indicated by Dr. Gaston Bechet* in a careful study which he made of the antecedents and collaterals of 40 families, epileptic members of which had come under his observation at the Ville-Evrard Asylum, in France: "Among the parentage (including uncles and aunts) of epileptics, Dr. Bechet finds a predominance of pulmonary affections, and particularly of phthisis, while neuroses and insanity are remarkably rare among them. The children of epileptics, on the other hand, suffer abundantly and frequently from convulsions and meningitis."

Van der Kolk, Jobert de Lambelle, Stuart Cooper and Rostan reported examples of disease of the pons varolii, oblongata and vagus, which were associated with pulmonary disintegration.† The histories of the following cases attest the truth of this intimate relationship, all of which are abstracted from Dr. Echeverria's interesting work.

Case 140. (P. 52.) Adult female, an epileptic of intemperate habits, died of double pneumonia, and at the autopsy her brain and its meninges were found bloodless. The straight sinus was thick and hard, and filled with a discolored clot. Heart fatty, and mitral and tricuspid valves were thickened. There were pleural adhesion, pleuropneumonia, with preponderance of the pneumonic element in the lower two-

* *Archives de Neurologie*, March, 1899, cited from *Lancet*, April 15, 1899, p. 1043.

† Cited from Echeverria's "Epilepsy," p. 76.

thirds of the right lung, and condensation passing into pus on the base of the left. Brain and oblongata in a state of fatty, granular degeneration.

Case 141. (P. 56.) Male, aged 54, an epileptic, with paralysis of the limbs on left side. Death after a seizure. Tuberculosis of the apices of both lungs. The oblongata and cord were very much congested, and the former deeply altered in structure.

Case 142. (P. 57.) Female, aged 40, very intemperate, with unilateral epilepsy and paralysis, and contraction of the limbs on the right side. Died in a paroxysm. Autopsy: Lungs congested, and the lower lobe on right side hepatized. Yellow softening of the cerebellum, oblongata and vagal nuclei.

Case 143. (P. 62.) Female, aged 37, an epileptic and intemperate, and also suffering from chronic bronchitis. After death there were found melanotic and tuberculous deposits in both lungs and degeneration of the oblongata and vagi.

Case 144. (P. 62.) Female, aged 26, a syphilitic and a sufferer from epileptic attacks, which were associated with troublesome coughing, vomiting and other nervous symptoms. Died in a fit. Both lungs were tuberculous, with extensive pleuritic adhesions on the right side, with gray hepatization of the lower lobe on the same side. The path and nucleus of the hypoglossus, the floor of the fourth ventricle and the fibres in the roots of the right pneumogastric were markedly degenerated.

Case 145. (P. 84.) Male, aged about 20, an epileptic from infancy, with paralysis and rigidity of one arm and hand. The epileptic attacks, some time before his death, diminished in frequency, but he took to his bed and, without any fever or marked visceral derangement, gradually sank into a low condition and died. There were found tuberculosis of both lungs and extensive disintegration in the oblongata. The left

cerebral hemisphere, left anterior pyramid and the right half of the spinal cord were atrophied.

Case 146. (P. 101.) Male, three years old, had been subject to epilepsy since his teeth began to appear. He was a bright looking child, with a well developed head, and died suddenly from double pneumonia a few days after having had a series of severe fits. The autopsy showed subphrenal ecchymoses in the posterior surfaces of the lungs, in a state of mingled red and gray hepatization. The oblongata showed extensive morbid changes. There was also extreme dilatation of the blood-vessels in the nucleus and in the course of the pneumogastric nerve, and the primitive fibres of the latter were in large part reduced to the cylinder axis and sheath.

Case 147. (P. 103,) Female, aged 22, became epileptic at the age of 9, in consequence of fright. She died of pulmonary tuberculosis. *Section:* Pleuritic adhesions of both lungs, with gray tuberculous infiltration and small suppurating cavity in the apex of right lung. The oblongata and principally the olivary bodies and the nuclei of the hypoglossus and vagus were degenerated.

Case 148. (P. 105.) Male, aged 24, who during the last two and a half years of his life had been troubled with epileptic fits, which were induced by an unsuccessful attempt to commit suicide by hanging. For an hour after the epileptic attacks he experienced difficulty in swallowing and in breathing. Symptoms of pulmonary consumption rapidly developed, and he died two days after the last fit. *Section* showed pulmonary phthisis. There were no lesions in the brain or cerebellum. The seat of injury lay altogether in the oblongata, in the center of the right olivary body, and in the marginal regions of the left there were speckled effusions from distended capillary blood-vessels. The pneumogastric and hypoglossal cells were deeply injured on the right side. "The trouble with deglutition and respiration, experienced by the

patient after the attacks are plainly accounted for by the lesions at the origin of the hypoglossus and vagus, which through the phrenic nerve acted on the diaphragm. As to the rapid development of phthisis, it probably depended on the lesion of the oblongata."

Case 149. (P. 313.) Female, aged 40, whose mother died of phthisis, had fits in infancy, after which they discontinued and remained away until two years before her death, when they frequently repeated themselves, occurring as often as two hundred times in a day. She became very much depressed and died. *Section* showed cerebral tissue congested throughout. A fibrous tumor, the size of a pea, pressed on the left corpus olivarium. The left pneumogastric was compressed, and the tumor formed a deep sulcus in the oblongata. The right lung was intensely congested at the base, and emphysematous at the apex. The left upper lobe was carnified and studded with tubercles, part of which had suppurated. "The relation between the injury sustained by the pneumogastric and the oblongata and the left lung is self-evident and requires no further remarks on the subject."

Case 150. Female*, aged 24, became epileptic on account of syphilitic infection, and died of double pneumonia three weeks after the attacks began. *Section:* Both lungs were in a state of red and gray hepatization, and the arachnoid was dense and thickened in several places. The membranes over the oblongata were thickened, and the sympathetic ganglia were enlarged.

Résumé. Epilepsy involves the oblongata, and that which is true of every disease which has been discussed thus far is eminently true of epilepsy, *viz.:* (1) its implication of the respiratory center and nerves, and (2) its intimate association in consequence, with phthisis and other pulmonary affections.

* Same author, *Jour. Mental Science*, vol. xxvi, p. 165.

Hysteria.

Hysteria is a so-called functional disease of the nervous ystem, and prevails principally in the female sex. It is characterized by emotional disturbances, by apparent unconsciousness, cutaneous hyperæsthesia and anæsthesia, general and local spasms, rise of temperature, polyuria, accelerated breathing, dyspnœa, aphonia, laryngeal and pharyngeal paralysis, etc., and is closely related to diseases of the pulmonary organs, either through inheritance or through its innate tendency to terminate in such disorders. In view of this tendency it is very peculiar that a number of eminent observers have fallen into the error of believing that hysteria lends a certain protection against pulmonary consumption. Thus Pidoux* states that "neuroses, hysteria, melancholia, the proteiform neuropathic state known as 'neuroism' often act as moderating agents. Phthisis in neurotic subjects has a very slow development, and periods of long intermission. I have no doubt that such subjects, among whom the disease is not rare, offer to it an extraordinary and almost indefinite resistance; and in teaching them we must not pay too much attention to their abnormal nerves." Largraud† makes use of the following language: "We shall show how consumptives, even when most threatened, may live, thanks to hysteria. . . . The neurosis appears to play the part of a protector, as if in its presence the pulmonary complaint had its symptoms mitigated and its progress arrested." Walshe,‡ whose opinions are undoubtedly tinged with the same leaning, expresses a more conservative view on this question. He says: "And, again, though hysteria and phthisis be, in the main, antagonistic diseases the antagonism is assuredly not absolute, and faith, too confident in its

* "Etudes sur la Phthisie," p. 151.

† *Thesis*, Montpelier, 1882.

‡ "Diseases of the Lungs," p. 479.

reality, has more than once led to serious error. It has every now and then happened that an hysterical woman, whose alleged chest-ailments were treated as pure figments, has died of genuine phthisis while supposed to be simulating the disease. In one instance which fell under my own notice the expectoration of elastic lung-tissue was the first circumstance that proved the sufferer was not a malingerer."

In a most interesting series of articles on *The Relation of Hysteria with the Scrofulous and the Tubercular Diathesis,* by Dr. J. Grasset,* M.D., Professor of Medicine in the Faculty of Montpelier, France—a contribution which, with certain limitations presently to be noted, is as remarkable for its rarity as it is for its erudition and display of practical knowledge—appear the histories of forty-four cases of hysteria, which are allied to the tubercular diathesis. In his introductory remarks and commentary on these cases he states that "the tubercular diathesis may betray itself by neuroses, hysteria especially, chorea, etc. I have recently seen two cases in which the hysteria seemed to be the manifestation of the tuberculosis. These facts have a great clinical importance, because frequently hysteria is developed in a young woman with a diathesic heredity; the neurosis is treated; it disappears, and then pulmonary accidents, sometimes of the utmost severity, are evolved. Or the hysterical phenomena may persist during the phthisical process, and present a series of compensatory rises and falls. Finally, hysteria may be the only expression of the diathesis, and phthisis reappear only in the succeeding generation."

On looking into the groundwork of Dr. Grasset's theory it will appear that he regards the tubercular diathesis as the fundamental basis from which hysteria, other neuroses and pulmonary consumption spring. By this he does not mean that hysterical subjects suffer from tubercles, but that the tubercular diathesis is a constitutional state found in a series

* *Brain*, vol. vi, p. 433; vii, pp. 13 and 161.

of generations of the same family, and among a certain number they are represented as neuroses. Thus, for instance, he says in a phthisical family you will see, of the children, one dying from tubercular meningitis, another becomes an ordinary consumptive, and the third escapes the diathesis, or the last may be neurotic, hysterical or a lunatic even. But the last has escaped the diathesis in appearance only. He is tubercular, like the rest, though he has no tubercles anywhere. It is his nerurosis which represents the diathesic affection.

But why hold the tubercular diathesis responsible for the production of hysteria, lunacy and pulmonary consumption when we know that tubercle plays but a secondary part in the great majority of cases which are affected with the latter disease and is, as a rule, absent from the two former? It would seem to be more in accord with truth if this order of things were reversed and the neurotic diathesis made answerable not only for the varied abnormal manifestations of hysteria, insanity, etc., but for the catarrhal and tubercular forms of pulmonary phthisis. Clinical experience certainly teaches, as has been demonstrated in the foregoing pages, and as will appear further on and as indeed most of Dr. Grasset's cases clearly show, that the neurotic element takes precedence in the development of the morbid process known as the tubercular condition. Bearing these points in mind, abstracts of the histories of Dr. Grasset's cases will now be given and afterwards an effort will be made to analyze their true bearing. He divides them into two groups: (1) Those of hysteria, with tubercular heredity but without marked pulmonary manifestations, and (2) those of hysteria with tubercular heredity and with well pronounced pulmonary disease. (The Roman numerals indicate Dr. Grasset's cases.)

Case 151. (I.) Female, aged 3, had a sister who died of meningitis, aged about eight months, with violent convul-

sions, and a brother who died from cerebral disease during dentition. Maternal grandmother was epileptic and died insane. Maternal aunt had great fear of being drowned. Maternal grandfather had cancer of the upper lip. Mother very lymphatic and nervous. Maternal uncles and aunts are all scrofulous and below the mental average. The patient has had lymphatic enlargements of the neck. An emotional shock, brought on by falling into a tub, was followed by an eczematous eruption. Three months after this she was seized with vertigo, followed by loss of consciousness, retraction of thumbs and convulsions of the eyeballs.

Case 152. (II.) Female, aged 13, whose father died from cancer and her mother from consumption, was scrofulous during childhood. At the time she was first seen she had extraordinary local sweatings, which followed violent pain and constriction of the lower part of her thorax. Her hysteria is of a subdural nature chiefly. Slight dulness in the apex of one of her lungs.

Case 153. (III.) F., aged 27, whose father, paternal uncle and paternal grandmother are insane, whose mother is herpetic, and who lost one sister of whooping-cough, one of cerebrospinal meningitis, and one of tuberculosis, was scrofulous and eczematous from childhood. She suffers from coldness and numbness of extremities, cutaneous hyperæsthesia, convulsive jerkings and has difficulty in swallowing, sialorrhœa, etc.

Case 154. (IV.) Female, aged 38. Mother and father and brother died from consumption. She became subject to hysterical fits at the age of 20, which were grave, sometimes cataleptic, and occurred repeatedly. She also has violent pain in the spine and thorax. Nothing in the chest except roughness at the apices, but coughs and frequently suffers fronm bronchitis.

Case 155. (V.) Female, aged 32. Father died young, of pneumonia, and her mother of heart disease. She is the

eldest of three daughters, one of whom died of phthisis and the other is hysterical. Fourteen years ago she had a still-born child, upon which grave eclamptic symptoms supervened, and, after which, frequent sick headaches. The violent frontal pains became real seizures, occasionally accompanied with very short loss of consciousness, preceded by vertigo and followed by loss of memory during a few seconds. Little by little she became worse, and the paroxysms of pain and unconsciousness became more frequent. Her condition was diagnosticated as hystero-epilepsy.

Case 156. (VI.) Female, aged 10. Mother died phthisical, and father and ten brothers and sisters are in good health. At this age she became subject to attacks of nervousness, fits of somnambulism, right ovarian pain, frequent sick headaches, anæsthesia of conjunctiva and right hemi-anæsthesia.

Case 157. (VII.) Female, became hysterical when she was 9 years of age. Her mother died of phthisis. She had attacks of syncope, with forebodings, pain in the left ovary, and anæsthesia of conjunctivæ and soft palate.

Case 158. (VIII.) Female, 37 years old; lost a brother from phthisis and has two ill sisters. From childhood until she was 29 years old she had a convulsive attack, with loss of consciousness every nine or ten days. These paroxysms ceased at that time, but she has remained hysterical.

Case 159. (IX.) Female, adult, whose father died of phthisis, and one sister of convulsions, and whose mother is hysterical, has been suffering with choking and spasms in her throat since her earliest childhood. At 7 she became completely hysterical; at 16 she had chorea, and at 17 she again became hysterical, which condition persists up to the present time.

Case 160. (X.) Female, aged 24, lost her mother and sister from consumption, and had her first hysterical attack at 5 years of age, which state persists until now.

Case 161. (XI.) Female, adult, lost both father and mother from phthisis. At the age of 7 she was frightened, and was immediately seized with convulsions and loss of consciousness. These attacks gradually became less frequent and disappeared when menstruation began, at the time when she was about 12 years old. When she arrived at the age of 20 she had another well marked attack of hysteria, which was also induced by fright.

Case 162. (XII.) Female, 17 years old, lost her father and mother from phthisis. She became hysterical at the age of 9, and although the attacks are less frequent they still continue.

Case 163. (XIII.) Female, aged 29, lost her mother, a brother and sister of phthisis, and had her first hysterical attack when she was 8 years old, and until 10 years she had a seizure almost every day. Then they diminished in frequency, but increased again at about 15 years, when menstruation set in with difficulty. Now the girl has an attack every week.

Case 164. (XIV.) Female, aged 29, has a hysterical mother, and lost her father from phthisis. From childhood she suffered from choking and epigastric oppression. Menses began when she was 12 years old, and at 15 or 16 she became subject to numbness of the limbs and muscular trembling. At 22 she began to have hysterical fits, which still continue.

Case 165. (XV.) Female, 22 years old, whose mother and sister died from phthisis, was delicate and impressionable until she was 17 years old, when she menstruated for the first time. She then became subject to amenorrhœa, left pleurisy with effusion and marked anæmia. About 18 or 20 her menses became regular, and her health was restored. At 20 she became hysterical on account of sorrow over the death of her sister.

Case 166. (XVI.) Male, aged 15, father had good health until he fell ill and died from smallpox. Mother and one sis-

ter are well, although they have scrofulous cervical glands. One sister died from consumption and a brother from tubercular meningitis. At the age of 10 he sustained a fall on the head, after which he became affected with convulsive seizures, globus hystericus, hemi-anæsthesia, etc.

Case 167. (XVII.) Adult male, mother well, but father and three children died of phthisis; all the other children, except the patient, are well. The patient has a weak constitution, suffers from pulmonary tuberculosis and appears to have been epileptic before he was 20. During eleven months he was subject to hysterical fits, which later assumed the character of hystero-epilepsy.

Case 168. (XVIII.) Adult female. Her father was an alcoholic, and drunkness was very prevalent among his paternal ancestors. Her mother, who was neither nervous nor had fits, died of phthisis at the age of 36. Her grandmother and two maternal aunts died of the same disease. Patient had two sisters; one died at the age of ten days, the other is 29 years old, weak, very nervous, liable to frequent outbursts of anger and continually complaining of cardialgia and leucorrhœa. She has two children—one a feeble looking boy and the other an idiotic girl. Her husband was an alcoholic. The patient had measles when she was five years old, after which she became scrofulous, and also had an eruption of the skin. At 21 or 22 she had a nervous attack after some provocation, followed by chorea, malaise, incessant vomiting with gastralgia, extreme mental uncertainty and outbursts of mania.

Case 169. (XIX.) Female, 25 years old. Her mother and grandmother died of phthisis and her father of insanity. She became hysterical when she was 19 years old.

Case 170. (XX.) Female, aged 42, her father died suddenly and her mother, who was subject to nervous attacks, probably died of pulmonary tuberculosis. Patient menstru-

ated when she was 12 years old, and she became hysterical after having been abandoned by her husband.

Case 171. (XXI.) Female, aged 18, her father is insane and her mother died of some chronic chest affection (cough, hemoptysis). There were nine children, of whom the patient is the eldest. Four of these died of convulsions and one of epilepsy (?). One boy has hemoptysis, one girl seems subject to attacks of weakness, and one boy is lost sight of. Patient had convulsions, in consequence of which she became paralyzed, deaf and dumb. Until she was 7 years old she had outburst of passion, attacks of measles, eczema of the head, etc. After 12 she had syncopal attacks, hemi-anæsthesia, etc.

Case 172. (XXII.) Female, 19 years old. Phthisis on the father's side. Hysterical contracture of traumatic origin, which suddenly disappears and reappears. She also has transitory aphasia and various nervous ailments.

Case 173. (XXIII.) Female, aged 10, whose mother, maternal uncle, and aunt were scrofulous. Patient was suffering from choreic movements for a time, and after they ceased there was a sudden partial paralysis of the right lower limb. After the paralysis began the choreic movements had nearly disappeared. In two months the paralysis gradually decreased and the day it disappeared completely the choreic movements returned, with increased severity. "The alternation between the paralytic and the convulsive phenomena was not altogether complete, but almost so." She also had gastric crises, hiccoughs, sleeping paroxysms, etc., and her case was diagnosticated as hysterical chorea.

Case 174. (XXIV.) Female, aged 42, lost her mother after a long illness, an uncle and aunt from phthisis, and one uncle from diabetes and another from heart disease. Menses began at the age of 10, and ten years ago she became violently hysterical. She also had frequent headaches, great

diminution of strength, pruritus, vulvæ, intense thirst and considerable polyuria.

Case 175. (XXV.) Male, aged 35, has a slight neuropathic heredity from his mother and maternal grandfather. His father and brother died of phthisis. Patient himself is scrofulous. His illness is essentially characterized by sensations of constriction of the throat and pain and weakness in the loins. Dr. Grasset says that the case would be called simply neurasthenia by many, but that all these neuroses belong to the same family.

Case 176. (XXVI.) Female, aged 18, was admitted August, 1872. Her father was irritable, had nervous attacks, and drank alcohol to excess and died of pleurisy, at the age of 42; her mother suffered from bronchitis; her paternal grandfather was neurotic, his maternal grandfather died of apoplexy, aged 60; and her maternal grandmother died from phthisis, aged 46, after having given birth to twenty-one children.

During childhood patient suffered from scrofula and bronchitis, and at the age of 13, July, 1871, had the first hysteric attack, from which fits occurred with all their typical characters. In January, 1876, the hysteria disappeared and she began to lose strength and had vomiting and diarrhœa. In May she began to lose flesh rapidly and showed the first symptoms of pulmonary phthisis, which disease carried her off on the 21st of July. The peculiar feature of this case is the disappearance of the hysterical condition on the advent of phthisis.

Case 177. (XXVII.) Adult female, admitted April, 1862; her father was a drunkard and died dropsical, and her mother had hemoptysis and died of bronchitis. She had six brothers and sisters, and they are all dead but one. She had several attacks of bronchitis during childhood; had her first hysterical fit at the age of 13, and at 21 she suffered from left hemiplegia. Coughs for the last four and a half years,

and had hemoptysis twelve months ago. Her expectoration is abundant, muco-purulent and somewhat fœtid, and she has occasional fits of dyspnœa. Dulness in right apex and moist râles here and in left apex. Sonorous râles everywhere. In 1871 her left leg became rigid, which was accompanied by tonic contraction of the left arm and by rotation of the head to the left. The hysteriform fits continued and sometimes epileptic manifestations alternate with the hysterical attacks. Concerning the condition of the lungs it is stated that in 1878 there were no particular accidents to note with reference to those organs. There was slight diminished resonance in the right apex and a rough vesicular murmur in the left, which was also weaker, and she coughed but rarely.

Case 178. (XXVIII.) Female, aged 20, her father died on account of injury, her mother is very nervous, and one of her sisters died of phthisis at the age of 18. Patient is chlorotic, very nervous, and laughs and cries without motives. She had hysterical fits with stercoraceous vomiting, and evident crepitation at the apex of the right lung. Later on she had epilepsy, with left ovaritis and hemi-anæsthesia. The recorder says that when her hysterical attacks came on the cough was less troublesome and the state of the lungs seemed to improve, and that when the nervous symptoms diminished in intensity the condition in the lungs grew worse. Finally both lungs became diseased and she died of phthisis. She had no hysterical attacks during the last six months of her life.

Case 179. (XXIX.) Male, aged 37, lost his mother in early life, either from the effects of a nervous or a cancerous disease; and his father died after two months of mental alienation with paralysis of the lower limbs. His sister suffers from a nervous affection. When he was 35 years old he had convulsive attacks, which were followed by weakness and numbness in the left side of the body and permanent pain in the middle of the back. At the time of his admission the

chest, with the exception of deficient resonance under the right clavicle and a weak vesicular sound in same area, was normal. After this, attacks of dyspnœa occurred, but he had neither cough nor expectoration. When he exerts himself to cough, to swallow or to move about freely one hears a kind of stridor during respiration, which resembles that of the first stage of a fit of whooping-cough, and suffocation seems imminent. At these times he is very frequently subject to convulsive attacks of hysteria, anæsthesia, etc. He gradually became worse and died of phthisis pulmonalis.

Case 180. (XXX.) Adult female; her father was an alcoholic and a libertine, and her mother was very nervous, and died of uterine cancer. Many of her relations are inebriates. One brother and a sister died of disease of the chest and another brother coughs a great deal and is always ill. Two years before her admission, after a violent rage, pigmentation of the face occurred, and six months before she had pleurisy of the right side, from which she rapidly recovered. She had a chill recently, after which she coughed and had two copious hemoptyses. She has night-sweats, evening fever and loss of flesh, and suffers from pulmonary phthisis in both apices. The report contains a detailed study of the hysterical phenomena which were observed or provoked in the patient.

Case 181. (XXXI.) Female, aged 20, whose family history is not given, had frequent hemoptyses, obstinate cough, intercostal pains, wasting, dyspepsia and crepitation at both apices. Applications of cold water to the extremities checked the recurrent hemoptysis, but the general state of the patient remained bad and the phthisis seemed to advance. She was now submitted to a hydrotherapeutic course and after a shower-bath a formidable hysterical attack broke out. A true neuropathic condition manifested itself, which lasted for a whole year and during this time the morbid pulmonary phenomena improved in a marked manner. After this she

had an attack of sciatica, which cut short the hysterical neurosis. The sciatica resisted every form of treatment for eight months, but ceased on the occurrence of a furuncular eruption (boils), and after this she showed nothing special in regard to either the lungs or the nervous system.

Case 182. (XXXII.) Female, 30 years old, had all the signs of softening at the apices of both lungs, and then suffered from a series of most violent hysterical fits and during the existence of the latter the morbid pulmonary condition improved from day to day. Two years later she bore a child, and at this time her lungs are entirely well.

Case 183. (XXXIII.) Female, suffered from hemoptysis and presented sure signs of softening at the apices. She became hysterical, and after this there was a gradual improvement in her phthisical condition. She remains nervous, but is pale and thin, and has a dry, fitful cough.

Case 184. (XXXIV.) Female, showed all the symptoms and conditions of pulmonary phthisis. After which she is seized with a large number of hysterical fits, and then the phthisis improved and disappeared, giving place to a hysterical paraplegia.

Case 185. (XXXV.) An adult female and a foundling, has been delicate from youth up, and presented sure signs of scrofula, such as ganglionic swellings of the neck and very obstinate eruption on the head. During a one year's stay in the hospital she had an obstinate cough, which alternated with hysterical fits. At the end of this time she was very much better so far as the cough and the neurosis were concerned, but her general health continued bad. She married and bore a child, which lived only a few days. After this her fits diminished in number and in intensity, but her cough returned with greater force and she began to suffer from an obstinate diarrhœa. At this time she had crepitation at both apices and prolonged expiration. She had night-sweats and as many as ten stools a day, which were of the color of clay

and of the consistence of pap. She had no convulsive attacks, but chattered incessantly and passed without reason from laughter into sobbing fits. The hysteria remained hardly noticeable for three months, but the phthisical degeneration progressed rapidly in her lungs. After this her hysterical fits came on so violently again one night that the nurse could hardly keep her in bed. These continued for some time and they noticed with astonishment that all the phthisical symptoms were daily improving, and in four months the lungs had nearly recovered their natural action and the patient had gained a little in flesh and in strength. She resumed her occupation, but finally died from diarrhœa.

Case 186. (XXXVI.) Female, aged 27, took a cold, which was followed by bloody expectoration, hemoptysis, night-sweats, weakness and loss of flesh. Physical examination showed crepitation at both apices and prolonged expiration. Patient grew worse daily until two months after being admitted; she was suddenly taken with a violent hysterical fit, which completely exhausted her. These fits recurred daily for four days, and since that time the pulmonary affection improved so rapidly that in a month every trace of the phthisical degeneration had disappeared. The hysteria went on, but assumed a benignant type, and the patient left the hospital very much improved.

Case 187. (XXXVII.) Female, 17 years old, is taken with severe cough, sanguineous expectoration, and showed crepitation in right apex and harshness of the respiratory sounds, and prolonged expiration on both sides. Two months after her admission she was suddenly taken one morning with a hysterical convulsion, which repeated itself in a slight form daily for two weeks, during which time the condition of the lungs and that of the constitution improved very much. In a month after this the hysteria was very much improved, and at the time of her discharge the symptoms of pulmonary phthisis had disappeared, and of the neu-

rosis there only remained a great proneness to lying and chattering.

Case 188. (XXXVIII.) Female, aged 7, whose father and mother are dead of pulmonary phthisis, and who, after a cold, became feverish, has an obstinate cough, slight hemoptysis, night-sweats, and is losing flesh rapidly. The right apex shows signs of softening. Three times these signs and symptoms abate and reappear. At last, eight months after the beginning, there occurred marked and lasting amelioration, and there remained only slight neuralgia in the intercostal spaces and in the legs. At about the same time she became subject to fits of somnambulism. Dr. Largaud, the reporter of this case, states "that the cure of the phthisis in this child of 7, which seemed likely to go through its different stages in a very limited time, is much less due to the treatment followed than to the appearance of a neurosis, which at first checked and afterwards removed the thoracic accidents."

Case 189. (XXXIX.) Female, aged 29, was admitted in May, 1873, and in the preceding April she had pneumonia of the right lung. In June she had symptoms of pelviperitonitis, and at that time became whimsical and nervous. In November she slept for three days, became very nervous and had persistent anuria. In January, 1874, she had left hemianæsthesia, in June paralysis of the left leg, and in August pleurisy with effusion. In October the anæsthesia and paralysis became paraplegic. In December she had a few fits of coughing, followed by slight hemoptysis accompanied by thoracic pains. In January, 1875, crepitation was found in right apex. Voice was harsh and almost toneless for more than three months. She had vomiting and ischuria. The hysterical state showed itself by a marked mental peculiarity, by diffuse neuralgia, by feelings of suffocation, which for some weeks past had taken the character of the usual globus, and there was also profound anæsthesia of the ocular and

pharyngeal mucous membranes. After March she had pulmonary congestion of the left side. In April she slept for seventeen days. In July she left the hospital, after which she did light work, but continued to show signs of pulmonary tuberculosis. In April, 1880, she had a slight cough, but auscultation revealed nothing but a little respiratory harshness at the apices.

Case 190. (XL.) Male, 25 years old, who has a sister dead of phthisis, is found to have dulness of the right apex and very profuse sweating. His mind after this became impressionable, irritable and anxious, and he cries easily and presents various nervous phenomena. He has developed epigastric constriction, hemiplegia and hemianæsthesia of the right side, aphonia, etc., and the signs of pulmonary tuberculosis have abated.

Case 191. (XLI.) A soldier, admitted June, 1880, lost a brother of convulsions, and has a sister who is afflicted with curious nervous disturbances at each menstrual period. He is scrofulous and sure he had measles in childhood; he is specially liable to bronchitis, and his expectoration is often sanguineous. About ten years before his admission he had an attack of hysterical convulsions, and at the time he is admitted the sensibility of his whole body is obtuse, with some hyperæsthesia, painful vertebral spots, etc. In the right apex there is evidence of tuberculosis. While being auscultated he suddenly became pale, lost consciousness and had a genuine hysterical outbreak. There was neither froth at the mouth, nor biting of the tongue, and his face did not even become turgescent. In three months the patient was dismissed in a good state of health, although he was not completely cured.

Case 192. (XLII.) Female, 57 years old, whose mother was of a nervous temperament, suffered from suppression of the menses when 17 years old, in consequence of a bath. After this she became afflicted with violent headache, insom-

nia, melancholia, and globus hystericus. In five months her menses were re-established, and she married at 25. After the death of one of her children she became nervous, had frequent headache, and sensations of globus as well as menstrual disturbances. When admitted (July) she had a dry, nervous cough of an intractable character. In November there was dulness in the right apex, prolonged expiration and harsh respiration in same area. In December there was moist crepitation in same area and elevation of temperature. After this excavation formed rapidly in the lungs and the patient died February 10th following. In this case the pulmonary phthisis rapidly superseded the nervous state.

Case 193. (XLIII.) Female, aged 55, whose father died of age, although he always suffered from a herpetic eruption, and whose mother died of pneumonia at the age of 58. Patient is nervous, often suffering from dartrous eruptions, her menses appeared when she was 22, and menopause occurred when she was 48. She has been treated for syphilis, and had hystero-epileptic attacks, which, in some instances, were suppressed by compression of the ovary. She also had frequent hemoptysis. At the time of her admission she had right hemianæsthesia and almost total loss of vision in right eye. About a week after this there were noticed crepitation and dulness in the right apex, and from time to time crepitation in left lung. Her expectoration is yellow and is tinged with blood. She continues to suffer from attacks of hystero-epilepsy and is subject to fits of vertigo.

Case 194. (XLIV.) Female, aged 20, whose father died of phthisis, but whose mother is strong and robust. She has violent fits of coughing, which last many minutes and threaten to suffocate her. The cutaneous sensibility is very much diminished in the upper and lower limbs on the left side. Nothing is found in her chest, and her cough is believed to be hysterical. After a few months tubercular lesions appear in the left apex and then the nervous phenomena

improve gradually, and in a short time the thoracic disease entirely displaces the neurosis.

Now what do these cases teach in regard to the origin of hysteria and phthisis? Do they furnish evidence of the correctness of Dr. Grasset's opinion, that hysteria is the outgrowth of the tubercular* diathesis? In other words, does the tubercular condition always precede the manifestation of hysteria, either in the family or personal histories of these forty-four patients? On analyzing their family histories we find that six had a neurotic, nineteen a tubercular, and nine a mixed heredity, while in ten no history is given. It seems certain, therefore, that the tubercular diathesis does not prevail universally in these cases. And if, on the other hand, we add the nine instances in which existed a mixture of the neurotic and tubercular tendency to the six in which there was a pure neurotic heredity it will appear that the neurotic family tendency was present in fifteen cases. Moreover, from the personal histories of these cases it appears very evident that the neurotic state precedes the tubercular in the individual; for among the forty-four cases there were twenty-five who became tubercular at some period of their lives, and in nineteen of these the neurosis antedated the tubercular outbreak, while in six the reverse was true. Taking a general survey of these cases there does not, therefore, seem to be any convincing evidence that the tubercular diathesis plays such a prominent part in the production of hysteria, or that the latter is the direct outgrowth of the former. On the other hand, if we believe that phthisis is fundamentally a neurosis, it becomes clear why these diseases should be intimately associated, why hysteria, epilepsy, or alcoholism, or insanity should exist in one branch of a family and phthisis, or some other neurosis, in another.

* The word tubercular in this paragraph is used synonymously with scrofula, and phthisis, and tuberculosis of the lungs.—Author.

Idiocy.

Idiocy is a form of mental incapacity which depends on arrested development or disease of the brain and nervous system, and begins before birth or during early childhood and youth. Given, then, a nervous organization which has been feeble from or before birth it is quite obvious that these children should be specially vulnerable to pulmonary phthisis if it is true that the latter is the direct outcome of a depressed and debilitated nervous system. It is of great interest, therefore, to inquire into the relation which exists between idiocy and pulmonary consumption, and this will probably show conclusively that the association between these two states is of the closest character. Thus the late Dr. Isaac Kerlin,* in a paper on "Classification and Causation of Idiocy," says: "In no department of medical practice can the physician be more efficient than in the timely and judicious treatment of families of the so-called scrofulous diathesis. Our inquiries show that phthisis is present to the extent of 56 per cent. in one or both families of the idiotic child. Apprehending this, all sensible precautions in the family life where this diathesis is strongly present should be taken to avert the greatest calamity and the most intense grief that can enter a home.

"Dr. Grabham, in examing the papers of 249 cases which were admitted to the Earlswood Institution, finds that in 55, or 22 per cent., there is a history of phthisis in the parents or near relatives—in 17 per cent. no other cause is given for the idiocy. The tables presented with my paper, if prepared by a special advocate to prove that consumption is the main factor in the generation of idiocy, could not be more startling. As they are the result of careful inquiry, without any theory to prove or to disprove, I ask for them your respectful judgment in this as in other details which they present.

* *Trans. Penna. Med. Soc.*, 1880, p. 172.

"In the 100 families represented by them, and in each of which occur one or more idiotic children and often other defectives, consumption, as found by our correspondents, is as follows:

Paternal grandparents of 17 of 100 idiotic children.
Maternal " " 27 " " " "
Fathers of 22 of 100 idiotic children.
Mothers of 27 of " " "
In grandparents and parents of 23 of 100 idiotic children.
In grandparents on both sides of 6 of 100 idiotic children.

"That is, we have consumption as a possible factor of idiocy in 56 of the 100 families."

What is still more surprising is the fact that a careful examination of Dr. Kerlin's table shows that in the families from which these 100 idiotic children sprang there were 145 other members of these families, including parents, sisters and brothers and grandparents, who were afflicted with pulmonary consumption.

In the article on "Idiocy," in Tuke's "Dictionary of Psychological Medicine," appears the table on the following page, which gives the causes of idiocy and imbecility in 2,380 patients who were received into the Royal Albert and Dareuth Asylums in England. These causes are divided into those which operated before, during and after birth, and it will be seen that phthisical heredity is one of the most powerful causes in the production of these diseases.

If this table accentuates one thing more than another it is the fact that phthisis plays a predominating hereditary influence in the production of idiocy and imbecility—ranking higher in this respect than insanity, epilepsy, intemperance, infantile convulsions or any of the other neuroses, whether latent or active. Is this not another reminder that phthisis, although differing in its exterior manifestation, is, after all, one of a group of diseases, the foundation of which is rooted in the nervous system?

Dr. J. Langdon Down in his work on *Mental Affections of*

TABLE OF CAUSES IN 2380 CASES OF IDIOCY AND IMBECILITY.

	Total Number of Cases.		
	Number Times Recorded.	Percentage.	
I. Causes Acting before Birth.			
Family history of (A) phthisis	674	28.31	
" " " (B) insanity	392	16.47	21.38
" " " (C) imbecility	117	4.69	
" " " (D) epilepsy	207	8.69	20.00
Other neuroses	269	11.30	
" " " (E) intemperance	390	16.38	
Parental or grand-parental consanguinity	100	4.20	
Abnormal condition of mother during gestation.	711	29.87	
II. Causes Acting during Birth.			
Premature birth	84	3.52	
Primogeniture	492	20.67	
Protracted pressure in delivery	339	14.24	
Instrumental delivery	79	3.31	
Twin birth	23	0.96	
III. Causes Acting after Birth.			
Infantile convulsions	652	27.39	
Epilepsy and cerebral affections	193	8.11	
Infantile paralysis	22	0.92	
Injury to head from fall, blow, etc.	147	6.17	
Fright or mental shock	73	3.06	
Sunstroke	13	0.54	
Scarlatina, whooping-cough, measles, typhoid fever, and smallpox	142	5.96	
Over pressure at school	4	0.16	

Childhood and Youth states (p. 219) that "no one who has had an opportunity of investigating the influences which are at work in the production of congenital mental diseases can fail to be struck with the fact that they are, for the most part, to be traced to some inherent vice of constitution in the progenitors. . . . Amongst the influences which have been regarded as connected with idiocy very little attention has

been given to that of tuberculosis, and I am not aware that any observations have been made with reference to the connection of these two maladies. Several writers have discussed the relations between insanity and tuberculosis, and have, I think, made it tolerably evident that there is more than an accidental connection between them.

"At the Earlswood Asylum, where the following observations have been made, the subjects of the inquiry are not likely to present an unfair proportion of tubercular idiots. Rather would they be likely to be below the average. The inmates are, for the most part, elected after great exertion, and the friends of a phthisical idiot would scarcely be likely to undertake the trouble for a manifestly short-lived child, even if the rules of the institution did not exclude it.

"During the past eight years, from June, 1859 to 1866, inclusive, there have been 201 deaths. During this time there have been two epidemics of measles, one of scarlet fever, and two of whooping-cough, which have all added to the mortality.

"The statistics of London show that the deaths from phthisis constitute 115 per 1,000 of the general mortality. My notes of the causes of death at Earlswood indicate that phthisis was the actual cause of death in 398 per 1,000 of the general mortality.

"It will be obvious that, in consequence of the greater readiness with which idiots succumb to epidemics or other diseases, the proportional deaths from phthisis are thereby much decreased. This element may be fully brought out by dividing the eight years, bringing together the four epidemic years, and comparing them with the four non-epidemic years. It will then be seen that during the epidemic years, 1860, 1862, 1863 and 1866, the deaths from phthisis numbered 297 per 1,000 of the general mortality, or considerably more than twice the ratio which rules in London; while in the non-epidemic years, 1859, 1861, 1864 and 1865, the deaths

from phthisis reached the enormous proportion of 570.58 per 1,000 of the general mortality. . . .

"The above details have reference solely to ante-mortem diagnosis and have included cases where the death was evidently caused by phthisis. I have, however, made an analysis of the last hundred of my post-mortem records and I find that no fewer than 62 per cent. were subjects of tubercular deposit—52 of which being phthisis of the lungs. . . .

"In several of the cases included in the above record most careful inquiry failed to discover any family history of tuberculosis; and the brothers and sisters were thoroughly vigorous. In these cases the tuberculosis appears to have been the sequence of the idiocy—a condition of idiocy resulting form accidental causes. Defective innervation in all probability led to malnutrition and predisposed to a tubercular condition. In some this was doubtless materially assisted by the imperfect mastication and insalivation to which the food was subjected.

"On the other hand, in a large number of cases the progenitors had also manifested a tubercular condition, and in some the tuberculosis of the parents had been, in my opinion, the prime cause of the idiocy of the offspring. . . .

"It appears to me that tuberculosis must be accepted as one important cause of idiocy; that it impresses special characters thereon, characters which impart a strong family likeness to the subjects of this class.

"It is no less clear to me that idiocy of a non-tubercular origin leads to tuberculosis. Whether this arises through the influence of the pneumogastric nerve, mal-assimilation of food, or defective innervation it cannot but be regarded that the connection between these two maladies is by no means accidental and that a due appreciation of this relation is necessary to those who would treat effectively congenital mental lesions."

Moreover, on page 190 (*op. cit.*), Dr. Down relates the

detailed histories of twenty families, each of which was burdened with idiocy, and in which he found thirty-five other members among the parents, sisters and brothers, grandparents, uncles and aunts of these families, who suffered from pulmonary consumption. Thirty-five sufferers from pulmonary consumption in twenty families would be equivalent to one hundred and seventy-five sufferers in one hundred families; and when this percentage is coupled with the statistics of Dr. Kerlin, in one hundred families of which there existed one hundred and forty-five other members (including the same relatives as in Dr. Down's cases) who suffered from pulmonary consumption, one can, at least partly realize the enormous influence which is exerted by pulmonary phthisis in the production and propagation of idiocy in the offspring. This relationship of the two diseases becomes still more obvious when we reflect that only about eighteen members of every hundred families among the general population suffer and die from phthisis, which means a mortality at least eight times greater among the former than among the latter. It is true that the statistics which are given here are somewhat higher than those of some other observers, yet in forming an opinion in regard to this matter we must bear in mind the trustworthiness and painstaking character of the investigators from whom the above information is obtained.

Nor is this all, but over and above the fact that the phthisical death-rate among idiotic children is enormous, further inquiry demonstrates that phthisis occurs earlier in life among this class of sufferers than it does among the general population. Thus the greatest general mortality of consumption takes place between the ages of 20 and 30; but in Dr. Down's record of eighty idiots who died of this disease the highest mortality-period is between 10 and 20 years, showing that congenital or infantile disease of the brain and nervous system is capable of making the highest mortality-

period of pulmonary consumption appear about ten years earlier than among the general population.

Deaf Mutism. There is evidence, too, to show that deaf mutism, which is likewise due to some congenital defect in the brain and nervous system, predisposes its victims to pulmonary consumption. Hartman notes* that in deaf mutes the lungs are imperfectly developed, and remarks that this cannot be due to loss of speech and of consequent functional inactivity of these organs, for many such children have the power of shrieking and of making noises with the voice. From *post-mortem* examinations Meissner† shows that in this affection the larynx is undeveloped, the lungs are poorly nourished and imperfectly expanded. Kussmaul is of the opinion‡ that there is a want of thoracic development and that on account of this fault these children are frequently predisposed to pulmonary phthisis. In sixty-one deaths among deaf mutes Meissner found thirty-one caused by phthisis; and Schmalz§ discovered that in twenty deaths thirteen died from phthisis and one from laryngeal phthisis.

Insanity.

That insanity and pulmonary consumption are closely associated in many respects has been noted by many authors. Van der Kolk‖ states that "since we so often see some members of a consumptive family afflicted with insanity, while others are spared from insanity but become the victims of phthisis, and that the two diseases frequently alternate with one another or co-exist, the question has often suggested itself to me whether we might not admit the existence of

* "Taubstummheit und Taubstummenbildung," p. 8.
† "Taubstummheit und Taubstummenbildung," p. 201.
‡ "Die Störungen der Sprache," p. 262.
§ "Ueber die Taubstummen und ihre Bildung," p. 117.
‖ "Atrophy of the Brain," *Sydenham Publication*, vol. xi. p. 170

a phthisis excentrica, *viz.*, one whose first cause is to be sought in an irritated condition of the oblongata and the vagus, such as so frequently occur in phthisis."

Dr. Maudsley* says "perhaps I might set it down as a true generalization that the morbid neurosis, when it is active and gets distinct morbid expression, may manifest itself in four ways: (*a*) In disorder of sensation, for example, paroxysmal neuralgia; (*b*) in disorder of motion, for example, epilepsy; (*c*) in disorder of thought, feeling and will —mental derangement; (*d*) in disorder of nutrition, whereof diabetes is the earlier and phthisis is the later stage." Dr. Blandford† states: "I have found, however, that phthisis and insanity do frequently co-exist in the same family, but that some members will be afflicted with insanity while others suffer from phthisis." Dr. Mosher‡, from whom the two previous extracts are taken, relates the following interesting case:

Case 195. Female, aged 16, was admitted as an insane patient September 25, 1893. She was insane for eight years, and her attacks had been of an epileptiform nature, characterized by sudden outbreaks of violence and probably associated with unconsciousness, either partial or complete. Heredity was the assigned cause, eight paternal great-uncles and great-aunts had died of phthisis and her paternal grandfather was epileptic in youth and neurotic. During her residence of a little more than six weeks she had an attack of intractable bronchitis and laryngitis, with marked laryngeal congestion and aphonia."

Dr. T. S. Clouston§ makes the statement that out of 1,082 deaths in the Royal Edinburgh Asylum, between 1842 and 1861 inclusive, 315, or nearly one-third, were due to phthisis.

* "Pathology of Mind," p. 113.

† "Insanity and Its Treatment," p. 56.

‡ *Medical Record*, March 30, 1895, p. 390.

§ *Journal of Mental Science*, vol. ix, p. 36.

He is satisfied, however, that this number does not actually express the whole number of deaths from phthisis among the insane, for he inspected the post-mortem records of 463 persons who died in this asylum from 1851 to 1863 and found that 277 had pulmonary phthisis. He furthermore states that out of 314 autopsies of the insane who died in the Prague Asylum 151, or 48 per cent., showed evidence of pulmonary phthisis, and that in the time of Georget phthisis was the cause of three-fourths of the deaths among the insane inmates of the Salpêtrière.

In the *post-mortem* lesions of 168 melancholics Esquirol* found the cranium 14, the chest 82 and the abdomen 72 times involved. The diseases to which this number of melancholics succumb were adynamic fever, 10; marasmus, 24; phthisis pulmonalis and chronic pleurisy, 62; diseases of the heart, 16; chronic phlegmasia of the abdomen, 32; scurvy, 26, and apoplexy 6. He states that a great many melancholics die from phthisis pulmonalis, whilst organic changes in the brain are rare.

In a more recent and very interesting contribution to this subject Dr. H. A. Tomlinson,† Superintendent of St. Peter's State Hospital for the Insane, St. Peter, Minn., records the results of the lung condition in 72 post-mortem examinations made in that institution since January, 1895, and among these there was chronic degeneration of the lungs in 47 cases, or in 60 per cent. of the whole number. Besides these, six deaths were caused by lobar pneumonia and one by pulmonary œdema.

Not only is it true that these two diseases are so closely allied pathologically that pulmonary consumption follows insanity in the individual in from one-half to three-fourths of the cases, but on investigation it will be found that they also

* "Treatise on Insanity," p. 225.

† *Transaction of American Medico Psychological Association*, May, 1898.

correspond in greatest age-prevalence. In other words, mortality statistics show that the human constitution is more vulnerable to both at certain periods of life than at others. Thus, in 17,711 cases of phthisis collected from the Health Reports of the City of Philadelphia and of the State of Rhode Island, it is found that the greatest mortality among these prevailed between the ages of twenty and thirty. And James* shows by a curve, based on 3,985 inmates of the Morningdale Lunatic Asylum of Scotland, that the greatest

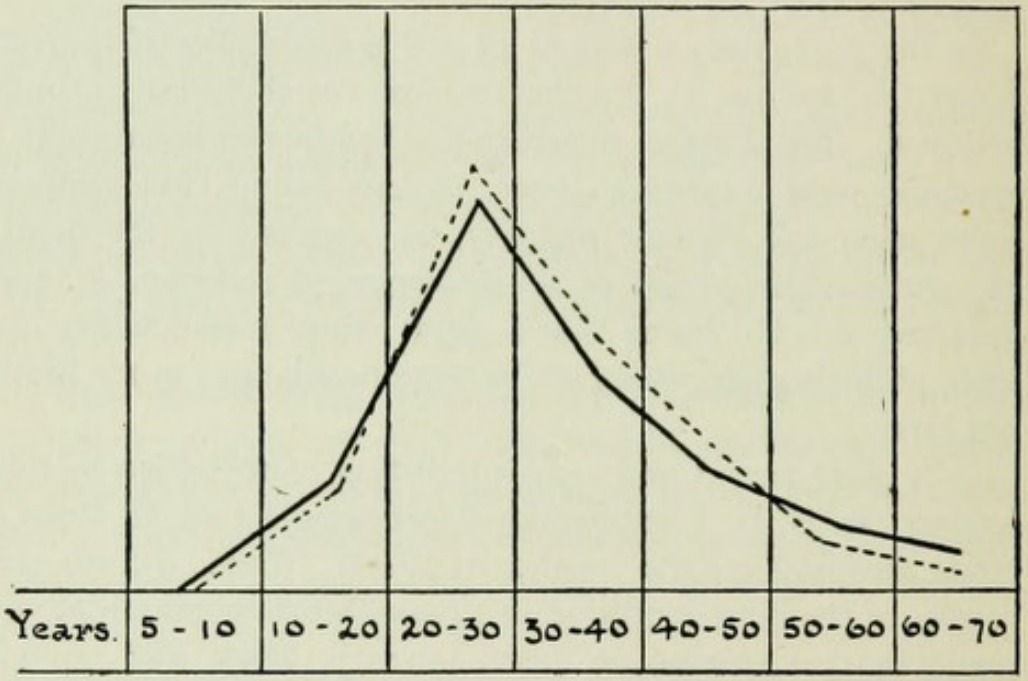

Fig. 3.—Age liability of pulmonary consumption and insanity—the dotted line represents the former and the solid line the latter disease.

number of lunacy cases develop between the ages of twenty-five and thirty-five. The *Report of the Pennsylvania Hospital for the Insane,* for the year 1888 gives the ages at which insanity first appeared in 9,543 patients admitted to that institution during its existence and shows the greatest prevalence of insanity to be between twenty and thirty years.

The figure shown above gives a comparative view of the relative frequency of these two diseases at different age-

* "Pulmonary Phthisis," p. 41.

periods. The cases of pulmonary consumption on which the dotted line is based number 17,711, and are derived from the sources above indicated; and the cases of insanity on which the solid line is based number 9,543, and are obtained from the Report of the Pennsylvania Hospital for the Insane for 1888. The fact is clearly brought out here that the two curves pursue a similar course throughout life, beginning at the base of the figure, between five and ten years, reaching the highest apex-point at between twenty and thirty years, and then gradually declining until the lowest level is reached, between sixty and seventy years.

The anatomy of phthisis bears another striking analogy to that of insanity in regard to the smallness of the heart and the delicacy of the arterial circulation. On the subject of the size of the heart in phthisis Louis* asserts that "in 112 cases where death was caused by phthisis we have only found three examples of an evident increase in the size of the heart. This increase was confined to the left ventricle, and might be estimated at one-third or one-fourth of the normal size of the organ. . . . Diminution of the volume of the heart was evident in the great majority of instances, both in patients whose disease had progressed slowly and in others where the fatal termination was not protracted beyond a few months." In confirmation of this Rokitansky† states that the anatomical predisposition to tuberculosis is characterized by "a long thoracic cavity, smallness of the heart, structural delicacy of the arterial vessels and external coverings, weakness of the muscles, etc."

Furthermore, von Wolff‡ makes the statement in a very interesting contribution to the cardiac anatomy of psychiatry, entitled "Die Hypoplasie des Herzens bei Geistes-

* "On Phthisis." Revised by Bowditch, p. 49.

† "Lehrbuch der Pathologischen Anatomie," 1858, Bd. 1, p. 303.

‡ *Allgemeine Zeitschrift f. Psych.*, Bd. li, S. 447; *Neurolog. Centralblatt*, Bd. 14, 1895, S. 606.

schwachen," that the heart weighs considerably less among the insane than among the sane. Thus in 123 hearts taken from the bodies of insane persons he found the ratio between the body and the heart-weight as 1 to 0.00351 grammes, while in the healthy the ratio is 1 to 0.00487 gr. The stature in relation to the heart-weight he found as follows: Among the insane 1cm. of the body-length equalled 1.13 gr. heart-weight, while in health 1 cm. equals 1.82 gr. heart-weight.

Another marked similarity between phthisis and insanity is the fact that one may be wholly translated into the other in the same person. This is well shown in the following histories:

Case 196. Dr. Isaac Ray* states that a female presented symptoms of pulmonary disease and soon after cerebral symptoms. She became so excited that she was sent to an asylum. The excitement abated under treatment, and she went home as rational as ever, but the pulmonary disease returned and she subsequently died of pulmonary phthisis.

Case 197. A male†, 25 years old, made a slow recovery from pneumonia. In the following year all objective signs of the pulmonary disease had disappeared. He then became melancholic, and his head was turned spasmodically to the right. He finally died with dyspnœa and dysphagia. On examination his brain was found congested and œdematous and there was a cavity in the apex of the right lung.

Case 198. Dr. Edward N. Brush‡ reported the case of a female, aged 21, who became melancholic, and received asylum treatment for nine months. During this time she improved and became perfectly rational during the last three months of her hospital residence. There was now observed an evening rise of temperature and an infiltration of the lung

* *American Journal of Insanity*, 1862, vol. xix, p. 40.
† *Schmidt's Jahrbücher*, suppl., 1847, S. 72.
‡ Private communication.

apices. She rapidly sank from consumption, but there was no recurrence of melancholia.

Case 199. Dr. Voisin* relates the case of a female who after suffering from melancholia, with stupor, for four years, was attacked with pulmonary consumption but recovered from her insanity.

The close intimacy that exists between phthisis and insanity is still further demonstrated by the fact that these diseases develop hand in hand among the colored people in our Southern States. This opinion is based on the following information which has been collected from time to time. On this subject Dr. T. O. Powell,† Superintendent of the Lunatic Asylum of the State of Georgia, says: "There has been a radical change in the susceptibility to certain diseases, notably insanity, phthisis and similar maladies in this class of our population, from which they were almost entirely exempt up to 1867. . . . The census of 1860 will show that there were only 44 insane negroes in the State of Georgia, or 1 insane negro in every 10,584 of the population, and consumption in the full-blooded negro was rarely seen. The census of 1870 shows 129 insane negroes in this State, or 1 in every 4,225 of the population. The census of 1880 gives 411 colored insane, or 1 in every 1,764 of the population, while in 1890 there were 910 colored insane, or 1 in every 943 of the population."

Dr. J. F. Miller,‡ Superintendent of the Eastern Hospital of North Carolina states that "from close personal observation, embracing a professional life of nearly forty years among the negroes, and from data obtained from professional brethren in different sections of the South, I have no hesitancy in declaring that insanity and tuberculosis were

* *British and Foreign Med.-Chirurg. Review*, lix. 59. p. 117.

† "Report on the Increase of Insanity and Its Supposed Causes."

‡ "The Effects of Emancipation upon the Mental and Physical Health of the Negro of the South," *North Carolina Medical Journal*, Nov. 20, 1896.

rare diseases among the negroes of the South prior to emancipation." This writer furthermore declares that the Eastern Hospital of North Carolina was opened August 1, 1880, for the exclusive accommodation of the colored insane; that during the first year there were admitted 91 insane negroes, which number represents the accumulation of this class of patients in that State during the first decade and a half after the Civil War; and that in 1885 there were under treatment in this institution 144; in 1890, 244; in 1895, 307, and in December, 1896, 377 insane negroes.

Dr. J. W. Babcock,* Superintendent of South Carolina Insane Hospital, says: "We cannot lose sight of the fact that on the basis of the census, as compared with insanity in the whites, mental disease in the negro has risen from one-fifth, as common in 1850, to one-half as common in 1880 and 1890."

Moreover, Dr. Miller (*op. cit.*) states that in the hospital in his charge the average mortality from consumption since its opening to 1896 is 25 per cent. of the total number of deaths. But the death-rate from this disease was much less in its early management; for up to 1884 it caused 14 per cent. of the total number of deaths, while in 1895 it produced 27 per cent. of all the deaths, and this in spite of a reduction of the general mortality-rate.

Dr. Powell declares (*op. cit*): "From observation and investigation I am forced to believe that insanity and tuberculosis are first cousins, or at least closely allied. The sudden outburst of insanity with the colored race of the South came associated with tuberculosis, hand in hand, keeping pace one with the other; hence, in obtaining histories of cases as they are brought to our institution, the hereditary predisposition to consumption is carefully inquired into. The prognosis of phthisical insanity is unfavorable. I am not surprised at

* "Tuberculosis in Asylums," *American Journal of Insanity*, October, 1894.

any time to find phthisis in a family strongly predisposed to insanity."

Dr. T. J. Mitchell,* Superintendent of the Mississippi Lunatic Asylum, says that among the negro patients of this institution the following number of deaths occurred in the years here named: In 1892 there were 44 deaths, and 14 of these, or about 32 per cent., were caused by consumption. In 1893 there were 29 deaths, and 16 of these, or about 55 per cent., were caused by consumption. In 1894 there were 40 deaths, and 18 of these, or 45 per cent., were due to consumption. In 1895 there were 35 deaths, and 11 of these, or about 35 per cent., were caused by consumption. In ten months of the fiscal year of 1896 there were 48 deaths, and 20 of these, or about 48 per cent., were the result of phthisis. These data show that about 42 per cent. of the total number of deaths in this institution were caused by consumption during these years, and estimating the death-rate among the white population from this disease at 20 per cent., the death-rate among the insane negro population is 22 per cent. greater.

Dr. E. D. Bondaurant,† Assistant Superintendent of Alabama Insane Hospital, says: "During three years and nine months, beginning October 1, 1890, 295 deaths occurred among the insane patients treated in this hospital. Of the 179 deaths among white patients, 51, or 28 per cent., were due to tuberculosis; of 116 deaths among negro patients, 49, or 42 per cent., were due to tuberculosis. In addition to this, a study of our clinical records discovers the fact that in the colored race the disease assumes a much more active and rapidly progressive form, the average duration of fatal cases being markedly shorter in the negro."

Dr. W. H. Barnes,‡ First Assistant Physician in Arkansas

* Cited from Dr. Miller's paper; see above.

† Also cited from Dr. Miller's paper; see above.

‡ In a private communication.

Lunatic Asylum, states that neither insanity nor pulmonary consumption were especially prevalent among the negroes while they were slaves.

Dr. R. J. Preston,* Superintendent of the Southwestern Hospital of Virginia, declares that there has been a great increase in both insanity and phthisis among the negro population since emancipation.

Dr. James D. Moncure,† Superintendent of the Eastern Hospital of Virginia, informs the author that the health of the negro, with regard to insanity and phthisis, was very much better before emancipation than since. Before that time there were about 60 insane negroes in the asylums of Virginia. Now (1896) there are over 1,000. In all hospitals the insane seem readily to contract tuberculosis, and this is a frequent cause of death among the insane.

From these facts the following conclusions may be safely drawn: (1) That insanity and consumption were comparatively infrequent in the negro race before the Civil War; (2) that both of these diseases have disproportionately increased in the same race since that time; and (3) that in all probability the causes which give rise to one of these diseases also produce the other.

Asthma.

Asthma is a paroxysmal dsypnœa which is usually followed by inflammation of the bronchial mucous membrane. While nominally a disease of the lungs or of the bronchial tubes it is essentially a neurosis and rather belongs to the domain of nervous than to that of pulmonary pathology. It is more than this. It occupies a middle position between organic disease and psychical disorder, inasmuch as it frequently develops into pulmonary consumption on the one

* Private communication.

† Private communication.

hand and strongly predisposes to insanity on the other. In confirmation of the former Fuller* states that, contrary to the belief that asthma and pulmonary consumption are antagonistic, many asthmatics die of the latter disease. Williams,† in tracing the origin of 385 cases of pulmonary consumption, shows that seven began with spasmodic asthma. On page 61 of the same work he declares that the tendency of asthmatic patients to have tuberculous children is hardly sufficiently recognized. James‡ asserts that asthma and whooping-cough are likely to terminate in or to predispose to pulmonary phthisis. The author's experience would seem to confirm the opinion of the above-named authorities. These facts demonstrate then that asthma is translated into pulmonary consumption, and the following histories of cases will serve to make it clear that it is also transmutable into insanity:

Case 200. Kelp§ reports the case of a male, aged 28, with a family history on both sides, who became asthmatic at 21 and had been taking large doses of morphine and chloral. After some time the asthma disappeared entirely and was superseded by mania. He presented extreme depression and anxiety, amounting to desperation, accompanied by a delusion that he was being poisoned. After a time the asthma reappeared and his mental condition gradually improved.

Case 201. Dr. Lorent‖ gives the history of a male adult, who had long suffered from asthma, fell ill of melancholia, when his chest troubles vanished. Nine months afterward he recovered from the mental affection and the asthma returned. A year after this the chest troubles disappeared again and the melancholia recurred.

* "Disease of the Chest," 1862, p. 323.
† "Pulmonary Consumption," 1887, p. 317.
‡ "Pulmonary Phthisis," p. 57.
§ *Zeitschrift f. Psych.*, xxix, 4.
‖ *Ibid.*

Case 202. Dr. H. B. Nunemaker,* Philadelphia, saw a male, aged 55, who presented paroxysmal mania for about four weeks, when attacks of spasmodic asthma came on and displaced the mania. These alternations occurred once in about eighteen months, and took place three times while the patient was under observation.

Case 203. Dr. J. C. Stevens,† Harrisburg, Pa., attended an intemperate male adult, who was asthmatic for ten years, after which he became insane. He had various delusions, and was subject to paroxysms of abuse and excitement. Occasionally, in the midst of these, an attack of asthma, lasting for a few days would come on. At these times he was apparently sane. After the asthma disappeared the mania returned. Finally the asthma became more or less constant, and the patient remained sane.

Case 204. Conolly Norman‡ gives the history of a female, 45 years old, who suffered from asthma for twenty years. Three months before she came under observation her asthma ceased suddenly and she became restless, anxious and melancholic. In two and a half years her asthma returned, but her mental condition did not improve.

Case 205. A. Robinson§ attended a female, aged 50, a victim of frequently recurring asthma since girlhood. At her climacteric period she began to suffer from acute mania, which lasted for six weeks, and during this time she was free from asthma. After this short immunity the latter returned with all its previous intensity.

Case 206. Conolly Norman‖ saw a male, aged 32, who had asthma from childhood. The asthma ceased, and he became gloomy, irritable and was haunted by the thought that

* Private communication.
† Private communication.
‡ *Journal of Mental Science*, vol. xxx, p. 1.
§ *Ed. Med. Journal*, April, 1888, p. 900.
‖ *Journal of Mental Science*, vol. xxxi, p. 1.

he was to kill his mother. A fortnight after admission to the hospital he had an attack of asthma. He improved mentally at once, became cheerful and the asthma recurred frequently, but his mental condition remained undisturbed.

Case 207. Male,* about 20 years old, had asthma for some time before he became insane. After being confined in an asylum for three years he became perfectly calm, and the asthma returned and continued for years.

Case 208. Female,† became asthmatic at the age of 30, and suffered from this until she was 40, when, on account of some financial distress, she became insane, suffering from delusions of suspicion. After about six-months' seclusion her mind cleared up. During this time she had no asthma. Her asthma returned, but so far as is known she had no recurrence of insanity.

Case 209. Male,‡ aged 30, had been a sufferer from asthma for many years. Two years before admission to hospital the asthma became less severe, and his friends date his insanity from that time. After being confined for a year or more his mind gradually cleared up, and the asthma returned in a severe form. The chronic, calm condition of partial dementia was well established before the onset of the asthma and the mind remained unchanged.

Case 210. Clouston§ states that a female became asthmatic at the age of 13. At 37 she became melancholic, with delusions, and the asthma ceased.

Case 211. A boy,‖ 1 year old, had an attack of infantile paralysis, followed by impairment of growth of the bones and muscles, with weakness and contracture of the right arm and lameness of the right leg. At puberty he became asthmatic;

* *Ibid.*
† *Ibid.*
‡ *Ibid.*
§ "Neuroses of Development," p. 122.
‖ *Ibid.*, p. 24.

at 19 the asthma ceased and he had a severe attack of melancholia, attended with excitement. This lasted for six months, before and during which time he became thin and anæmic. As the melancholia passed off the nutrition improved, and the asthma returned.

CHAPTER VIII.

ETIOLOGY CONTINUED: INFLUENCE OF SECOND-CLASS OF CAUSES. DUST-INHALING OCCUPATIONS, AND ENTRANCE OF FOREIGN BODIES INTO THE BRONCHIAL TUBES.

DUST-INHALING OCCUPATIONS. Comparatively few cases of phthisis arise on account of exciting causes of this kind, and those which are produced in this way it seems as if the nature of dust is of greater importance in the operation than the quantity. Thus, in a table* which gives the comparative mortality from phthisis of men from 25 to 65 years of age, who were engaged in various dust-inhaling occupations, Dr. Ogle shows the following results.

Fishermen†	55
Coal-miners	64
Carpenters, joiners	103
Bakers	107
Cotton workers	137
Wool workers	130
Masons, bricklayers	127
Stone and slate quarry-workers	156
Pottery workers	239
Cutlers (scissor makers)	187
File-makers	219
Cornish miners (metal miners)	348

From this we learn that coal-miners enjoy a greater freedom from phthisis than any of the other class of laborers here named, excepting the fishermen; that, as a rule, carpenters and bakers are less liable to this disease than are workers in wood or cotton; that the two latter occupations

* Reported at session of International Congress of Hygiene in London. *Boston Medical and Surgical Journal*, April 5, 1894, p. 339.

† The death-rate of phthisis among fishermen, whose occupation is free from dust, is taken as a standard.

are more prone to produce it than those of the bricklayer and of the mason; that in potters the mortality from this disease is nearly twice as great as among the last-named, while among workers in iron, like in the case of the Cornish miners, the death-rate rises still higher.

On the whole it appears that the inhalation of dust arising from vegetable or animal sources is less noxious in this respect than dust which is given off by earthy, or metallic substances, of which iron is a striking example. More than this, it seems as if the occupation of mining and of manufacturing coal is a protection, in a certain sense, against the disease in question. Here is a class of men and boys who are living in and breathing an atmosphere black with coal-dust and smoke during every active day of their lives. So vigorously is this dust inhaling process carried on in coal-miners that the author has personally witnessed the lung-apices of a mine employee, who died from aortic aneurism, so densely permeated with particles of coal as to cause grating of the knife when an incision was made into them, and on the cut surfaces of which the pieces of carbon were seen to glisten and sparkle in the sunlight. The tenacity with which coal-dust adheres to the walls of the air passages during life is not less remarkable, for every practitioner in the coal-fields has seen instances in which coal dirt was expectorated off and on for more than half a dozen years after work in the mines had been abandoned. Yet so far as it can be ascertained no actual disease is ascribable to the influence of this foreign material in any of these cases beyond some dyspnœa. Nor is coal dirt productive of any greater harm on other parts of the body. It is well known that cicatrices of the skin, as the result of gunpowder and of coal wounds, generally remain in a state of black discoloration for life without causing any disturbance. The author has also observed that two wounds co-existing in the same part of the body, one being made by coal and the other by iron or rock, the one

produced by the former unites more promptly than the one caused by either of the latter agents.

In view of the fact that the inhalation of foreign particles of matter is almost universally followed by disease of the lungs, it is certainly very extraordinary that coal-dust should be comparatively innocuous in this respect. One might be tempted to seek an explanation for this immunity in the influence of carbonic acid and other gases which are more or less present all the time in coal-mines, and which have been recommended by high authorities in the treatment of phthisis, if it were not for the fact that those who are engaged in the manufacture of coal in breakers outside of the mines, and who do not inhale these gases but are exposed to as much dust as those who work underground, are just as little liable to pulmonary affections.

It appears obvious, therefore, that dust itself is not such an active cause of phthisis as one might be led to anticipate; but that the power to bring this about lies in the nature and peculiar action of certain kinds of dust, among which iron and clay are prominent examples, while other dust like that of coal may be inhaled with almost perfect impunity.

Entrance of Foreign Bodies into the Bronchi.

The inspiration of foreign bodies, like pins, needles, buttons, nails, coins, seeds, pieces of bone, tacks, etc., into the bronchial tubes always leads to local pulmonary disorders, and frequently to pulmonary phthisis. The nerve-reflexes which are called forth by the irritation of such bodies are both intense and widespread, and manifest themselves in attacks of suffocation, which may end in sudden death, violent paroxysms of cough, aggravated dyspnœa, nausea, vomiting etc. In reporting his own case Dr. A. Sander* states that almost immediately after the inspiration of the foreign body

**Deutsches Archiv f. klin. Medicin*, Bd. xvi, S. 333.

(a small, horny button in his case) his appetite left him, and his nausea became so marked that even the sight of food disgusted him. On the very day, however, on which the body was expelled, after being impacted in the right lung for seven months and after doing a large amount of local injury, he began to consume inordinate quantities of food, and his nausea abated at once, together with the paroxysmal cough and other symptoms which had been incited by it. Evidently all these manifestations were kept up largely by the morbid nervous impulses which were generated by the offending body.

CHAPTER IX.

ETIOLOGY CONTINUED: INFLUENCE OF THE THIRD CLASS OF CAUSES. INFECTION.

INFECTION. The tubercle bacillus is regarded in many quarters as the most active cause of phthisis at the present day. That this micro-organism or some accompanying virus possesses infective properties there can be no doubt, but whether its influence as an etiological factor is as potent and as widespread as it is supposed to be is another question which will now be considered under the headings of inoculation, feeding, inhalation and contagion.

INFLUENCE OF TUBERCLE-INOCULATION. That tubercle can be propagated in animals through inoculation is beyond controversy; although Koch* declares that "phthisis such as is found in man cannot, on the whole, be produced in animals." He attributes this to the fact that tuberculosis does not develop in the same way in one animal species as in another. On the other hand, it appears to be much more difficult to successfully inoculate human beings than animals with tubercle. Thus Lepelletier,† Goodload and Deygallières‡ inoculated themselves with tuberculous or scrofulous material without suffering any ill effects. The first inoculation with genuine tubercle on record probably was accidental and was practised by Laennec on himself accidentally, about

* *Berlin. klin. Wochenschrift*, 1885, Nos. 37a, 37b, S. 22.

† "Traité complete de la maladie scrofuleuse," 1830, cited after Waldenburg.

‡ "Theorie nouvelle de la maladie scrofuleuse," Paris, 1829, cited after Waldenburg.

twenty-five years before his death, the cause of which was pulmonary phthisis. He describes this experience in the following felicitous language*: "While examining some vertebræ containing tubercles I grazed slightly the forefinger of the left hand by a stroke of the saw. The scratch was so small that I paid no attention to it, but on the following day it was slightly inflamed and there gradually formed in it, and almost without pain, a small, roundish tumor, apparently confined to the skin, and which at the end of eight days was of the size of a large cherry stone. At this period the epidermis cracked and showed us the small tumor within, which was yellowish, firm and in every respect like a crude yellow tubercle. I cauterized it with the deliquescent hydro-chlorate of antimony and felt no pain from its operation. At the end of a few minutes, however, after the fluid had penetrated the whole substance of the tumor, I detached it by gentle pressure. The part soon healed, and I have since found no further effects from the accident."

In 1834 Albert† contributed five cases of dissection inoculation of tubercle, with negative results.

In 1872 Demet, Paraskeva and Zallonis‡ inoculated a man 55 years old with tubercle, who was apparently free from tuberculosis but who was dying of gangrene of the left foot on account of obliteration of the femoral artery. Three weeks after the inouclation, signs of tuberculosis began to manifest themselves in one lung, and on the thirty-eighth day after the inoculation the patient died of gangrene and a post-mortem examination showed tuberculosis of both apices.

Furthermore, Lindmann,§ E. Lehmann,‖ Hofmokl¶ and

* "Treatise on Diseases of the Chest," Third Eng. edition, p. 336.

† *Rust's Magazine*, 1834.

‡ "De l'inoculabilité de la tuberculose," *Gaz. Méd. de Paris*, No. 17, 1872.

§ *Deutsche med. Wochenschrift*, 1883, No. 30.

‖ *Deutsche med. Wochenschrift*, 1886, No. 9–13.

¶ *Wien. med. Presse*, 1886, No. 22–23.

Elsenberg* contribute cases in which exhaustion of circumcision wounds by the mouths of phthisical operators was followed by tubercular infiltration and caseation of the inguinal glands.

Influence of Tubercle-Feeding. Tuberculosis may also be communicated to man and animals through feeding with tuberculous material. Herterich† relates the case of two children who, on account of their phthisical mother premasticating their food, became phthisical. Baumgarten,‡ Imlach,§ Zippelius,‖ Demme,¶ Uffelmann,** and Ebstein†† saw typical tuberculosis produced by feeding animals chiefly with milk coming from tuberculous cows. Instances are also recorded where chickens and dogs consumed the sputum of phthisical persons and became tuberculous. So far, however, as milk is concerned as an infective agent, the evidence seems rather conflicting. For if milk is liable to convey tubercular infection to any great extent, then it necessarily follows that animals which subsist on this food almost wholly should show confirmatory evidence of tuberculosis. But the contrary is apparently true. For Goring, in his extensive report on the sanitary condition of animals in Bavaria, states‡‡ that in 1878, among 1,000 bulls, the same number of bullocks, cows, heifers and calves, 5.84, 1.39, 2.50, 0.35 and 0.09 per cent. were tuberculous respectively. The same authority reports that in 1877 of 4,976 cattle recognized as tuberculous

* *Berlin. klin. Wochenschr.*, 1886, No. 35.

† *Münch. arztl. Intell. Bl.*, 1883, No. 236.

‡ *Centralbl. f. k. Med.*, 1884, No. 2.

§ *British Med. Journ.*, 1884, July 26.

‖ *Wochenschr. f. Tierheilk. u. viehz.*, Bd. 20, S. 205.

¶ "Ber. über d. Thatigkeit d. Jennerschen Kinderhosp.," i. Bern, 1879, S. 27.

** *Archiv f. Kinderheilk.*, 1880, i, S. 414.

†† *Prager Vierteljahrsschrift*, 1878, S, 115.

‡‡ See "Influence of Heredity and Contagion on the Propagation of Tuberculosis." Lydtin and Fleming van Hertsen.

64, or 1.31 per cent. were less than one year old; 551, or 10.2 per cent., from one to three years old; 1,730, or 34.5 per cent., from three to six years, and 2,360, or 46.5 per cent., more than six years old.

Strobl* and Magin report that in 1879 out of 1,125 tuberculous cattle slaughtered in Munich 2, or 0.2 per cent., were under one year; 81, or 7.1 per cent., from one to three years; 378, or 33.5 per cent., from three to six years; 604, or 59.2 per cent., were over six years old. Among 160,000 calves examined at the Munich abattoir 2 were found tuberculous in 1878, 1 in 1879, none in 1880, none in 1881, and 2 in 1882.

From the above statistics these authors come to the following conclusions:

(1) About 2 per cent. of the cattle sent to these abattoirs are tuberculous.

(2) The greatest number of tuberculous animals are more than six years old.

(3) After these the largest number is from three to six years old.

(4) Very young animals are almost exempt from tuberculosis.

It is not necessary to go into statistics to show that the greatest liability to phthisis in the human family, like that in the lower animals, does not correspond with that period of life when the greatest amount of milk is used. The largest quantity of milk is consumed in infancy, yet the greatest age-liability to pulmonary phthisis in the human race comes between twenty and thirty years, and the disease is practically absent during early infancy. This is true of America, England and of all continental countries of which we have reliable records.

The ingestion of tuberculous meat as a source of tubercle-infection is a point of great practical importance, although the chief interest in this question is not so much the possi-

* Cited from Lydtin and Fleming van Hertsen, p. 57.

bility of conveying tuberculosis through eating meat of this kind as it is whether infection through this channel occurs practically in every-day life. A great many experiments have been made on this subject and the evidence which has been gathered from them is rather contradictory. Taking the work of Chauveau, Gerlach, Klebs, Zürn, Bollinger, Möller, Brell, Roloff, Orth, Toussaint, Aufrecht and others as a basis, Johne* makes the following analysis: In 259 animals fed on raw material 47.7 per cent. of the experiments were followed by positive, 48.9 per cent. by negative, and 3.3 per cent. by uncertain results, while in 62 experiments, in which the tubercular meat was boiled for 10-15 minutes, 35.5 per cent. were followed by positive, 64.5 per cent. by negative, and 1.6 per cent. by indefinite results. From this he draws the following conclusions: (1) Tuberculosis may be communicated from animal to animal and from man to animal through the ingestion of tuberculous material, but less successfully than through inoculation; (2) tuberculosis is more readily conveyed through tuberculous meat than through milk.

In Germany, in the seventeenth century, severe laws were enacted against the sale of flesh of tuberculous cattle, which provided that carcasses of cattle that showed the least trace of the disease were not allowed to be handled by butchers, and were committed to the care of the public executioner for burial. These draconic measures weighed very heavily against the breeders and feeders, and, stimulated by the report that the executioners did not bury the diseased carcasses, but sold them, and consumed them even in their own families without apparent injury, an intense feeling was aroused against these regulations, and in the middle of the eighteenth century a strong public protest was made against them. Prominent medical men of that period, among whom were

* "Die Geschichte der Tuberkulose mit besonderer Berücksichtigung der Tuberkulose des Rindes und die sich heran knüpfenden Medizinal und Veterinärpolizeilichen Konsequenzen," Leipzig, 1883.

Zink (1764), Ruhling (1774), Heim (1782), and Granmann (1784), took active interest in the matter and taught that tuberculosis was not contagious, and that the flesh from such animals might be eaten with impunity. Certain physicians publicly advertised the good quality of such flesh. Zwierlein, a doctor of medicine and of philosophy, took twenty-five pounds of the flesh of a tuberculous ox and consumed it himself in order to demonstrate that such meat is not prejudicial to health. He also prepared a quantity of broth from tuberculous nodules and drank the same in the market place before a large assembly of people, without doing himself any harm.

The popular pressure became so great against these laws that in 1785 (June 27th) the government of Prussia, as well as that of other German States, rescinded them. The proclamation that was issued by Prussia stated that, in case of tuberculosis existing in a carcass, the diseased portion should be removed and that the remainder should be used as food. In a circular made public by the Imperial Government of Lower Austria (June 11, 1788) it was stated that tuberculous growths are found in the healthiest animals, as well as in the diseased, and that the flesh of the latter is in itself healthy. If, however, tuberculous animals are cachectic, their flesh should be considered as injurious to the health of mankind. In Southern Germany, Austria and Switzerland the flesh of tuberculous animals was always more or less rejected, although it was admitted at the same time that the flesh of cattle in good condition, even if some tuberculous nodules were found in the pleura or peritoneum, was not unfit for food after the nodules were removed.*

It is interesting to observe how clearly the views which were held in these countries a century ago regarding the non-injurious effects of meat, when slightly tainted with tuber-

* The above historical facts are cited from Lydtin and Flemingvan Hertzen (pp. 104-106) already quoted.

culosis, correspond with the conclusions which have been reached by the health authorities of one of the most modern Continental cities. In 1894 the Comité Consultatif d'Hygiene of Paris decided that unless an animal is thoroughly invaded by tubercles it is not to be condemned, but the diseased portions are to be removed and the remains to be sold for food. According to M. Lefevre, veterinary surgeon at Havre, the minimum of tuberculous animals killed at the principal French slaughter-houses amounts to 4,000; of these only 1,000 were condemned, and, therefore, 3,000 were offered for sale after the parts presenting tuberculous lesions had been removed.*

Influence of Tubercle Inhalation. Tuberculosis may also be communicated to animals through the inhalation of air loaded with finely divided particles of tubercle, or through the introduction of the same into the respiratory passages by means of tracheal fistulæ. This method of infection is less effectual, however, than that of inoculating or of feeding tubercle. Schweninger† produced tuberculosis in dogs who were made to inhale pulverized phthisical sputum. Tappeiner‡ confined dogs a short time each day, for from twenty-five to fifty days, in a small room in the atmosphere of which fine particles of phthisical sputum were suspended. Tuberculosis was developed in the lungs, liver, spleen and kidneys of these animals. Schottelius§ repeated Tappeiner's work, with some variation. Instead of causing his dogs to inhale the sputum from tuberculous persons only, some were made to respire the sputum or bronchitic, but non-tuberculous persons; others, particles of Limburger cheese sus-

* Letter from Paris, *British Med. Jour.*, March 10, 1894, p. 548.

† "Ueber künstliche Erzeugung der Tuberkulose." Gesammelte Arbeiten von Dr. E. Schweninger, Bd. 1, p. 242. Berlin, 1886.

‡ Ueber eine neue Methode Tuberkulose zu erzeugen," *Virchow's Archiv*, Bd. 74, p. 393: 1878.

§ "Experimentelle Untersuchungen über die Wirkung inhalierter Substanzen," *Virchow's Archiv*, Bd. 73, 1878, p. 524.

pended in air, and still others were made to breathe finely-powdered brain substance. In all these cases nodules, analogous to miliary tubercle, were developed. These results call into question somewhat the soundness of the experiments of Tappeiner, as the latter indeed proved by his subsequent observations.*

Celli† and Guarnieri devised the following experiments to test the question of tubercle infection through inhalation: In the first series of experiments they placed three ventilating devices, the inner surfaces of which were well covered with Koch's culture gelatin, at different elevations in the chamber of a consumptive patient. The air of the room was strained through these devices for twelve nights in succession. Part of the gelatin, at the end, of this time, was examined microscopically, and part was inoculated into the eye, the peritoneal cavity and into the subcutaneous connective tissue of rabbits and of guinea-pigs.

The second series consisted in allowing a number of phthisical patients to respire for a long time into a reservoir, which was well coated with Koch's culture of gelatin, after which the gelatin was examined microscopically and inoculated in the same manner as in the previous series.

In the third series they experimented with air which was aspirated through tubes containing tubercular sputum. The sputum was evaporated by heat, and the air thus exposed was forced through a tube containing sterilized blood serum and Koch's gelatin. The experiments were continued for many hours, and in many instances were repeated. The culture media were then tested in the same manner as in the two former series, *viz.*, by microscopic examination and by inoculation.

* "Neue experimentelle Beiträge zur Inhalationstuberkulose der Hunde," *Virchow's Archiv*, Bd. 82, p. 353; 1880.

† "Sulla presenza del bacillo de tubercolo ne'varii prodotti tubercolari," *Gaz. d. Ospital*, 1883, No. 37 and 40.

Every one of these experiments was followed by negative results. In no case did the microscope reveal a bacillus, nor was tuberculosis produced in any of the animals which were inoculated. Bollinger repeated the work of these two investigators on a vast scale, and confirmed their results in every particular.

Influence of Man's Exposure to Contagion. The question of the contagiousness of phthisis under the ordinary circumstances of life is one of great moment. It affects the happiness of more people than any other problem within the domain of medicine. It is one, too, of great antiquity, although its fortunes ebbed and flowed at random until the last third of the past century, when it received a fresh impulse through the researches of Villemin, and later from those of Koch and his followers. That the disease is transmissible from man to animal, and from animal to animal, is very conclusively proven by what has been said on the subject of the inoculation, feeding and inhalation of tubercle. And these facts have given rise to the deep-seated and prevalent belief that phthisis is a most contagious disease, and that if measures of isolation and disinfection are properly enforced the disease, as is held by some, will be exterminated in a single generation. By common consent it must be admitted that if this doctrine of the stamping out of this disease is correct it should be regarded as a sacred trust, and should receive the sanction and encouragement of every loyal citizen. If, on the other hand, it is erroneous, it is equally clear that those who are afflicted with the disease are terribly wronged and injured and that the public is deceived concerning the true nature, cause and prevention of consumption.

Now, while it is admitted that the evidence which comes from experimental data is undoubtedly in favor of the contagious origin of phthisis, it must not be overlooked that before coming to a final conclusion on this subject the evidence which emanates from its clinical side, and which is just

as legitimate and scientific, must also be consulted. In invoking the assistance of this testimony it is perfectly logical to start out with the fundamental and self-evident proposition that, if other things are equal, those who are most exposed to a contagious disease are most liable to contract it. To hold the opposite would be a *reductio ad absurdum* of the question. This may be very aptly illustrated by means of accidents due to railway travel. While only a portion of those exposed to such accidents are injured or killed, it still remains true that the mortality-rate from such casualties is higher among those who travel in cars than among those who do not. This principal holds true in the case of smallpox, measles, etc., and is the *experimentum crucis* in the case of consumption, if, like them, it is a disease propagated exclusively or even in a large measure through contagion. On the other hand, it is also proven that the germs, or contagious particles, are most abundant in the environment of consumptives. It is inevitable, therefore, that, if consumption is contagious, physicians, nurses, attendants, intimate friends and relatives of consumptives are more subject to the disease than those not so exposed—a conclusion from which there is no escape.

What, then, are the data on which to base an opinion? Physicians who are constantly exposed to consumption are much less subject to this disease than are others who scarcely come in contact with it, except by chance. The statistics of the Brompton Hospital for Consumption, in London, show that during a period of thirty-six years not a single clearly authenticated case of consumption arose within its walls among its twenty-nine physicians and assistant physicians, its one hundred and fifty clinical assistants and its one hundred and one nurses, of which there existed a health record.* The statistics of Friedrichshain Hospital, in Berlin, as furnished by Dr. Fürbinger, demonstrate that during a period

* Theodore Williams, *British Med. Jour.*, Sept. 30, 1882.

of sixteen years out of 459 male nurses there were 4 (2 of whom were tuberculous before entering); of 339 female nurses, there were 2; of 83 physicians, there were 3 (1 of whom entered with the disease) who became consumptive. Of 108 Victoria sisters, who were engaged as nurses in the same institution from two to five and a half years, only 1 became consumptive.

These figures are also strikingly confirmed by those which come from the private sanitarium for consumption in Görbersdorf, Germany. Dr. Brehmer, who had been in charge of this large institution for twenty years, states* that since the year 1854 more than 10,000 consumptives resided in the hospital, who daily walked the streets of the town and commingled with its inhabitants. The latter were, therefore, continually respiring an atmosphere which was more or less impregnated with tubercle bacilli emanating from the dried expectoration of these consumptive visitors, yet, in spite of these favorable conditions for contagion, it appears that the mortality from this disease is 50 per cent. less among the Görbersdorf population since than it was before the establishment of the hospital.

The same is true of Falkenstein,† a town near Frankfort, Germany, in close proximity to which Dr. Dettweiler located a private sanatorium for consumptives in 1877. The health statistics of this place show that during twenty years previous to the establishment of the institution the death-rate from consumption among the Falkenstein inhabitants was 4.0 per 1,000 living, while for eighteen years since its existence the death-rate from this disease fell to 2.4 per 1,000 living inhabitants.

Dr. Haupt,‡ of Soden, a resort for consumptives in the south of Germany states that among the inhabitants of this

* "Die Aetiologie der chronischen Lungenschwindsucht," p. 19.

† *Münch. med. Wochenschrift*, Oct. 1, 1895.

‡ *Berliner k. Wochenschrift*, March 31, 1890, p. 311.

town there are 101 individuals who let lodgings to consumptive visitors during the summer months. These patients are nursed and cared for chiefly by the inmates of the families—the work of making the patients' beds, cleaning their rooms, beating the carpets, removing the expectoration, etc., being performed by female servants. During the winter months the rooms are reoccupied by members of the landlord's families. From 1855 to 1888—a period of thirty-three years—10 of the 238 members of the local families died from consumption, and 5 of the 415 servant girls died of the same disease, but in none of these instances, so far as could be ascertained, was the malady traceable to contagion.

Dr. J. Adams,* of Colorado Springs, Colorado, states that this place has been a health resort for about seventeen years, and comprises about 11,000 inhabitants, and that the majority of the rooms in the many boarding and lodging houses are and generally have been occupied by consumptives. After a diligent search throughout the whole city he only found a record of seven cases of consumption that originated among the local inhabitants during this time, and so far as could be found out none of these cases were specially exposed.

Dr. P. Langerhans, who practised medicine for nine years in Madeira, an island which is visited every winter season by about 400 consumptives, observes† that these invalids are lodged, boarded and in great part nursed by English colonists, varying from 210 to 250 in number, who live in about 100 houses. The rooms which are occupied by consumptives in the winter are reoccupied during the summer by the colonists' families, thus insuring the closest intermingling of the well with the sick. The health records of this island, which have been accurately kept since 1836, show that four of the English colonists died from consumption during this time

* Private communication in 1891.

† "Zur Aetiologie der Phthisie," *Virchow's Archiv*, Bd. 97, 1884.

and that one of these suffered from the disease before he came on the island.

The testimony which relates to the contagiousness of this disease between husband and wife is of a similar negative character. Dr. Schnyder, of Switzerland, gives a record* of 844 cases of consumption occurring among married people. In 445 of these the husband only, and in 367 the wife only, was consumptive, while in 32 instances both husband and wife were affected, showing that in 812 of these cases there was no proof of contagion. Dr. Schnyder furthermore says that four out of the thirty cases came to him fresh from the matrimonial altar affected with the first signs of consumption, and he believes that in spite of all warnings young people are frequently married while suffering from this disease. Out of 1,000 phthisical patients Cotton† met with 11, 7 men and 4 women, who had previously lost a husband or a wife from this disease. Reginald Thompson,‡ out of 15,000 consumptives, records 15 cases in which wives had been apparently infected from their husbands. Out of 6,167 patients the second report of the Brompton Hospital for Consumption (1863) gives 239 widowed persons, 83 males and 156 females, who had previously lost a husband or wife from phthisis, *i. e.*, 1.7 per cent. Dr. Austin Flint§ contributes the history of 670 cases of consumption affecting husbands and wives, and among these there were only five in which a suspicion existed that the disease might have been contracted from one or the other; but it is certain, he says, that the instances in which transmissibility may be suspected can also be accounted for as coincidences in a disease which is so widespread as consumption. M. Leudet‖ shows, too, that

* *Correspondenz-Blatt f. Schweizer Aertzte*, 1886, Nos. 10, 11 and 12.

† "Consumption," p. 9.

‡ *Lancet*, 1880, vol. ii, p. 727.

§ "On Phthisis," p. 419.

‖ *Medical and Surgical Reporter*, Feb. 1, 1890, p. 142.

out of 112 widows and widowers whose consorts died of consumption only 7 (4 women and 3 men) became phthisical.

THE OPINIONS OF AUTHORS OF TREATISES ON PULMONARY CONSUMPTION REGARDING THE CONTAGIOUSNESS OF THIS DISEASE. In this connection it is of interest to know the conclusions which those have reached on the question of contagion who have made phthisis a life-long study, who have written special treatises on this disease, and who are, therefore, entitled to an authoritative opinion on this subject. Laennec* in his illustrious work says: "We frequently observe, among the poorer classes, a numerous family sleeping in the same apartment with a consumptive patient, and a husband occupying, to the last, the same bed with his wife without any communication of the disease. The woolen apparel and the beds of consumptive subjects, which it is the custom to burn in some countries, are not even generally washed, much less destroyed, in France, and yet I have never seen the disease communicated by them. It would be well, nevertheless, were it merely on the score of prudence and cleanliness, that greater precautions were taken in this respect. It is well ascertained that a disease not usually contagious may become so in certain circumstances." Dr. Forbes, the translator of this work, in a foot-note (p. 336) states: "Although myself sceptical as to the contagious powers of phthisis, from never having witnessed, among the thousands of cases of this disease I have attended, one unequivocal instance of the fact, it must be admitted that the thing is in itself neither impossible nor improbable." Portal† says that he was brought up in the contagious belief but abandoned it. Ancell‡ believes that "the doctrine of contagion has at all times been based on very vague and insufficient evidence, such as isolated cases of the occurrence of the disease in individuals who had pre-

* "Diseases of the Chest," p. 335. Translated by Forbes, 1830.

† "Phthisis Pulmonalis," i. 46: citation from Wilson Fox's "Treatise on Diseases of the Lungs and Pleura," p. 560.

‡ "A Treatise on Tuberculosis," p. 481.

viously been in constant attendance upon the sick, or in husbands or wives, where both had slept in the same bed until the fatal termination of the disease in the one first affected. In appealing to these facts as evidence of contagion no account is taken of the anti-hygienic influences to which the individuals had been subjected or of the probability of a common or independent source of hereditary transmission or of the predisposition or the actual disease acquired previously. Against the few facts which tend to support the doctrine of contagion there are tens of thousands against it." Dr. Aufrecht, after referring somewhat extensively to the contagion theory, says:* . . . and less justifiable are the reckless conclusions drawn by Koch concerning the etiological indication of the tubercle bacillus." Dr. Dettweiler† writes of the freedom from phthisis of those who are engaged in the care of the phthisical in hospitals, and then says: "My own fourteen years' experience in hospitals for consumption is in perfect accord with this." The late Dr. Hermann Brehmer,‡ the founder of the large and world-renowned hospital for consumptives in Görbersdorf, Germany, and who has done so much to place the treatment of consumption on a scientific basis, opposed the contagion theory of this disease most strenuously. Dr. Arthur Ransome§ declares that "at the present time the dread of infection from consumptive persons is out of all proportion to the danger, and goes far beyond what the facts of the case justify. In its results this alarm is likely to cause much injustice to many poor invalids, and in some cases to endanger their prospects of cure. Already persons affected with

* "Die Lungenschwindsucht," Magdeburg, 1887.

† "Die Behandlung der Lungenschwindsucht," Berlin, 1884, p. 9.

‡ See his last three works: "Die Aetiologie der chronischen Lungenschwindsucht," Berlin, 1885. "Die Therapie der chronischen Lungenschwindsucht," Wiesbaden, 1887. "Mittheilungen aus Dr. Brehmer's Heilanstalt für Lungenkranke," Wiesbaden, 1889.

§ "Treatment of Phthisis," London, 1896, p. 25.

almost any chest disease find it difficult to obtain places as domestic servants. The close ties of family affection are not always strong enough to induce the relatives of consumptives to undertake what is considered to be the dangerous duty of nursing them. The sites for consumption hospitals are becoming as difficult to find as those for smallpox hospitals, and utterly unfounded reports as to the spread of phthisis by such institutions are recklessly made, even by medical officers of health." On page 31, in continuation of the same subject, he says: "I have never yet found any satisfactory proof of infection, direct or indirect, in any well ventilated house in this country, and this in spite of close contact, as in the attendance of a wife upon her husband or in the nursing and sleeping together of near relatives and friends." The late Dr. James R. Leaming* uses the following example to illustrate the false notion of tubercular contagion: "A mother, after watching her children, three or four in number, through scarlatina of a severe type, began to cough, lose weight and finally died of phthisis. She was well when the children were taken ill; she was a loving, anxious mother, and as they were attacked successively the time of her anxiety was prolonged. The children all recovered, but the mother was sacrificed. She was not aware of having taken cold. The cough was so insidious that no one could tell when it commenced. Had there been the same prolonged anxiety over a case of phthisis, followed by inconsolable despair at the loss of the loved one, it would have seemed to prove the communicability of consumption. But scarlatina germs do not originate phthisis." Dr. Alexander James† makes the statement that " many examples of contagion, real or apparent, have, of course, been brought forward, but the records of consumption hospitals and the fact that one often sees in a general hospital a phthisical case with numerous

* "Diseases of the Chest," New York, 1887.

† "Pulmonary Phthisis," Edinburgh and London, 1888.

bacilli in his sputum, having alongside of him patients with fibroid, bronchiectatic or syphilitic disease, and yet in whose sputum or lungs no trace of bacilli can be discovered, seem incompatible with a belief in contagion." Dr. Douglass Powell* says: "My own personal experience and observation convince me that, apart from artificial conditions—such as those brought about by experiment—and in the ordinary circumstances of life, phthisis is not an infectious malady." Dr. Wilson Fox† clearly condenses his views, as follows: "There are few writers who have not admitted the possibility of some contagion, but I venture to think that the evidence, as it stands, shows that even if this possibility has an authentic foundation the extent and degree to which contagion ordinarily extends are singularly small. The results observed in the hospitals for consumption, if we compare these with the contagion of typhus, smallpox, scarlatina, erysipelas or puerperal fever, are at once sufficient to support this proposition." Dr. Theodore Williams‡ declares: "My own experience is that for the last twenty years I have carefully watched for cases of infection in hospital and private practice, and though I have come across a certain number of apparent cases they have never stood the test of close inquiry, there being always some additional element to explain the causation of disease."

THE MEANS OF PREVENTION THAT HAVE BEEN INVOKED IN THE PAST BY THE CONTAGION DOCTRINE. It is doubtful whether the history of any medical idea is invested with more curiosity and interest than that which hangs over that of the contagiousness of phthisis. The doctrine is a very old one—dating back to Aristotle's time—but in the last quarter of the eighteenth century it had gathered

* "Diseases of the Lungs," 4th edition, London, 1893.

† "Treatise on Diseases of the Lungs and Pleura," London, 1892, p. 574.

‡ "Pulmonary Consumption," 2d edition, London, p. 88; 1887.

so much force and importance in some of the Italian* States that stringent laws were passed concerning the disinfection of the rooms in which consumptives died, and of the clothes which they had worn. In 1754 the Grand Duke of Tuscany, being imbued with the truthfulness of the contagious doctrine of phthisis, addressed the College of Physicians of Florence on this subject, but the latter, not being able to view the question in the same light, made a negative report on it. In spite of this action an ordinance was passed by the government making it compulsory on all medical practitioners to report, under the penalty of a fine of 100 scudi, every case of "true confirmed phthisis" to the Health Tribunal in Florence, and to the governor, commissioners and magistrates having criminal jurisdiction in the other provinces. In every case of this kind the magistrate ordered an exact inventory of all articles in the patient's room, or used by him, so that a thorough disinfection could be made in case of death. After the consumptive's death those who had taken care of him, and those who had charge of his clothes, were compelled to report themselves to the proper authority. The owners of houses inhabited by phthisical patients were not allowed to eject them, because such action might render them homeless and wanderers, and facilitate the spread of the disease. The heirs of consumptives were forbidden to sell anything that had been used in their illness for a month after their death. Patients were enjoined to expectorate only in special vessels of glass or glazed earthenware, which were to be emptied and cleaned frequently. These laws were strictly enforced for thirty-nine years—until 1783—when, on account of a want of support on the part of the medical profession, they were repealed by the Grand Duke, Pietro Leopoldo, as being "a cause of bitterness, dissatisfaction and vexation."

* For the substance of this account the author is indebted to "A Medico-Literary Causerie," published in the *Practitioner*, July, 1898, p. 54.

In 1767 the State of Lucca passed similar laws, in which the rules for cleansing and disinfection that had to be adopted in cases of death from phthisis were set forth in minutest detail.

In 1772 the Guardians of the Public Health of Pesaro asked the corresponding officials at Venice for advice concerning the disposal of the clothes of persons who had died of phthisis. The latter referred this question to their Protomedico, Giambattista Paitoni, and he replied in a monograph in which he held that in the course of a long experience he had seen many evil effects from the neglect of necessary precautions "to protect one against a disease of such a nature." He recommended that all things used by consumptives should be dealt with in the same manner as in the case of "contagious pestilential influences." He laid special stress on the importance of taking care that the clothes of a consumptive should not fall into the hands of "filthy and miserly second-hand dealers, who then sell them again with impunity, thus trafficking in the health of men."

On the strength of this opinion the Venetian government issued an ordinance, December 24, 1772, to the effect that no one in any part of Venetian territory should "under any pretext whatever sell, or in any way part with or dispose of, receive or purchase clothes or other effects, which had been used by persons suffering from phthisis, unless they had first been properly disinfected. Breach of this regulation was punishable by death, imprisonment, or the galleys. Medical practitioners were compelled to notify all cases of death from phthisis so that the sanitary officer might give the necessary orders 'with that charitable forethought which considers that common health and safety.' For greater security secret notifications were received, and those giving information, authenticated with their names, to the authorities were rewarded."

On August 19, 1772, the Sacra Consulta of Rome issued a

circular to all the Papal States urging them to exercise the most vigilant care to prevent the sale of clothing belonging to persons who had died of that "pernicious communicable disease" (phthisis). "Medical practitioners were enjoined to notify all deaths from this cause, and to draw up an inventory of the things which had been used by the deceased. It was expressly directed that for this inventory no charge should be made, and it was further provided that if it was found advisable to burn any part of the belongings of the dead, and a poor family was thus deprived of things needful for domestic purposes, limited compensation should be made."

"At Bologna, the second city of the Papal States, an ordinance was issued in 1773 in which a further provision was made to that of the Sacra Consulta of Rome. By this the introduction of clothes, linen or other things used by consumptive persons into the city or its suburbs from any other region, without an official certificate of disinfection, was strictly forbidden. Not only physicians and surgeons but parish priests were bound to notify cases of death from phthisis, in order that the authorities might see that disinfection was carried out. Three years later the scope of the ordinance was extended so as to make the notification of illness compulsory as well as those of death. Secret notifications were received, and half the fine imposed for breach of ordinance was assigned to the informer. The physicians of Bologna appear to have treated the ordinance with scant respect, and the Cardinal Legate accordingly threatened to proceed against them. They, therefore, selected two of their body to draw up a memorial setting forth the grounds of their disbelief in the contagiousness of phthisis. . . . The authorities, however, stood firm."

In 1782 most stringent laws for disinfecting the belongings of the phthisical were introduced into the kingdom of Naples by Ferdinand IV., and which were maintained for more than

fifty years. The penalties for non-observance of the regulations were extremely severe. For interfering with the sanitary officers in the discharge of their duty "ignoble" persons were punished with three years of the galleys or prison; "nobles" by three years' confinement in a fortress and a fine of 300 ducats. Physicians received a similar fine for the first offense and ten years' banishment for the second. "Purchasers of infected clothing were punished by three years of the galleys, while those who sold them were fined three times the value of the articles sold." In every case the ceilings, walls, floors, doors and windows of rooms in which consumptives died were torn out and burned, and new ones were substituted. The bedding and furniture shared the same fate, and such dwellings were not inhabitable for one year. If owners rented such houses before the expiration of the prescribed time they were imprisoned for three years and the tenants were exiled. All the phthisical patients were forced to enter the hospital for incurable in Naples, and were detained there until they were either cured or dead. The family with phthisis in its midst was shunned and driven to want, and houses in which consumptives died came into disrepute and many of their owners were turned into beggars.

Laws of a similar character were introduced and enforced in certain parts of Spain and Portugal.

That which is of the greatest interest to us here is as to the practical benefit which followed the introduction of these draconic measures. Brehmer* states " concerning a diminution in the death-rate from pulmonary consumption in Naples and Portugal the medical historians of that period are ignorant." According to Uffelmann,† Dr. de Renzi, the historian of Italian medicine, states that the injury which had been inflicted on Naples by these laws was simply indescribable, and he denounces the Neapolitan medical faculty in the severest

* "Die Ætiologie der Chronischen Lungenschwindsucht," p. 495.
† *Berlin. k. Wochenschrift*, 1883, p. 369.

terms for participating in their practical introduction. Among other things Dr. A. L. Pierson* wrote of a Neapolitan hospital, in 1834, as follows: "One can hardly realize that so much has been said and written to recommend this city as a residence for consumptives, when some of the best informed Neapolitan physicians estimate the deaths from consumption among the residents at one-fourth of the whole mortality." One of the most reliable medical publications† in the English language states that Drs. Spattuzzi and Somma have paid great attention to the mortuary returns in the City of Naples (about 1866), and affirm that one-sixth of the whole mortality is due to phthisis; and Dr. de Renzi marvels greatly, in 1863, that the City of Naples is fully as much liable to this disease as either London or Paris, though the salutary condition of the climate should render it far less common.

It seems, therefore, if the death-rate from consumption was the same in Naples at the time these laws were abolished as it was in other cities in which segregration was never practised, that the practical value of such measures was entirely negative.

* *Practitioner*, July, 1898.

† *British and Foreign Medico-Chirurgical Review*, vol. 45, p. 112.

CHAPTER X.

ETIOLOGY CONTINUED: PREDISPOSING INFLUENCES.

Predisposing Influences. Under this head those influences will be considered which have the power of impairing the general nutrition and resistance of the constitution and directly or indirectly of the nervous system, and thus create a tendency to the development of pulmonary consumption. These factors will be discussed in the following order: Sex, age, stature, social condition, race or nationality, type of breathing, heredity order of birth, parental resemblance, occupation, dampness, air, confinement, poverty, prolonged grief, physical and mental excesses, civilization, pregnancy, Pott's disease, rickets and cardiac diseases.

The Influence of Sex. On the question of the relative frequency of phthisis in the sexes there is a division of opinion. Pollock, Bennett, Williams and others claim that it prevails more largely among the male sex; Louis, Laennec, Walshe, Hutchinson and James believe that it preponderates among females, while Niemeyer, Ruehle and others consider that one sex is as liable to the disease as the other.

One reason for the belief that more females than males die from consumption rests probably on the ground that she is the weaker of the two, is confined more within doors, leads a more sedentary life, is more deprived of sunlight, undergoes the enervating processes of gestation and lactation, and is, therefore, generally regarded as being more exposed to the causes which are supposed to give rise to the disease.

The author's own view is that more males than female die of phthisis. This opinion is based on a large mass of statis-

tics bearir.g on this question which he collected from the health records of the principal cities in Europe and America, about ten years ago, and found that the evidence pointed largely to this conclusion.

Influence of Age. The question of age—liability has already been considered in connection with the greater age-prevalence of insanity (see p. 174).

Influence of Stature, Weight and Chest Measurement. There is a certain relationship between the height, weight and chest measurement of healthy individuals which has been reduced to mathematical precision. Thus an adult male weighing 140 pounds should be five feet five inches high, and have a chest measurement of 36.83 inches. For a maximum chest measurement, two-thirds of the stature equals the circumference of the chest. For a minimum chest measurement one-half of the stature, minus one sixty-first of the stature, equals the circumference of the chest.

Tall persons have long, narrow chests, a tendency to a reduction in lung activity, and a sparseness in flesh. Such persons, especially when they follow sedentary occupations, are predisposed to pulmonary consumption. Children who grow vary rapidly duringand immediately after the age of puberty frequently present a disproportion between height, weight and chest capacity, and are similarly predisposed.

Influence of Race and Nationality. It is well known that of all the races in this country the Africans and the Indians are the most liable to pulmonary consumption, and, according to the author's investigation, made several years ago, the pure Indian is more susceptible to it than the half-breed or the mixed-blood. Of the different nationalities in this country the Irish are most liable, and the English and Germans most exempt from this disease.

Influence of Change of Climate and of Social Customs. In discussing these influences it must be borne in mind that, according to Spencer, constant friction exists be-

tween life and its environment, and that the former is most favorably situated where this antagonism is the least. Vegetation flourishes most profusely in the tropics, where the unfriendly action of the climate is reduced to a minimum, but it becomes scant in the extreme north or in high mountain altitudes, where the low temperature suppresses it almost completely. So with animal life in its primitive state it is encouraged by warmth and discouraged by cold; hence our earliest civilization sprang up near the equator, while life is scarcely tolerable in the arctic regions.

In spite of this natural opposition there has, through a long series of actions and reactions, gradually arisen an adaptation or reconciliation betwen man and the influences which surround him; and from this it follows that this adjustment is most firmly established in that environment in which man and his ancestors have become accustomed to live, or, in other words, if everything else is the same, he attains the best health and longest life where this harmony has been established the longest.

Many illustrations can be given in confirmation of this deduction. Thus Darwin* states that European dogs do not thrive in India. Most animals from any climate will survive only a few years in the zoological gardens of our cities, and the disease of which they generally die is pulmonary consumption. It does not seem to make any difference whether the migration takes place from warm to cold climates or the reverse, for the results are apparently the same. Negroes exported to Ceylon, Egypt or the West Indies die more frequently of phthisis than they do in their native climate. Ancell† declares that the natives of India who serve as troops in Ceylon and China, and who are very little liable to consumption in their own country, are much more prone to this disease than they are at home, and that the Laplanders, who

* "Domestication of Animals and Plants," vol. ii, p. 368.

† "Tuberculosis," p. 528.

at home are exempt from consumption and scrofula, are said to become liable to both on being transported to Denmark. Statistics show also that the liability to consumption among the English troops is about twice as great in the West Indies as it is at home. Dr. Forbes* states that in the high table-lands of Peru there lives a tribe of Indians, which is closely allied to the Quichua, whose chests are developed to extreme sizes, and that they are so thoroughly acclimated to their cold and elevated abodes "that when formerly carried down by the Spaniards to the low eastern plains, and when now tempted down by high wages to the gold-washings, they suffer a frightful mortality."

This section cannot be concluded more appropriately than by the following quotation from Ancell,† who says on this point: "Statistics lead to one general conclusion, *viz.*, that as respects the etiology, the habits and customs of mankind ride over all climatic influences; that if the Europeans inhabitants of Calcutta or Alexandria were to adopt the modes of life pursued in the metropolis of the British Empire (London) tuberculosis would, after a period, be as frequent in those localities as it is here, and conversely if the inhabitants of the densely populated countries of Europe were to improve their habits and customs as to labor and rest, indoor and outdoor occupation, the size, construction, modes of ventilation and sites of their houses, streets and towns, with other circumstances of hygiene, after a sufficient period had elapsed for hereditary influence to wear itself out, that they would probably be more free from this disease than the inhabitants of Lapland, Canada, Greenland, India, Egypt or Ceylon."

Influence of Occupation. The subject of dust-inhaling occupations has already been considered. A sedentary occupation like that of a clerk, typewriter, stenographer, telegraph operator, shoemaker, tailor, cigarmaker, etc., tends

* "Darwin's Descent of Man," p. 35.

† *Op. cit.*, p. 542.

to predispose to phthisis by producing stoop-shouldered and round-chested people, and by interfering with the full and normal expansion of the lungs. Lombard found that in Paris, Geneva, Vienna and Hamburg nearly 50 per cent. more persons die of phthisis who lead a sedentary than those who follow an active life. The occupation of firemen is exceedingly unhealthful. In an investigation of this subject made by the author about eight years ago it was found that the death-rate from phthisis among the firemen of the leading cities in this country is about 17 per cent. higher than it is among the general adult population. This high death-rate is, in all probability, due to the enervating and depressing physical influence, the deprivation of rest and sleep and other exposures incidental to this particular kind of life.

Influence of Exercise. There is no doubt that bodily exercise possesses a marked influence in determining the degree of lung expansion. Darwin* says that the lungs in improved breeds of cattle, which naturally take little exercise and are domiciled much of the time "are found to be considerably reduced in size when compared with those possessed by animals having perfect liberty," and Waldenburg states that the vital lung capacity is smallest in persons who lead sedentary lives, such as professional men, students, clerks, etc., and is greatest in those who follow active outdoor occupations, such as sailors, recruits, etc. Chassagne and Dally, in their joint work on the "Influence of Gymnastics on the Development of Man," report that at the Military School of Gymnastics out of 401 individuals subjected to gymnastic exercises for five months 307, or 76 per cent., showed an increase of an average of 2.5 centimetres in the mammary circumference of the thorax. According to Dr. Abel, 75 per cent. of those who practise gymnastics in Germany experience an increase in the measurements of the chest. It may be taken for granted, therefore, that the ex-

* "Domestication of Animals and Plants, etc.," vol. ii, p. 361.

change of a life of physical activity for one of ease and indolence will not only tend to diminish respiratory capacity but will also be conducive to pulmonary consumption.

Influence of Dampness. That dampness predisposes to pulmonary consumption under certain conditions has been well established by numerous observers, notably among whom are Bowditch and Pepper of our own country, and Buchanan, of England; yet there is ample evidence on the other hand to prove that moisture, either in the soil or in the atmosphere, does not universally lead to the production of this disease. This opinion is warranted by the facts that fishermen are more exempt from consumption than many inland inhabitants; that the death-rate from this disease is very much smaller in the navies than in the armies of the world; that Iceland, the Hebrides, the Shetlands and the Faroe Islands are almost exempt from this malady, in spite of being under the influence of the continuous moisture arising from the Gulf stream. Evidence is not wanting to show also that if the inhabitants of a moist climate are transferred to one that is dry their liability to consumption increases.

Dampness of itself is, therefore, not capable of producing consumption unless it operates on an unacclimated constitution. For this reason dampness has justly come to be regarded as very dangerous to health. Many chronic pulmonary diseases are set up in people who move into houses with freshly-plastered walls, or inhabit dwellings the cellars and surroundings of which are scarcely ever free from water or dampness, and this frequently explains why, under certain circumstances, consumption is more rife in moist than in dry localities.

Influence of Air. Fresh air is considered the *sine quâ non* in the cure of consumption, and impure air one of its principal causes. Innumerable plans and methods have been devised for improving the ventilation of our dwellings, hospitals and workshops; volumes have been written on the ill

effects of breathing vitiated air, and the immaculate purity of country and mountain air has come to be universally regarded as a certain guarantee against the ravages of pulmonary diseases. These, like many other current notions, contain a germ of truth, but actually are delusive and dangerous, inasmuch as they exaggerate the effects of a small evil and afford a false sense of security against the real source of danger in the production of pulmonary consumption.

One argument which lends color to the belief that pure air affords immunity from phthisis is that those who occupy elevated or mountainous regions are less liable to this disease than those who live near the sea level. This is in all probability true, but while not in the least underrating the value of wholesome air in the prevention and treatment of this disease the author is convinced that pure and impure air play but a very unimportant part either as causative, curative or preventive agents. According to Dr. Schnyder* the mortality from consumption is greater in the cantons of Switzerland than it is the cities of that republic. This is also true of the phthisical death-rate between the agricultural districts and the cities of Prussia. Probably the most accurate and extended statistics on this question in this country are those published by the State Board of Health of Rhode Island. These figures show that the death-rate from pulmonary consumption during twenty-five years—from 1860 to 1884—was higher in the city of Providence than in Bristol and Newport counties, but lower than in Kent, Providence and Washington counties, and somewhat under the average death-rate of this disease throughout the whole State.

If it were true that phthisis is the result of breathing vitiated air how can we account for the fact that the inhabitants of Iceland, Greenland, Lapland and of other cold countries of the north, who live in dwellings which are notoriously want-

* *Correspondenz-Blatt f. Schweizer Aertzte*, 1886, Nos. 10, 11, and 12.

ing in ventilation, are practically exempt from this disease? Of the Icelanders Mr. Warnford Lock,* who is very familiar with these people, says that their life is "one long exposure to the elements, and during the night they live in dwellings devoid of ventilation and which, if not buried beneath the earth, are built of turf and often become grass-grown, a very bad feature being the excessive stuffiness of the common living and sleeping rooms, when, owing to the absence of fires, the greatest possible crowding and plugging are necessary in order to maintain a tolerable degree of warmth." And yet Dr. Cullimore† says "that consumption in Iceland is never indigenous, but is always, when it does occur, imported from abroad and but seldom extends to the second native generation." Again, miners and laborers who are employed in coal-mines, and who continually respire a damp and musty atmosphere loaded with impurities, are, as has already been shown, comparatively free from the disease under consideration.

Now, when these observations are considered, with the well known fact that the people of the tropical regions of the globe, who enjoy an uninterrupted revelling in pure, fresh air, both day and night, summer and winter, are by no means spared by phthisis, it is quite plain that pure and impure air do not play such a very significant part in the evolution of this disease.

Influence of Type of Breathing. Since the time of Boerhaave it is known that a fundamental difference exists between male and female breathing, that the former respires chiefly with the lower portion of the chest—which is called the diaphragmatic or abdominal type of breathing—and that the latter breathes principally with the upper portion of the chest—which is called the costal type of breathing. These

* "The Home of the Eddas," S. Low, 1879.

† "Consumption as a Contagious Disease," p. 73. Ballière, Tindal & Cox, 1880.

types of respiration may be graphically illustrated on a registering cylinder by means of a pneumograph, as is shown in the following tracings: Fig. 4 shows a typical tracing of a male, and Fig. 5 that of a female. The first half of each tracing represents the abdominal, and the second half the costal movements. It will be observed that the contrast between

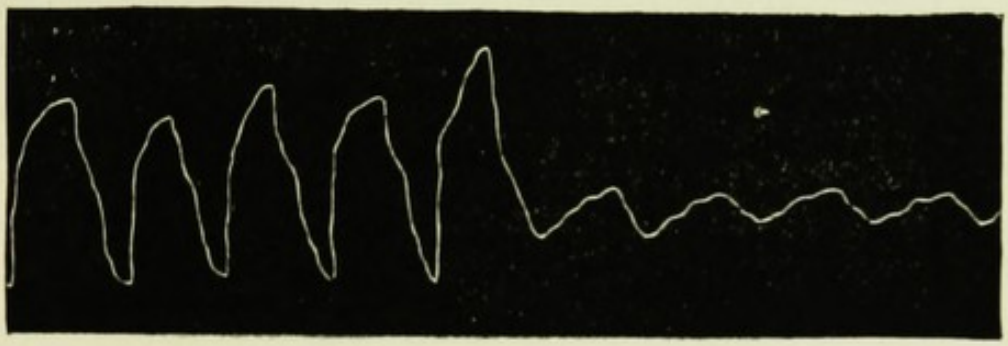

Fig. 4.—Respiratory movement taken from chest of civilized male.

a typical male and female respiration is well brought out, the curves showing the greatest amplitude in the abdominal and the least in the costal movements of the male, while in those of the female this order is exactly reversed.

About twelve years ago the author examined the chest-

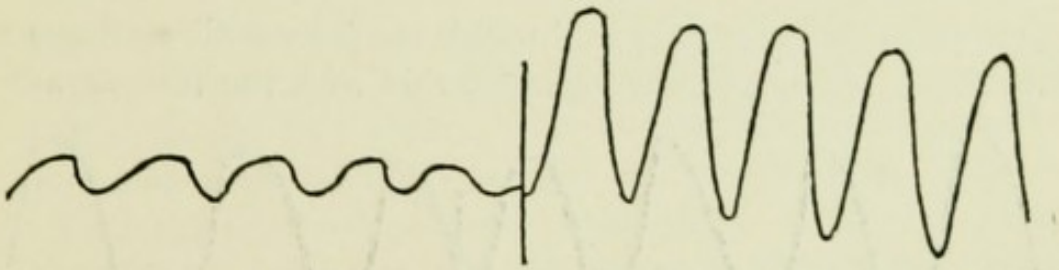

Fig. 5.—Respiratory movement taken from chest of civilized female.

movements of eighty-two Indian females in the Lincoln Institution of Philadelphia—a school for Indian girls—and found that practically they all possessed the abdominal type of breathing, as shown in Fig. 6.

That both types of breathing may be modified is shown by the following tracings. Fig. 7 is taken from the chest

of a civilized female who had never worn a corset, and Fig. 8 is taken from the chest of a civilized male, with tight belt around abdomen.

From these observations it is quite manifest that the primi-

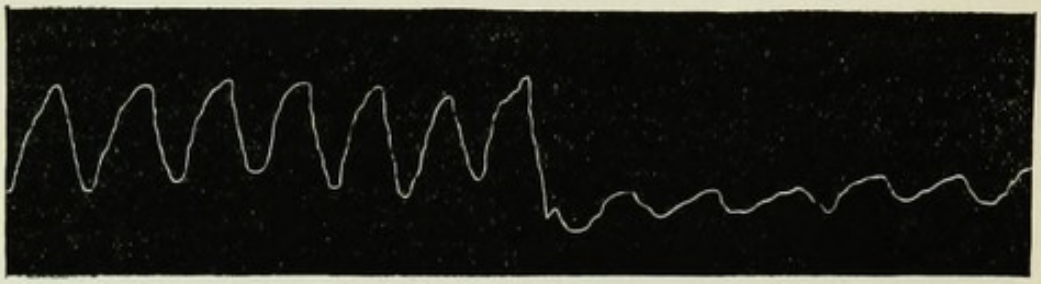

Fig. 6.—Respiratory movement. Taken from chest of an Indian female.

tive type of respiration in both male and female is abdominal, and that the costal type of the civilized female differenti-

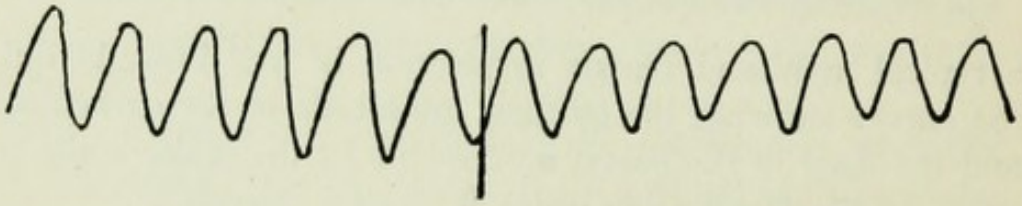

Fig. 7.—Respiratory movement. Taken from chest of a civilized female who had never worn a corset.

ated from the original type through the constrictive influence of dress. Now when we connect this with the fact already

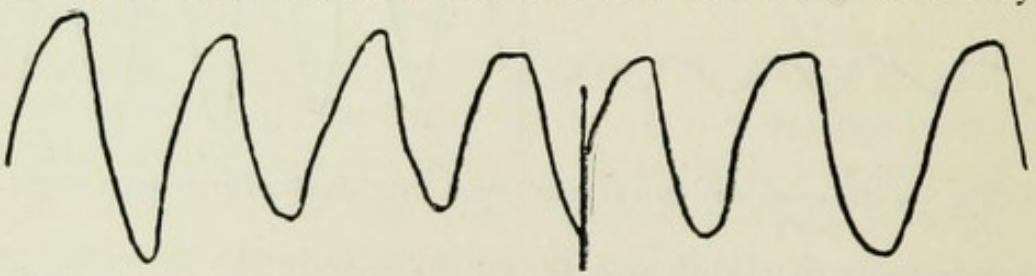

Fig. 8.—Respiratory movement. Taken from chest of civilized male with tight belt around abdomen.

shown that in the aggregate more civilized males than females die of consumption, and that the latter disease as a rule begins in the apices of the lungs, are we not justified in believing

perhaps that defective costal respiration and the beginning of pulmonary consumption bear some intimate relationship to one another, and that the female partly owes her greater freedom from this disease because she is endowed with fuller apical expansion?

Influence of Heredity. Pulmonary consumption is not only transmitted from generation to generation, but evidence has already been adduced to show that this disease and many nervous disorders are transmutable conditions, and that the former is frequently preceded by insanity, idiocy, hysteria, epilepsy, etc., in the parentage. In this connection it is well to make it plain that heredity does not mean the transmission of disease, but merely the imparting of a certain abnormal tendency by the parent to the offspring, which produces in the latter a greater susceptibility to the disease than that which exists in those who are born of different stock. It is probably true that every individual is burdened with some diseased heredity, and marches to the grave in a direction which is dictated by this condition. Some suffer from a rheumatic diathesis, some from an insane tendency, some from a consumptive trend, others from a cancerous burden, and so on, showing that heredity is a widespread factor in the propagation of disease.

Heredity implies the existence of two factors: (1) An influence, and (2) a medium upon which the influence operates and impresses itself more or less permanently. Now of all the organic tissues the nervous system is the one which is eminently endowed with the property of receiving, transmitting and recording impressions. This marked sensitiveness of the nervous system not only explains why so many diseases which affect it assume a chronic form, but also why such affections are more readily transmitted than those which involve other textures. The hereditary nature of pulmonary consumption is, therefore, another strong proof that it has a nervous and not a pulmonary origin.

Heredity in phthisis may be discussed under two heads: (1) Direct, when it exists as such in the family; (2) indirect, when it is preceded in the family by insanity, idiocy, epilepsy, hysteria, or some other form of nerve degeneration.

Direct Heredity. In estimating the potency of direct heredity it must be taken into account that statisticians differ in their conclusions, because they include various degrees of relationship. When parental influence alone is considered the percentage of cases is, of course, lower than if that of the whole family is embraced in the problem. "The 1,010* cases of the Brompton Hospital reports included only parents, and gave an average of 24.4 per cent. Dr. Fuller's 385 cases embraced grandparents, uncles and aunts, and furnished 59 per cent. Dr. Cotton's 1,000 cases included parents, brothers and sisters, giving 36.7 per cent., and Dr. Pollock's 1,200 cases, similarly estimated, showed 30 per cent., while the 1,000 cases collected by the author show a percentage of 48.4 on the same basis." Homan estimates it at 70 per cent., Russ at 83 per cent., while Dr. Schnyder† investigated the whole family influence in 3,461 cases of consumption and found it present in 66.03 per cent. The mortuary experience of *The Mutual Life Insurance Company of New York* shows that "among an equal number of consumptives and of non-consumptives who were insured in that company, nearly twice as many of the former had consumptive blood relatives as those of the latter."

Professor Langerhans‡ investigated the death-rate of the offspring of healthy colonial parents on the island of Madeira, and also that of consumptive parents who came on the island for the purpose of restoring their health. During thirty-six years there were born of healthy parents 147 chil-

* Williams, "Pulmonary Consumption," p. 62.

† *Loc. cit.*

‡ "Zur Aetiologieder Phthisie," *Virchow's Archiv*, 1884, Bd. 97, S. 289.

dren; one of these died of consumption, but he was strongly addicted to alcoholic excess and contracted the disease while absent in England. During the same period there were born of consumptive parents 106 children, 8 of whom died, and 8 are suffering from the disease, making 16 in all who became phthisical before 1883.

INDIRECT HEREDITY. In considering the relation between insanity and phthisis citations were given from Van der Kolk, Maudsley, Clouston, Blandford, Mosher and others in illustration of the fact that insanity in the parent is frequently transmitted as pulmonary consumption to the offspring; the same transmutation has been shown to occur in the case of idiocy, in that of hysteria and in that of epilepsy, and it will now be shown that nerve deterioration brought about by other influences will likewise generate a proneness to terminate in phthisis.

There is no question that *alcoholism* in the parent is frequently a forerunner of phthisis in the child. Let those who have any doubts on this point cast a retrospect over the family histories of consumptive cases in which there is an absence of a family taint of the disease and it will frequently be found that either an immediate or a remote blood relative was addicted to alcoholic excess. The following cases illustrate this: Male, aged 42, began the excessive use of spirits after the death of his wife. He became a steady drinker and was practically intoxicated all the time. His mother and two sisters died of consumption, and his grandfather used spirits to excess for years. One paternal uncle died from excess of drink and another of consumption. Another paternal uncle died from phthisis after many years of alcoholic excess. His grandfather on his mother's side drank more or less all his life and died from some rheumatic trouble (*Crothers*). M., aged 48, is a dipsomaniac. His mother and two maternal aunts died of consumption. His maternal grandfather drank to excess (*Crothers*). Female adult, is suffering from pul-

monary phthisis, and she had a brother and sister who died of chest disease. Her father and a number of her relatives are alcoholics (*Grasset*). Male, aged 44, is subject to periodical hemoptysis, but abstains from all alcoholic drinks. His father is living at 68, but he is an alcoholic. His grandfather died of alcoholism, and a paternal uncle of consumption. No phthisis in the family prior to this death.

Male, came to this country in 1798. He was a harness-maker, a beer-drinker, and after middle life drank rum to excess until he died, at the age of 61. His wife was a healthy woman, and lived to be 80 years old. Eight sons grew to manhood and married. Six of them died of consumption, under 45 years of age; one was killed by accident and one died from the excessive use of spirits. Two daughters grew up and married; one died of consumption and the other in childbirth. They left four children; two were inebriates, and the two others were eccentric and died of consumption. Of the children of the eight sons only ten grew up to manhood; four of these drank to excess and died, three of the six remaining died of consumption, and two others were nervous invalids until death, in middle life; the last one, a physician of eminence, has become an inebriate and is the only surviving member of all this family. The male members of this family were farmers, tradesmen and men of more than average vigor in appearance. They married women (so far as can be ascertained) without any special hereditary history of consumption or inebriety (*Quarterly Journal of Inebriety*, October, 1888, p. 390).

This, then, being the relation between alcoholism and phthisis, it is very readily understood why these two diseases should so frequently change places in different members, or in generations of the same family, and why they are so often associatel with various other nervous disorders. Moreover, on account of the past and present abuse of alcohol throughout the civilized world, there is very little ques-

tion that it ranks as a tremendous factor in maintaining the ranks of the thousands who are annually slain by pulmonary consumption.

Eminent authorities have stated that they regard pulmonary consumption and scrofula as refined inheritances of *syphilis*. That there is a germ of truth in this proposition cannot be doubted by any one who has seen much phthisis among the lower classes, although its application is not as sweeping as it is supposed to be. That pulmonary consumption may be engendered by syphilis is demonstrated by the remarkable experience of a negro settlement on three plantations in the southern part of Georgia, as cited by Dr. Powell (*op. cit.*). Prior to the war these negroes enjoyed remarkable mental and physical health. Consumption, insanity and similar wasting diseases were altogether unknown among them. At the time of their emancipation there were upon these three plantations, including children, between 400 and 500 as healthy individuals as could be found, and free from all hereditary taint or tendency to any disease whatever. The negroes, as a rule, remained on the plantations where they were born. It was not long, however, before syphilis appeared among them, and it gradually spread over these plantations, the disease in nearly all cases going into the tertiary stage. Some ten or fifteen years later consumption and insanity began among the adults, and many of their children died from scrofula and phthisis, and it was the exception rather than the rule that the children lived. Bodily deformities and idiocy were frequent among them.

That which is true of alcoholism and syphilis is also true concerning *chronic mercurialism*. Scrofula and pulmonary consumption are its common inheritances. In the discussion of the relationship between mercury poisoning and phthisis this predisposition is fully brought out, and in that connection a remarkable instance is related by Bäumler in which

consumption in the children was clearly engendered by mercurial intoxication in the parents.

Influence of the Order of Birth. In this section an effort will be made to show that the order of birth and the size of families are determining factors in the predisposition to pulmonary consumption. Birth is a form of nutrition or growth, but, according to Spencer, there is a direct antagonism between birth and growth. He says*: "Whether a deduction is made from one parent or two. . . . it remains, in any case, a deduction, and in proportion as it is great or frequent, or both, it must restrain the increase of the individual."

From these principles it follows that reproduction is most perfect when growth is completed and before bodily decay sets in; that the higher the rate of nutrition during the reproductive period the higher the rate of reproduction, and that if the bodily expenditure is greater for other purposes than for reproduction there is a corresponding deduction from the resources which would otherwise be devoted to the maintenance of the latter process. In other words, offspring will be, if other things are equal, most vigorous when the growth of both parents is complete and the state of nutrition is good; and least vigorous and most prone to decadence when the state of nutrition in the parents is low, when their expenditures for other purposes is great, or when reproduction takes place rapidly or follows at short intervals, or takes place before the body is fully developed, or during the period of bodily decline. All other things being equal, then, the last or the youngest children, as well as those who are born before the bodies of the parents are fully developed, should be the weakest, and those born during the remainder or the middle part of the reproductive period should be the strongest.

Let us see whether and to what extent these principles are confirmed by actual experience. Dr. Brehmer in his great

* "Principles of Biology," vol. ii, p. 428.

work* records the histories of four hundred cases of phthisis, in which are given the age of each patient, the number of patient's sisters and brothers, the order of each patient's birth, the number of paternal and maternal uncles and aunts, and in many instances the order of birth of father and mother. An analysis of these cases leads to the following conclusions:

1. That as a whole the children of large families are less vigorous than those of small families.

2. That not all the children of a family are equally resistant or predisposed to consumption.

3. That the youngest members of a large family, provided their parents are healthy and come from small families, are most liable to pulmonary consumption. This is illustrated by the first one hundred, and by the first fifty of the third one hundred cases.

4. That both the youngest and the oldest members of a small family are more susceptible to the disease than the intermediate members, even though the parents and grandparents were healthy and became aged, provided that either or both parents come from the youngest members of large families. This is illustrated by the second one hundred, and the second fifty of the third one hundred cases.

5. That children born of parents having phthisical antecedents are subject to the same law of liability as those children whose parents are healthy but come from among the youngest of large families. It is not necessary, however, for one or both parents to come from the youngest of their families. This is shown by the fourth one hundred cases.

6. That those children who are born within a year after the birth of the preceding members are more liable to pulmonary consumption than those who are born two or three years apart.

* "Die Aetiologie der Chronischen Lungenschwindsucht."

7. That children born of phthisical parents are liable to pulmonary consumption three years earlier, on the average, than those born of healthy parents. This is shown by contrasting the average ages of the first one hundred cases with those of the fourth hundred cases.

8. That parents with a family history of consumption are less prolific by nearly one-half than those who are healthy. This is shown by comparing the average number of children in the patients' families of the first one hundred with those of the fourth one hundred cases.

Influence of Parental Resemblance.—This demands the serious consideration of every physician. Parental resemblance implies a transmission of the parent's constitution, appearance, action, weight, temperament, etc., to the child, and is a guarantee, if other things are equal, that the life of the parent will be duplicated by the child; or, in other words, that the child will move through the world in the same general direction as that which was pursued by the parent. Hence, the child which bears the likeness of a consumptive parent should be considered much more liable to contract this disease, if other things are the same, than if it resembled the well parent. This obtains especially in the case of the mother, for, like in insanity, she transmits consumption more readily than the father.

Influence of Poverty. Poverty and its concomitants are also predisposing conditions to pulmonary consumption. People who live from hand to mouth, who are overworked and underfed at the same time, housed in over-crowded and insanitary quarters, frequently liable to alcoholic and other excesses, many a time indifferent to what seems a trivial cold and cough, cared for improperly during convalescence from disease or injury, and often compelled to go to work before they are fit, make an array of devitalizing circumstances which induce consumption and of which those who have means and affluence know nothing about.

That consumption is more common among the poorer than it is among the richer classes has been brought out very prominently by an investigation which the author made some years ago concerning the relative prevalence of this disease in the different wards of the city of Philadelphia. He found that of all the wards in this city the mortality from consumption in the Seventh stands in bold relief when contrasted with that of the Eighth, which bounds it on the north, being about 25 per cent. greater, proportionally, in the former than in the latter. Here are two wards of about equal size, lying parallel to one another, with similar soil, subsoil and climatic environment, differing in no respect except that in the Seventh the population-density is just about again as great as it is in the Eighth; that the former, besides containing a comparatively large heterogeneous foreign population, also comprises the largest colored population of any ward in this city, while the latter is practically without either of these classes, and that the people of the former live mostly in small, rented and sub-rented houses while those of the latter live chiefly in large and luxurious homes, being, on the whole, a combination of influences which act as factors in the production and abatement of this disease.

Influence of Prolonged Grief, Etc. Fright, violent and painful emotions and disappointment in love have been recognized by many authorities as potent factors in the production of pulmonary consumption. Dr. O. Kohts* observed that fright markedly aggravated all diseases of the respiratory apparatus during the siege of Strassburg, and hemoptysis often occurred for the first time among the phthisical. He relates the interesting case of a woman who, without being particularly sick, suffered from periodical cough, to which, after sudden fright, hoarseness, pain in the larynx and aphonia were added. Laryngeal pain and hoarseness

* *Centralblatt f. d. med. Wis.*, 1873, S. 826.

developed later when excited, but the laryngoscope showed neither ulceration nor swelling of the vocal cords.

Laennec* states that among the occasional causes of phthisis he knew of none of more assured operation than the depressing passions, particularly if strong and of long continuance, and on page 334 relates the following interesting experience: "I had under my own eyes, during a period of ten years, a striking example of the effect of the depressing passions in producing phthisis, in the case of a religious association of women, of recent foundation, and which never obtained from the ecclesiastical authorities any other than a provisional toleration on account of the extreme severity of its rules. The diet of these persons was certainly very austere, yet it was by no means beyond what nature could bear. But the ascetic spirit which regulated their minds was such as to give rise to consequences no less serious than surprising. Not only was the attention of these women habitually fixed on the most terrible truths of religion, but it was the constant practice to try them by every kind of contrariety and opposition in order to bring them, as soon as possible, to an entire renouncement of their own proper will. The consequences of this discipline were the same in all; after being one or two months in the establishment the catamenia became suppressed, and in the course of one or two months thereafter phthisis declared itself. As no vow was taken in this society, I endeavored to prevail upon the patients to leave the house as soon as the consumptive symptoms began to appear, and almost all those who followed my advice were cured, although some of them exhibited well-marked indications of the disease. During the ten years that I was physician of this association I witnessed its entire renovation two or three different times, owing to the successive loss of all its members, with

* "On the Chest," Forbes' Translation, p. 333.

the exception of a small number, consisting chiefly of the superior, the gate-keeper, and the sisters who had charge of the garden, kitchen and infirmary. It will be observed that these individuals were those who had the most constant distractions from their religious tasks, and that they also went out pretty often into the city, on business connected with the establishment. In like manner, in other situations, it has appeared to me that almost all those who became phthisical, without being constitutionally predisposed to the disease, might attribute the origin of their complaint to grief, either very deep or of long continuance." In a foot-note Dr. Forbes, the translator of this work, states that "the influence of the depressing passions in giving rise to diseases of the lungs, and particularly phthisis, has been noticed by many writers. It is well known that Morton has entitled one of his species of consumption *Phthisis a Melancholia*. In many parts of his "Phthisiologia" this author's opinion respecting the great effect of mental causes in producing this disease is strongly expressed. . . . In relation to this subject the observations of Avenbrugger respecting the effect of nostalgia in producing diseases of the chest are highly worthy of attention."

Ancell* states that "the writers who have assigned this cause have said little of the effect of the chronic emotions or passions on the corporeal frame beyond enumerating them among the causes of one of the varieties of tuberculosis; but in order to estimate the influence of such a cause it is essential to bear in mind the corporeal effects of such affections of the mind—dejection, constant anxiety and sadness, distress, sorrow, grief, remorse, melancholy and despair.

"When the operation of the depressing passions is slow and long-continued among the most prominent effects we find the bloom of health disappearing, the face grow pale and emaciated, the adipose support of the eyeball gradually dimin-

* "Treatise on Tuberculosis," p. 466.

ishes and the eye becomes sunken, the fat generally is absorbed and the muscles become weak and relaxed. They appear to depress the vitality of the blood and to undermine the source of all the vital energies, without any previous increase of action, which may be sustained for many years, ultimately proving fatal and the patient dying exhausted."

Influence of Pott's Disease. Angular curvature of the spine, or Pott's disease, is a tubercular affection and on the score of infectiousness this disease should frequently be associated with pulmonary consumption, either as a cause or as a concomitant. The author's experience indicates, however, that this complication is exceptionally rare, for he has neither record nor recollection of more than two cases of this kind. This seems to be confirmed by the observation of others. Dr. James* states that "malformation of the chest walls, the result of rickets, chicken-breast, spinal curvature, etc., are according to most observers, rarely found in phthisis." Flint, in work on "Phthisis,"† found only two instances of angular curvature of the spine among the 670 cases of phthisis which he collected and analyzed in this book. In both of these cases the spinal deformity antedated the phthisical condition and in one the latter affection became non-progressive and in the other it was entirely arrested. In Brehmer's‡ collection of 500 cases of phthisis not a single case of angular curvature of the spine is to be found.

Influence of Peritoneal Tuberculosis. Tuberculosis of the peritoneum has, from the time of Louis up to a recent period, been largely regarded as an almost necessarily fatal disease. At present it is not a rare experience to see a surgeon open an abdominal cavity and expose to view a roughened peritoneum that is "studded with myriads of miliary

* "Pulmonary Phthisis," p. 55.

† Page 166.

‡ "Die Aetiologie der Chronischen Lungenschwindsucht," von Dr. Hermann Brehmer, sen., Berlin, 1885.

tubercles," and all that it seems necessary for him to do to relieve this condition is to drain off the accumulated fluid, to dust the peritoneal surface with iodoform, or to apply some other antiseptic, and allow the wound to close up. Indeed some surgeons claim that mere drainage and flushing of the abdominal cavity with simple warm water will effect a cure in many cases. On the other hand, it does not appear that tuberculosis of the peritoneum is invariably followed by tuberculosis of the lungs, and in case such an event occurs relief of the abdominal affection often affords amelioration if not abatement of the pulmonary trouble.

INFLUENCE OF PRISON LIFE. It has long been observed that prison life laregly increases the liability to pulmonary consumption. Ancell* states that nearly half the deaths and half the pardons on medical grounds were due to tubercular diseases in the Millbank Penitentiary during a period of eighteen years, and that during one year the mortality from this disease in this prison was nearly four times greater than it was among the free population. Dr. Leach† collected the following data relating to the number of deaths which occur from pulmonary phthisis in various prisons throughout the United States:

Names of Prisons.	Period of Years.	Total Deaths.	Phthisis—Deaths.	Percentage of latter.
Moyamensing (Phila.).....	32	282	177	62.76
Maryland Penitentiary.....	20	187	72	38.50
Vermont "	2	6	5	83.33
Kentucky "	12	116	37	31.89
Penna. " (Eastern).	6	31	30	96.77
Connecticut Prison.....	30	117	107	91.44
New Jersey "	10	79	60	75.94
California "	5	53	19	35.84
Sing Sing " (N. Y.).....	20	339	113	33.33
Auburn " "	51	575	231	40.17
Clinton " "	1	6	2	33.33
				56.65[1]

[1] Average Percentage.

* "Treatise on Tuberculosis," p. 487.

† California Board of Health, 1874 and 1875, p. 56.

Furthermore, Dr. A. Baer,* in his investigation of the death-rate of criminals in the prisons of Germany, found that from 64 to 90 per cent. of them die from pulmonary phthisis.

Now, when this death-rate from consumption among criminals is compared with that of the general adult population, it will be seen that it is enormous. For the average mortality from pulmonary consumption among the latter, between the ages of 20 and 70, as founded on the statistics of a number of large American cities, is 27.29 per cent., showing the mortality from this disease is more than twice as great.

To what is this inordinate mortality from phthisis due? Is it caused by long-continued confinement? This is a conclusion to which every inquirer is naturally led, and it is quite probable that it may in part be accounted for on this score, but there seems to be sufficient evidence to show that other and more powerful causes have a share in bringing it about. In his paper† on the mortality from phthisis in Millbank prison, England, Dr. Baly states that "the number of new cases thus increased gradually during the first eighteen months; it then remained nearly stationary, or rather diminished." From this the inference may be drawn that it is not those who undergo the longest terms of imprisonment that become most vulnerable to phthisis. This is also in complete harmony with the testimony of Dr. R. Boyd concerning the influcnce of confinement in producing phthisis among the English insane. He says‡ of those insane who resided in Somerset County Asylum from one to two years 29.7 per cent. died of phthisis, those resident from two to five years 31 per cent. became phthisical, and of those resident five years and upwards 34.4 per cent. became phthisical. The in-

* "Ueber das Vorkommen von Phthisis in den Gefängnissen," 1884.

† See appendix, p. 336, in "Scrofula and Its Treatment," by Benjamin Phillips, Phila., 1846.

‡ *Journal of Mental Science*, vol. xv, p. 196.

creased liability is wholly confined, however, to the female residents, for among the males it is 3½ per cent. less in those who remained five years and upwards, than in those who remained only from one to two years.

Nor is it likely that infection plays any considerable part in the causation of this disease among those undergoing imprisonment. For when Dr. A. Baer shows, in the paper already quoted, that the death-rate from consumption in the prisons of Germany amounts to 64 and 90 per cent. he also points out that prisoners who suffer solitary confinement have a death-rate of 20 per cent. higher than those who are allowed to associate with each other outside the cells. It is hardly reasonable to suppose that the cells of the solitary prisoners are more virulently infected than those in which the prisoners dwell who suffer congregrate confinement, who commingle with each other and who are, therefore, exposed to the disease if it exists, as it generally does, among their fellow prisoners.

It is quite likely that food exerts an important part in the genesis of consumption under these circumstances. For Cless* states that the death-rate from consumption in the prison of Würtemburg, from 1850 to 1859, was 24 per 1,000 while from 1859 to 1876, a better prison diet being introduced during that time, the same fell to 8 per 1,000. There was no change in anything except in the matter of food.

In looking for a cause of this excessive death-rate, does it not seem probable that imprisonment in itself has a powerfully depressing and devitalizing influence on the mental constitution of men whose brain and nervous system are already in a degenerate state; that these devitalizing effects are more pronounced in the case of solitary than in that of congregate confinement; that poor diet and the lack of variety of the same, and that poor nutrition, the absence of sunlight and

* *Vierteljahrschrift f. öffentliche Gesundheitspflege*, Bd. 11, p. 396.

exercise and the accumulated effects of vice are largely instrumental in originating this disease among these men?

INFLUENCE OF CIVILIZATION. Every fact seems to point out that pulmonary consumption is a disease which accompanies civilization, and is absent from the primitive conditions of human life. This is such a remarkable concurrence that it will be of great interest to inquire briefly into the conditions which separate the higher from the lower forms of social life. In doing this it may be said that there is no doubt that the progenitors of the present civilized race were at one time in a state of savagery, and that the gulf which differentiates the civilized man from the savage of to-day is no smaller than that which exists between the latter and the anthropoid ape. For during the period of man's development the process of adjustment between internal and external relations has wrought such a complete revolution in his existence that his habits and customs, his civil and his social and intellectual interests are diametrically opposed to those of his savage ancestry. When we come to look into the causes which have bronght about this difference between the civilized and the savage man it will be found that, physically, they consist principally of a change of occupation, of shelter and of clothing. The occupation of the savage consists of hunting and fishing while that of his civilized neighbor comprises the complicated industries and the varied agencies that have been evolved and accumulated during thirty centuries of intellectual development; the shelter of the savage is a rude hut, or the canopy of heaven, while the modern man dwells in comfortable houses and magnificent palaces, and the clothing of the former, regardless of climate, is hardly more than the bare skin, while the body of the latter is covered and embellished with the most elaborate dress, which cools him in summer and warms him in winter.

These deductions are confirmed by the following observations: The African negro is comparatively free from pulmo-

nary consumption in his native climate, and remained in this condition as an American slave until he was emancipated during the civil war. At this time his whole environment was reversed, and his habits and customs were completely changed. Instead of being a simple, unaccountable being, whose every want was constantly provided for, he became burdened with the cares and responsibilities of social manhood. The transition was so sudden and the demands of his new position became so vast and so exacting that he is now often unequal to the task of meeting them successfully, and in consequence of this his brain and nervous system, upon which the brunt of the contest falls, breaks down and he lapses into insanity and consumption—two disease to which, as we have seen, he is notoriously liable since the war.

That which is true of the negro is also true of the North American Indian. All the information that can be gathered on this point teaches that in his aboriginal and native state he was, and is now, free from pulmonary consumption, but that in his unequal warfare with modern civilization he falls an easy prey to this disease.

The natives of Van Dieman's Land furnish a good illustration of the effects of a sudden change of environment on the human constitution. These savages in their native condition were, so far as can be ascertained, almost entirely exempt from pulmonary consumption before the advent of the white race; but on account of their frequent depredations the white settlers confined them on an island in Bass's Straits and furnished them with food, clothing, houses and suitable superintendents. "But to a wandering race accustomed to rove at will through a large and extensive country, to procure their food as they pleased by hunting and fishing, and, regardless of dress, to live where and how they liked, this confinement to a narrow, little island, and immediate change of their own free and unfettered habits to the more constrained and artificial ones of civilized life, proved speedily

fatal. They died in great numbers, and the majority from pulmonary consumption."*

On the other hand, it is not sufficiently appreciated, though no less a potent fact, that civilization also plays a powerful rôle in promoting pulmonary consumption among civilized races, and this is the reason why civilized man is more vulnerable to this disease between the ages of twenty and thirty than he is at any other period of his life. This vulnerability is not due to any morphological or functional change in his lungs at this time, as is supposed by some, but because it is the period during which his brain and nervous system undergo the most varied and intense functional development (it being well established that during the development or maturity of an organ it is most liable to disease). It is the period in which he is compelled to face problems which are untried and difficult. He is removed from parental protection, and left to struggle for his own existence in the battle of life. He becomes burdened with family cares and duties. He is confronted by a social condition, with its education, knowledge and inventions, its diverse manners and customs, its changeable institutions, its rankling politics, its innumerable arts, sciences, and manufactures, its multiplicity of industries and employments, its burning life struggles and tragedies, and its accompanying proneness to vices, excesses and abuses of all sorts, which demand of him the exercise of the highest attainable skill and qualification to maintain his well being. He is like an animal on which are imposed new physical conditions incidental to a succession of geologic changes, and to which it must adjust itself in order to preserve its life. His environment is not just of the same character as that of the animal, but its power is just as real and as great. The strain of his battle falls principally on his brain and nervous system,

* "Climate of Van Dieman's Land," *Dublin Journal*, vol. xxiii, p. 88. Cited according to Ancell, *op. cit.*, p. 544.

for they are the instruments through which he adjusts himself to his new relationship and if from any acquired or inherited weakness these prove inadequate he will fall in the contest.

CHAPTER XI.

ETIOLOGY CONTINUED: RÉSUMÉ AND COMMENTS.

WHAT, then, is the true interpretation of the conditions and influences which are here said to have the potency to incite pulmonary consumption? Is there a unanimity in their trend, and how much support does one give to the other? A summary of these points will be of great assistance in appreciating and perceiving the causes of pulmonary consumption in a true light. At the outset it may be said that the histories of the first fifty-six cases related in the preceding pages illustrate that injury or disease of, or pressure on the vagi, is followed by pulmonary consumption. The next twenty-two cases show that poisoning of the nervous system, and especially of the vagi by alcohol and syphilis leads to pulmonary consumption. In the next place it is shown that mercury and lead have a similar toxic action on the nervous system and likewise exert a special proneness toward the genesis of pulmonary phthisis. Then, in the order in which they are named, come the toxines of typhoid fever, diphtheria, measles, whooping-cough, mumps, influenza, cerebrospinal meningitis and beriberi, which have the power of disintegrating the nervous system, and very frequently are followed by phthisis and other forms of lung disease. Diabetes and leprosy, two diseases in which the nervous system is always markedly implicated (especially in the case of the latter) are often associated with consumption. Then, lastly, it was demonstrated that multiple neuritis, locomotor ataxia, bulbar paralysis, epilepsy, hysteria, idiocy, mutism,

asthma and insanity display a strong predilection toward the genesis of pulmonary consumption.

Then, again, it was found that those influences that predispose to pulmonary consumption most powerfully did so principally by undermining the integrity of the nervous system. Among these is heredity, direct and indirect; direct when the disease is transmitted from a consumptive parentage, and indirect when insanity, epilepsy, hysteria, asthma, alcoholism, syphilis, mercurialism or any other form of intoxication of the nervous system are transformed into pulmonary consumption in the offspring. Indirect heredity is one of the most important factors in the propagation of phthisis, and undoubtedly accounts for many cases of this disease that are ascribed to other than to nervous causes. Indeed the term itself is so bound up with the neurotic origin of consumption that it becomes a superfluity when used in any other sense. Besides the above-named predisposing causes which act, perhaps, wholly by virtue of a weakened resistance in the nervous system, there are a certain order of birth, prolonged grief, prison discipline and civilization which dispose to a similar downward trend.

In addition to these, and which are really more of the nature of exciting than of predisposing causes, are the inhalation of dust and the entrance of foreign bodies into the bronchi. By acting as local irritants they form inflammatory foci preparatory to the lighting up of the phthisical process.

The next and last point in this connection is the part which infection plays in the maintenance of phthisis. The facts in regard to tubercle inoculation, feeding, inhalation and exposure have been impartially collected, and they certainly demonstrate that when tubercle is experimentally inoculated, fed or inhaled, tuberculosis follows as a consequence in many instances, but clinical experience does not indicate that when exposed to the contagion of consumption in the ordinary ways of life, man is liable to contract the disease to any extent. In fact the evidence which has been adduced seems

to prove conclusively that the possibility for acquiring consumption through contagion is extremely small. This is made clear by the statistics of the Brompton Hospital for Consumption in London, of Friedrichshain Hospital in Berlin, of Dr. Brehmer's Sanatorium at Görbersdorf, of Dr. Dettweiler's Sanatorium at Falksenstein, of Soden, Colorado Springs, and Madeira, popular resorts for consumptives, as well as by the statistics which relate to the contagiousness of this disease between husband and wife, which are furnished by Drs. Schnyder, Flint and Leudet.

Moreover, the opinions of the leading authorities on this disease in the world have been quoted respecting the contagiousness of consumption. They are twelve in number, and comprise such names as Laennec, Portal, Ancell, Aufrecht, Dettweiler, Brehmer, Ransome, Leaming, James, Powell, Wilson Fox, and Williams; and, with singular unanimity, not one gives his unqualified endorsement to the contagious theory.

Finally, the history is given how the doctrine of contagion gathered popular force in some of the Italian States and in other countries in the eighteenth century, how the governments of these States passed rigorous laws for the suppression of consumption on the score of contagion, and how in the end these measures, after being operative in some cases for more than half a century, led to disaster and ruin and without any appreciable effect on the death-rate of this disease.

Taking, then, on the one hand, the successful experiments which have been made on the inoculation, feeding and inhalation of tubercle, in connection with the favorable views which were and are held to-day in regard to the contagiousness of this disease, and, on the other, the undivided evidence, as it is gathered from the clinical side of this problem, there appears to be an irreconcilable contradiction between the two. This is, however, more apparent than real. When the an-

tagonism is sifted it will be found that, so far as the origination of consumption is concerned, laboratory experiments are absolutely silent. All that they show is that the disease may be transplanted by a certain method after it has been called into existence by other causes. Clinical medicine shows that consumption often finds its exact equivalent in insanity, hysteria, idiocy or some other form of nervous disease, that it may be frequently transmuted into any of these diseases or they into it from one generation to the next, and that it is engendered anew along family lines and not transmitted from person to person through the medium of contagion. The great difficulty in the discussion of this problem has always been a neglect to distinguish between the origin and the transplantation of consumption. These phenomena are usually treated as if they were one and the same thing, yet they differ as much as sunlight differs from moonlight.

This is well shown in skin-grafting. Particles of skin are planted on denuded bodily surfaces and become thoroughly incorporated and form new skin, yet this artificial procedure gives us no knowledge of the origin and mode of genesis of the skin that is transplanted. Nor does vaccination give us the remotest idea of the source of the cowpox virus. A vegetable graft is also capable of communicating the peculiar properties of the fruit, color of leaves, or of the tree or plant from which it is taken, to the *whole* tree or plant on which it is grafted. Dr. Darwin,* after relating a number of cases in which grafting or budding infused the peculiar characteristics of plants and trees into the stocks which received the grafts, concludes that the process may be regarded as the inoculation of a disease. Now the experimentalist who holds that because grafts possess the power of transmitting their properties to the plant-stock, and that, therefore, all the diversified wealth of vegetable form and beauty is the product of inoculation by grafting, takes about the same position as he

* "Domestication of Animals and Plants," etc., vol. i, p. 474.

who assumes that because pulmonary consumption may be produced by artificial inoculation nature pursues a similar course in producing this disease originally.

Another point will have to be briefly touched upon here before this subject can be dismissed. Notwithstanding all that has been said concerning the causation of pulmonary consumption through a vitiated nervous system and through other influences, it may still be held that the degree of constitutional or nervous depression is immaterial so long as the bodily soil is not impregnated by the tubercle bacillus. That the tubercle bacillus is present in pulmonary consumption and plays a causative factor in its propagation is beyond question, but it does not follow that it is always the cause of the disease, for this is contraindicated by the facts which have already been presented and also by the following considerations: In the first place the bacillus has been found in persons who were, to all intents and purposes, in a healthy state. Pizzini* discovered that out of thirty healthy persons who had no special contact with phthisical people and who died suddenly of cerebral hemorrhage, cardiac syncope, poisoning or suicide, eleven, or 37 per cent., had bronchial glands, with which guinea-piges were inoculated and made tubercular. Straus† examined twenty-nine healthy, or at least non-tubercular persons, who had daily intercourse with phthisical people, and found that one-third of the cases harbored tubercle bacilli in their nasal passages.

In the second place tubercle nodules are frequently acquired by those who dissect tubercular bodies, and without serious consequence. This is certainly tubercular infection. So in the case with Pott's disease, or angular curvature of the spine, which is a tubercular infection, although very rarely followed by pulmonary consumption. The same is true of peritoneal tuberculosis. This is tubercular infection,

* *Zeitschrift f. klin. Med.*, Bd. 21, 314, 1892.

† *Correspondenz-Blatt f. Schweizer Aertzte*, 1894, p. 551.

yet this disease, as has already been stated, is almost invariably relieved by surgical interference.

Here, then, are instances of clear and unadulterated tubercular infection in which the bacilli had effected entrance into the fluid of the body as much as if they had been introduced experimentally with a hypodermic syringe. Why does the infection fail to spread and cause pulmonary tuberculosis as in the case of animals? Is it because the soil is non-receptive? This might be true in healthy persons, but can hardly be said of cases of Pott's disease, and of tuberculosis of the peritoneum. Is it because there is a difference in the vital resistance to the bacilli between man and animals? Or is it principally because the efficiency of the tubercle bacillus as a cause of pulmonary consumption in the human subject is overestimated? All the data that can be gathered on this subject seem to favor the correctness of the latter conclusion.

CHAPTER XII.

PATHOLOGY OF PULMONARY CONSUMPTION.

The Structure of the Respiratory Organs.

The lungs are enclosed within the thoracic cavity, and are divided into five lobes—of which there are three in the right and two in the left lung. The arrangement and topography of these organs are fully described in the following figures:

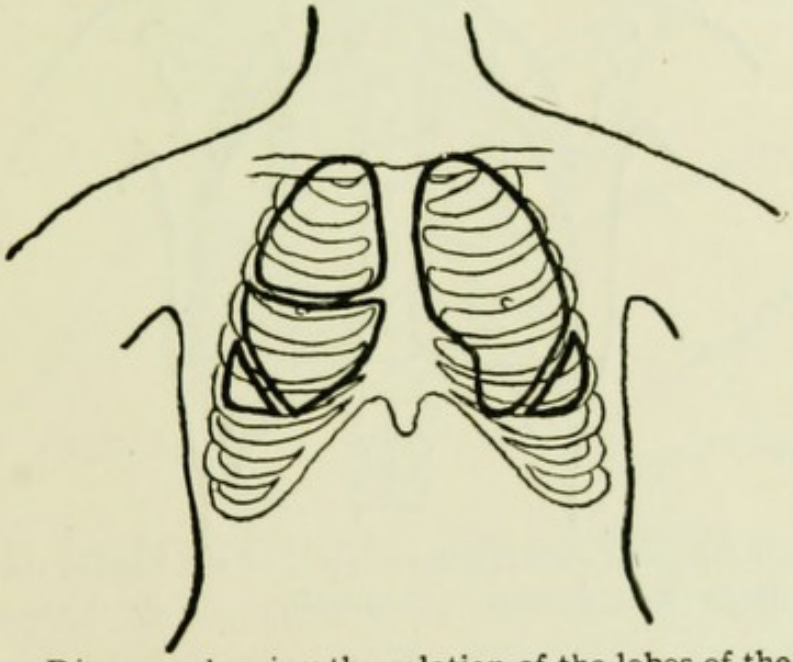

Fig. 9.—Diagram showing the relation of the lobes of the lungs to the front wall of the chest.—*Fowler.*

It may be said that each lobe has a cone-like shape, with its apex directed either forward or backward. Thus the upper lobe of the left and the upper and middle lobes of the right lung have their apices behind and above, and their bases in front; while the lower lobes of both lungs have their apices in front and below, and their broad bases behind.

This is partly illustrated in the diagrams of figures 9 and 10, which show a side view of both lungs. The septum, which separates the upper and lower lobes of the left lung, begins near the top behind and extends diagonally downwards and forwards and ends in front near the base of the chest. The septa which divide the right lung into three lobes also begin at the top behind and extend downward and forward. It appears, therefore, that the upper lobe of the left lung occupies the whole of the front of the chest except a

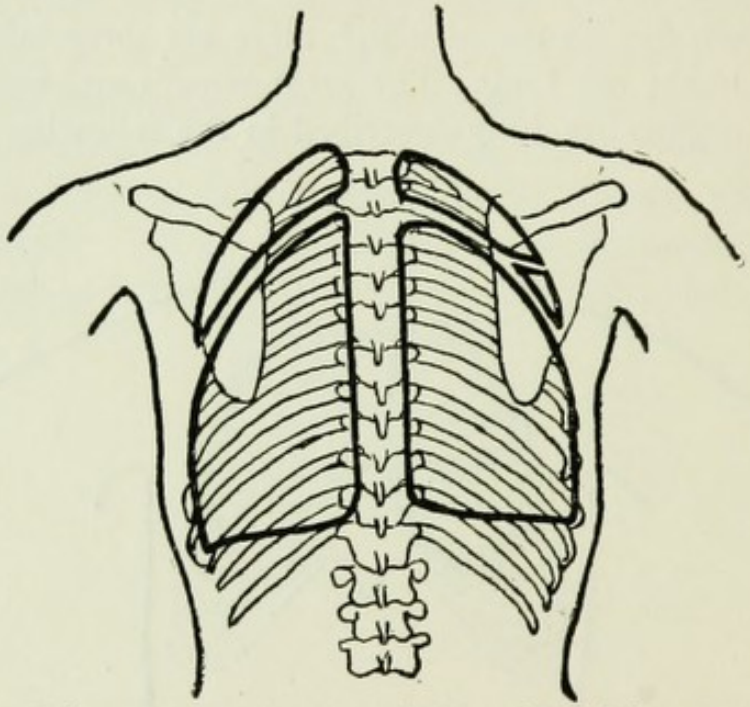

Fig. 10.—Diagram showing the relation of the lobes of the lungs to the wall of the chest, posteriorly.—*Fowler.*

small area at the side, where the anterior part of the lower lobe is seen. The upper lobe of the right lung occupies the front of the chest as far as the fourth rib, and the middle lobe extends from here down to the base of the chest, only allowing a small point of the lower lobe to appear laterally. On the back the lower lobes absorb nearly the whole surface on both sides—extending form the third dorsal spine to the base of the chest. The upper lobes take up the space above

the third dorsal spine, and the middle lobe of the right lung is just visible at the side.

In a biological sense the lungs develop from the ventral wall of the intestinal canal in the form of a short, blind tube, which is attached to the anterior wall of the pharynx. This stalk by simple growth lengthens into a long, thin, blind tube, which becomes the trachea. By subdivision of this tube the bronchi are formed. At various points along the interior of these blind bronchial tubes there are given off, by means of cell multiplication, an ever-increasing number of new sacs, forming a "tree-like branched gland, bearing berry-like ap-

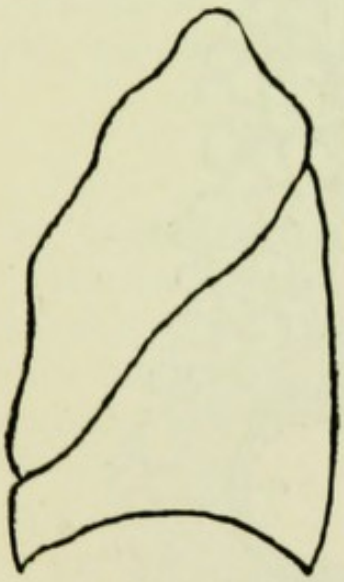

Fig. 11.—Left lung. Side view.

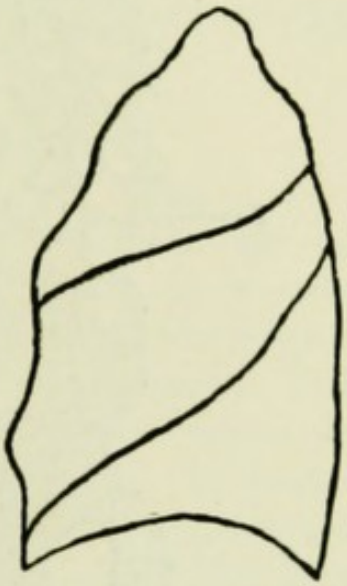

Fig. 12.—Right lung. Side view.

pendages." In the walls of the trachea and bronchial tubes ring-shaped cartilages develop, which keep these structures distended.

The lungs, like all other organs, possess a framework to support their constituent structures. This consists of the pleura, the interlobular and interlobar and intra-alveolar septa, and from these are suspended the bronchial tubes, air cells, nerves, blood-vessels and lymphatics, the relation of each of which will now be considered to the lungs as a whole.

THE PLEURA. The exterior surface of the lungs is covered by the pleural membrane, which consists of an outer, dense layer—the pleura proper—and an inner loose layer, which dips into the lung and divides it into lobes and lobules, as is shown in figure 13. The matrix of the pulmonary

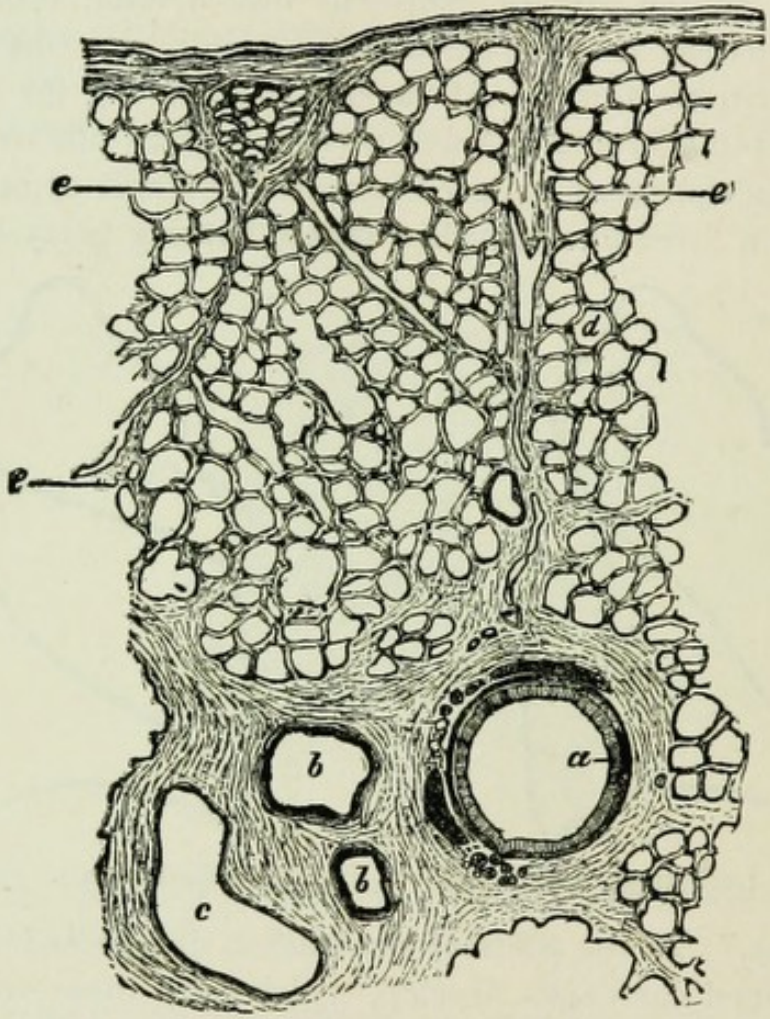

Fig. 13—Section of normal human lung—x 50 diams reduced ¼. *a*, small bronchus; *bb*, branches of pulmonary artery, *c*, branch of pulmonary vein; *d*, air vesicles; *e*, interlobular septa.—*Hamilton.*

pleura is very similar in structure to that of other serous membranes, like the synovial sacs, peritoneum, pericardium, etc., except that it contains an abundant supply of unstriped muscular fibers, between the meshes of which pass the lymphatic vessels in their course towards the bronchial

glands at the root of the lungs, and it will be seen hereafter that the pleural muscular coat bears an important relation to the absorption of lymph and serum from the pleural cavities and to the circulation of the same through the pleural membrane. For on inspiration the intermuscular meshes are widened, the lymphatics are distended and absorption is facilitated, while on expiration the muscular fibres are brought closer together, compress the lymphatics, and in this way propulsion of the fluid contents of these vessels is favored. The pulmonary pleura is covered with a single layer of endothelial cells, between which stomata, or true openings, are situated and which are the beginnings of one set of pulmonary lymphatic vessels.

The Bronchial Tubes.

The bronchi are accessory organs to the air cells, and while they do not occupy a position which is entirely independent it must be remembered that their structure and function differ materially from those of the latter. Transverse section of a bronchial tube shows that its mucous membrane consists of (1) the epithelial layer, (2) the basement membrane, (3) the inner fibrous coat, (4) the muscular layer, and (5) the outer or adventitious coat. This is well illustrated in the following section. If we begin our examination of the different layers of the bronchial mucous membrane from the outside we find that the size and form of the epithelial cells vary at different depths. For it will be seen that those on the very surface are columnar in shape (*b*), and that those at the bottom of the layer are flat and resemble endothelial cells. The latter make up the flat layer of Debove (*c*). It will be observed that between the two layers there are cells that do not seem to be fully developed. They represent the transitional stage of development between the two other forms. The epithelial cells retain their character-

istic forms throughout the extent of the bronchial tubes. At the junction of the latter with the infundibulæ and air vesicles the columnar and transitional forms disappear, and the flat layer alone remains.

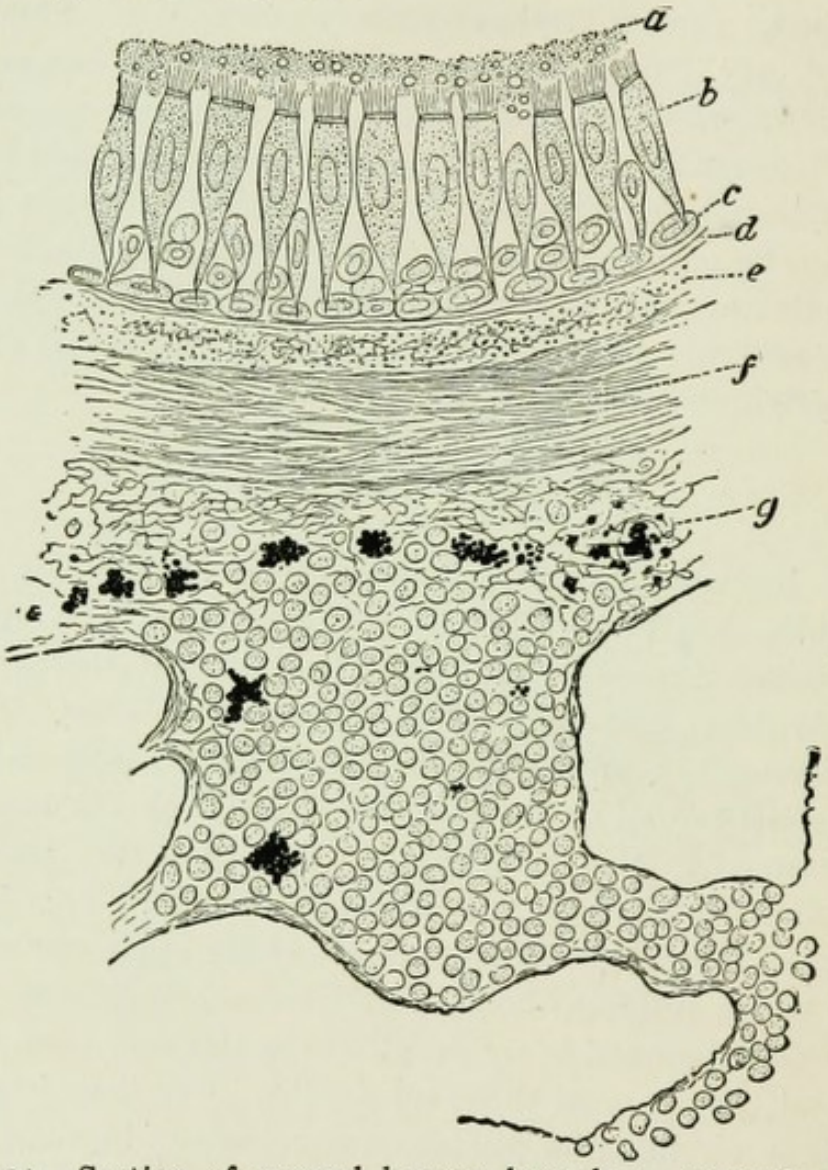

Fig. 14.—Section of normal human bronchus—x450 diams: *a*, precipitated mucus on surface of epithelium; *b*, columnar epithelial cells; *c*, deep germinal layer of cells (Debove); *d*, elastic basement membrane; *e*, inner fibrous coat; *f*, muscularis; *g*, outer fibrous coat with lymphadenoid deposit in it.—*Hamilton.*

Beneath the epithelial layer lies a homogeneous structure called the basement membrane (*d*), the exact nature of

which is not understood. It is clear, however, that it bears a very intimate relation to the epithelial cells, for some of the latter not only rest on but are imbedded in it. It probably serves as a matrix for the production of epithelial cells. It plays a prominent part in bronchial disease, and it seems to act as a shield against the entrance of foreign material into the substance of the lungs.

Next in order comes the inner fibrous coat (*e*), which is chiefly composed of elastic fibres and which gives the fibrous character to the mucous membrane. This coat contains an abundant blood supply from the branches of the bronchial arteries, which form a ramifying plexus immediately under and projecting into the basement membrane, and besides these vessels it also contains a network of lymphatics.

The muscular coat (*f*) comes next. This is composed of interlacing bundles of non-striated muscular fibres, between which are found lymphatic vessels and spaces.

Another important investment of the bronchi is the outer fibrous or adventitious coat (*g*). It has already been shown that the lower layer of the pleura sends fibrous bands down into the lung to form the interlobar and interlobular septa. Now, on the other hand, the fibres of the bronchial adventitious coat unite in as many as three and four different places around the bronchus, with the fibrous bands coming from the pleura and so place the bronchus in the same relation to these bands as that which the hub of a wheel bears to its spokes.

The smallest bronchioles expand into infundibulæ, and these are the chambers with which the surrounding alveoli communicate in common. The alveolar wall consists (*a*) of a flat layer of epithelial cells; (*b*) elastic tissue and small bundles of white, fibrous tissue; (*c*) an abundant supply of capillary blood-vessels derived from the pulmonary arteries, and (*d*) a system of plasmatic spaces, which on the one hand

P, Pneumogastric nerve.

SA, Spinal accessory nerve.

H, Hypoglossal nerve.

GP, Glosso-pharyngeal nerve. F, Facial nerve.

S, Superior cervical ganglion of the sympathetic.

gr, *Ganglion* of the *root* of the vagus.

gt, *Ganglion* of the *trunk* of the vagus.

1, Auricular branch of the vagus (Arnold's nerve).

2, Pharyngeal branch.

3, Convergence of nerves to form *pharyngeal plexus*.

4, Superior laryngeal nerve.

4', Internal branch of superior laryngeal nerve.

4'', External branch of superior laryngeal nerve.

6 6 6, Cardiac branches of the vagus.

5, Inferior laryngeal nerve (recurrent).

5' Cardiac branch of inferior laryngeal nerve.

7, Convergence of branches of vagus to form cardiac plexuses.

8, Pulmonary branches.

9, Œsophageal branches.

10, Gastric branches.

11, Splenic branches.

12, Hepatic branches.

id, Internal division of spinal accessory.

ed, External division of spinal accessory.

IC, IIC, IIIC, IVC, Cervical nerves.

Fig. 15.—Diagram of the pneumogastric nerve and its connections. —*Hermann.*

communicate with the alveolar cavity through the stomata between the alveolar epithelial cells and, on the other hand, with the lymphatic vessels of the interlobular septa and of the deep layer of the pleura.

Nerve Supply.

The nerve supply of the lungs comes from four different sources—the pneumogastric, the spinal accessory, the hypoglossal and the sympathetic. The pneumogastric (*P*) (see figure 15) which is the largest and most important nerve in the body, arises from two nuclei, one of which is situated in the lower half of the floor of the fourth ventricle and the other in the oblongata, near the olivary body. In the jugular foramen, through which it emerges from the skull, is the ganglion of its root (*gr.*) or the jugular ganglion, which in all probability is the homologue of the ganglion on the posterior root of the spinal nerve. From this ganglion is given off the auricular branch (1) which supplies the external ear and the membranæ tympani. Only a short distance below this points is another enlargement, about an inch long, which is known as the ganglion of the trunk (*gt.*). Here this nerve anastomoses freely with the spinal accessory (*SA*) nerve, and receives filaments from the sympathetic (*S*) and hypoglossal (*H*), and gives off the pharyngeal nerve (2). The superior laryngeal nerve (4), which supplies sensation to the mucous mebrane of the larynx, epiglottis and base of the tongue also arises from this ganglion. Next arise the cardiac branches (6.6.6), which converge at 7 to form the cardiac plexus. Then comes the œsophageal branch (9). The next branch is the recurrent, or inferior laryngeal, which is the motor nerve of all the intrinsic muscles of the larynx, except the crico-thyroid, which receives its innervation from the superior laryngeal. The inferior laryngeal branch also gives off a cardiac branch,

which converges at 7 and forms a part of the cardiac plexus. Next in order comes the pulmonary branches (8) which, in connection with the sympathetic, form the anterior and posterior pulmonary plexuses, and which enter the root of the lung and are distributed along the bronchial tubes, the pulmonary arteries and throughout the lung tissue. On the outer surface of the bronchial tubes, as well as in the textures of the lung, are found a large number of small ganglia in connection with these nerve tubes, and in the lung of the calf these ganglia are so large as to be macroscopic. In the comparatively simple lungs of the frog, nerves with numerous nerve-cells in their course are found(Arnold, Stirling), and in the very simple lung of the newt there are also numerous nerve-cells disposed along the course of the intra-pulmonary nerves. Some of these fibres terminate in the uniform layer of non-striped muscle which forms part of the pulmonary wall in the frog and newt, and others end in the muscular coat of the pulmonary blood-vessels (Landois). The nerves of the pleural coverings are also derived from the pulmonary plexuses, and those fibres which are distributed to the pulmonary pleura also have ganglia attached to them. Bischofswerder,* from a series of carefully conducted experiments on rabbits concluded that the vagus as well as the sympathetic act as vasomotor nerves of the lungs. The simultaneous extirpation of the uppermost thoracic ganglion and both vagi was followed by a much stronger hyperæmia and infiltration of the lungs than division of the vagi only.

Other branches of the pneumogastric are those which go to the stomach, liver, etc. Both the left and right pneumogastric nerves go to form the gastric plexus which innervates the stomach. Fully two-thirds of the right pneumogastric form the cœliac plexus, the branches of which are distributed to the arteries of the liver, pancreas, small intestines,

* "Vagus und Sympathicus, die Vasomotorischen Nerven der Lungen," *Dissertation.* Greifswald, 1875.

kidneys and suprarenal capsules. The spleen is also supplied by the same nerves, for irritation of the peripheral end of the vagi produces contraction of this organ (Oehl). The same observer also showed that similar irritation brought on contraction of the bladder.

Blood Supply.

The lungs have a double circulation, consisting, first, of the bronchial or nutrient arteries, which arise from the thoracic aorta and run along with, divide and subdivide, on the corresponding bronchial tubes on each side, nourishing these, as well as the tissue of the lungs, the bronchial glands, and the œsophagus; and, second, of the pulmonary artery, which gives the functional and by far the larger supply of blood to the lungs. The pulmonary artery arises directly from the base of the right cardiac ventricle. It is a wide but short vessel, and ascends as far as the under surface of the arch of the aorta, where it divides into the right and left pulmonary arteries. These branches enter the lung with and cling close to the bronchial tubes, and divide and subdivide with these until they reach the alveoli. From these terminal arteries, which are about one-thousandth of an inch in diameter, the capillary network arises which covers and surrounds each alveolus. The capillaries of this meshwork are very fine and so superficial that only a thin layer of tessellated epithelium intervenes between them and the air circulating in the alveoli, and they frequently project in a loop-like manner into the cavities of the latter. It is also important to remember in this connection that the mucous membrane of the bronchioles, where these join the air cells, receives its blood supply from the branches of the pulmonary artery.

The only connection between the bronchial and the pulmonary blood-vessels is maintained at the point where the bronchioles join the alveoli; but the communication is only

from the former to the latter, and not in a reverse direction, for the pulmonary capillaries can be injected through the bronchial artery, but no liquid can be forced from the pulmonary artery into the latter.

Each of these circulating systems has its corresponding veins, the bronchial veins emptying into the vena azygos and into the superior intercostal veins, and the pulmonary veins are collected into two large vessels which empty into the left auricle. On account of the peculiarity just mentioned some of the blood which is carried by the bronchial arteries is returned to the heart through the pulmonary veins. The pulmonary veins have no valves, but freely anastomose.

From these anatomical considerations it is plain that the amount of blood in the pulmonary circulation is vastly greater than that in the bronchial; that the vessels of the latter are firmly supported by the rigid bronchial tubes to which they closely cling, while the pulmonary capillaries are held loosely and in a sort of a semi-suspended state by the very mobile inter-alveolar septa; and that the walls of the bronchial vessels are strong as compared with the thinness and delicacy of those of the pulmonary capillaries.

The Lymphatic Vessels.

The lymphatics of the lung, although they freely anastomose and form plexuses, and although they all communicate, either directly or indirectly, with the bronchial glands at the root of the lungs, originate from three different sources. First, those which have already been described as being in communication with the pleural cavity; second, those which arise in the walls of the alveoli; and, third, those which come from the bronchi. When they follow and twine around the pulmonary blood-vessels they are called perivascular lymphatics, and when they are in close contact with the bronchial tubes they are called peribronchial lymphatics. The latter arise between the epithelial cells, which line the bronchial mucous membrane, and the former compose a

dense plexus of spaces in the inter-alveolar septa and around the pulmonary capillaries, whence they course along the interlobular and interlobar septa, and through the deep-seated layer of the pleura and finally terminate in the bronchial glands at the root of the lung.

Pathology.

Pulmonary consumption is a slowly progessive disease, the beginning of which, so far as the lungs are concerned, is marked by signs of consolidation and the ending by evidences of excavation. An effort will be made here to consider the morbid changes which these organs undergo during this period and strive to give them a proper pathologic interpretation, and in doing this the author will take the liberty of drawing on the investigations which are contained in the most excellent work* of Professor D. J. Hamilton, of Aberdeen University, Scotland, to whom a lasting obligation is incurred and acknowledged with thanks by the author for permitting this privilege.

Now to any one who is familiar with the material of a dead-house it is clear that many varied *post-mortem* appearances present themselves in phthisical lungs, but on deeper inquiry it will be found that nearly all these varied changes may be separated into the two following forms:

In the *early stage* of the first form there is an absence of acute pleurisy, and, therefore, pleural adhesions are rare; the lung feels vesicular and crepitant throughout, although in the midst of this are found spots of condensation; over its cut surface are seen greenish, yellow pneumonic patches, corresponding about to the size of a lobule; the shape of these patches is sometimes wedge-like, and their borders are extremely well defined; the mucous membrane of the bronchi is always very much congested, and from the cut end of the latter a yellowish catarrhal fluid may be pressed;

* "The Pathology of Bronchitis, Catarrhal Pneumonia, and Tubercle." MacMillan & Co., 1883.

and the cut surface of the lung feels like a mass of frog's spawn.

Second Stage of First Form. Fibrous adhesions of the two pleural surfaces are more frequent; in the pneumonic patches there are formed hard, rounded and irregular-shaped nodules, varying in size from a millet-seed to that of a walnut, which are dry and caseous and have a cream-like color. The small nodules coalesce and form large masses until the greater part of a whole lobe is infiltrated. In the center of such a mass a bronchus is sometimes present. The lung tissue between these nodules is vesicular and somewhat congested. The bronchial mucous membrane is inflamed and discharges a purulent fluid. The bronchial glands are swollen, and are either caseous or tending strongly in that direction.

Third Stage of First Form. The pleural adhesions are usually complete, and the pleural cavity is obliterated. If a portion of the pleural surface remains free, gray gelatinous nodules, which move freely with the pleural membrane, are seen in abundance in such an area. These nodules are confined to the deeper layer of the pleura, and are different in structure from those which are found in the deeper parts of the lung. More will be said of them when the anatomical nature of the other kind of phthisical degeneration will be discussed. The bronchial mucous membrane is in a state of inflammation, and the tube is filled with yellow material. The pneumonic patches which were described as being present in the first and second stages now become very dense and heavy, and their nodules begin to soften in the center. The softening process spreads from this point until the greater part of the nodule, in which it is located, is transformed into a yellow liquid mass. As a rule communication is set up with a bronchial tube, and the fluid is ejected and a cavity remains behind. This is well shown in Fig. 16. Sometimes a num-

ber of small cavities are formed in this way in the same neighborhood, and through the destruction of the intervening partition they are all thrown into a large cavity. Tubercular nodules similar to those found in the deep layer of the pleura are now frequently found on the vocal cords or somewhere in the larynx, and sometimes on the tracheal mucous membrane.

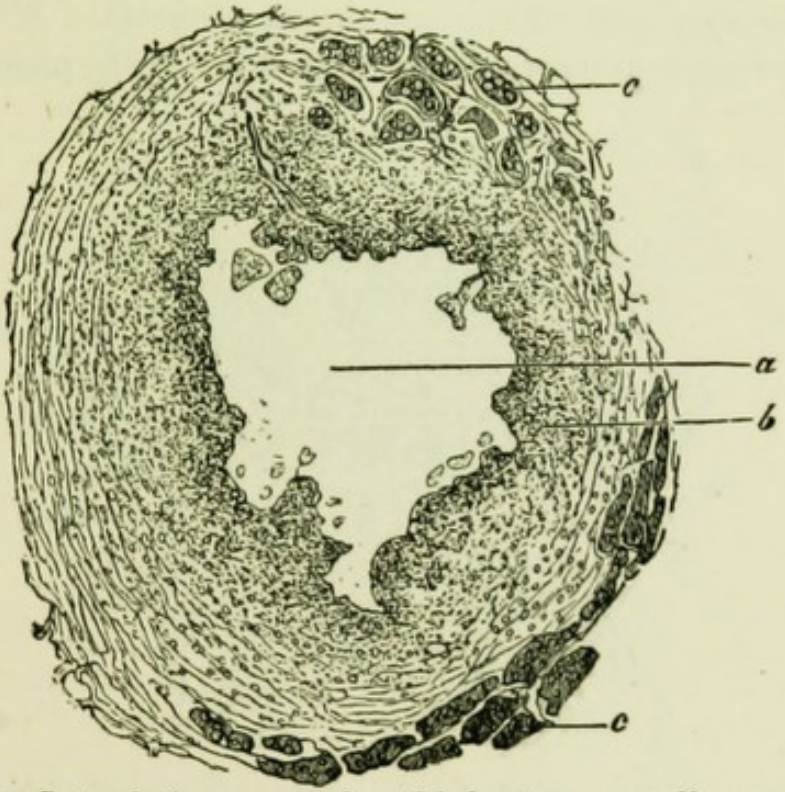

Fig. 16.—Catarrhal pneumonia, third stage x 50 diams.: *a*, the cavity formed by dissolution of the center of the caseous pneumonic mass; *b*, the caseous edge; *c*, the compressed air-vesicles filled with caseating catarrhal products.—*Hamilton.*

Microscopic examination of the center of such a pneumonic patch in its first stage usually shows a small bronchus filled with bronchial epithelial products, while the other part of the patch contains the air cells, which are filled with catarrhal elements and which are grouped around the bronchus, as in shown in Fig. 17.

The catarrhal products in the air vesicles give rise to the condensation, and it is these which may be pressed out.

Fibrin is rarely present in the alveoli, differing thus from croupous pneumonia, in which the fibrous exudation is the principal cause of the consolidation.

The contents of the alveoli at this stage consist of cells and mucous fluid. The cells differ in size and contour. Some are large and flat, and usually have two or more nuclei, while others are small with single nuclei. Some undergo fatty degeneration and others contain oil globules. A very few leucocytes are present. In the deep layer of the pleura and in

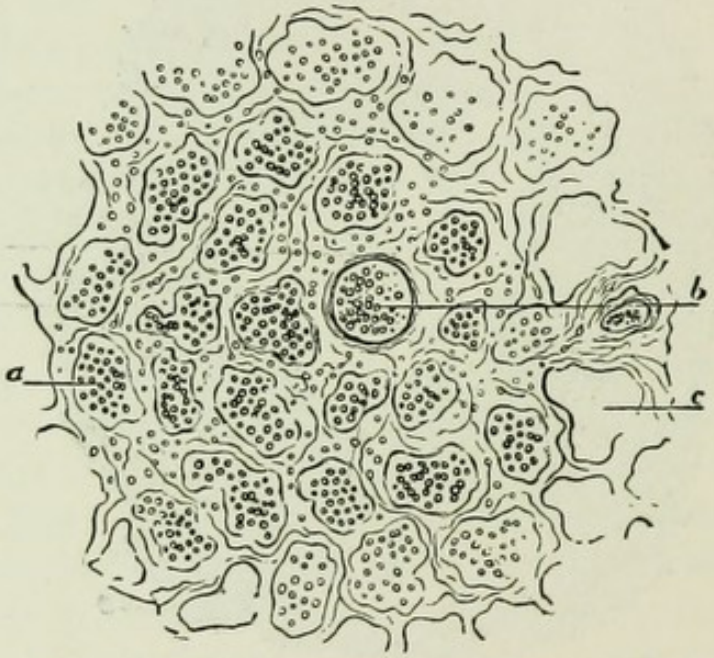

Fig. 17.—Acute catarrhal pneumonia. Group of air-vesicles and small bronchus infiltrated with catarrhal effusion x50 diams. *a*, infiltrated air-vesicles; *b*, small bronchus, also infiltrated; *c*, an empty air-vesicle at the periphery of the pneumonic patch.—*Hamilton.*

adjacent alveoli slight hemorrhages are seen. The cells originate from the epithelium which covers the walls of the alveoli. These germinate rapidly and accumulate. Fig. 18 gives a view of an alveolus and its contents at this stage of the process.

If the disease process is acute the catarrhal contents of the alveoli undergo fatty degeneration, and the products of which are discharged; but if it assumes a chronic character

the accumulation becomes more dense, tubercle bacilli develop and caseation ensues.

When a single nodule of such a pneumonic patch as is described above has advanced to the stage of caseation it presents the following appearances: Under a low power it is seen to be composed of a group of vesicles distended with

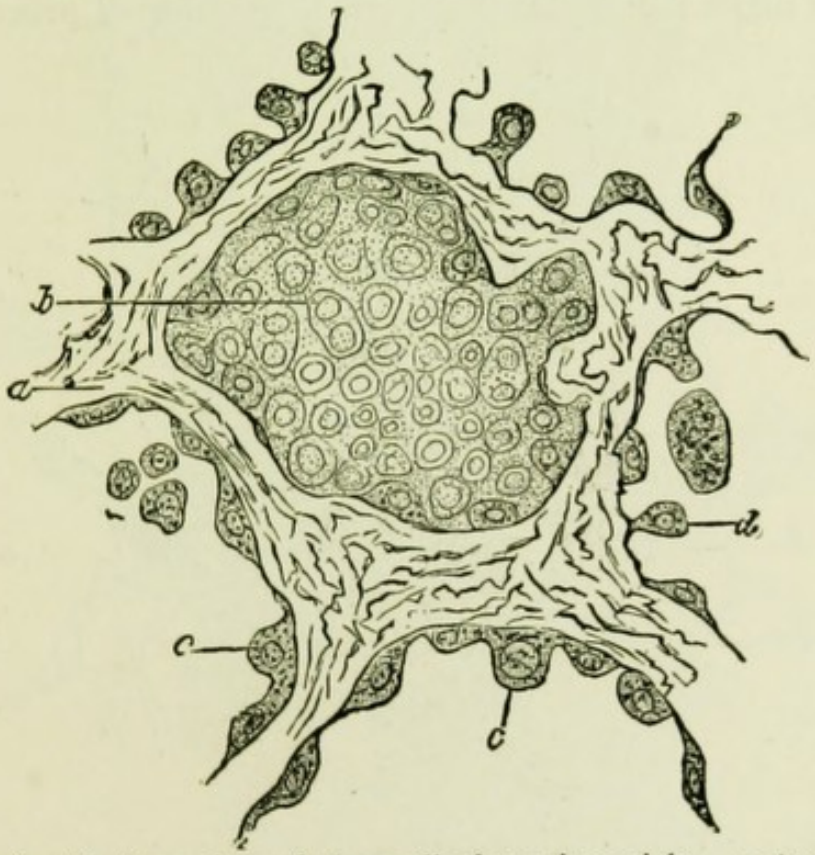

Fig. 18.—Surface view of the wall of an air-vesicle. *a*, transverse section of alveolar wall; *b*, alveolar cavity, showing the alveolar wall covered by germinating epithelium; *c*, germinating epithelium seen on profile; *d*, one of these germinating cells separating from alveolar wall. x400 diams.—*Hamilton.*

solid constituents, as represented in Fig. 19.

In the center of the nodule is located an infundibulum, and, according to Professor Hamilton, if this had "been traced a little upwards it would in all probability have been found to be continuous with a small bronchus similarly occluded. Around the center are grouped the infiltrated vesicles, and at

the periphery of the nodule are seen the air vesicles in a comparatively healthy state.

When a similar nodule and its surroundings are seen under a higher power we get such a view as is represented in Fig. 20.

This represents an injected preparation, and the capital letters mark the different shades of the morbid process. In

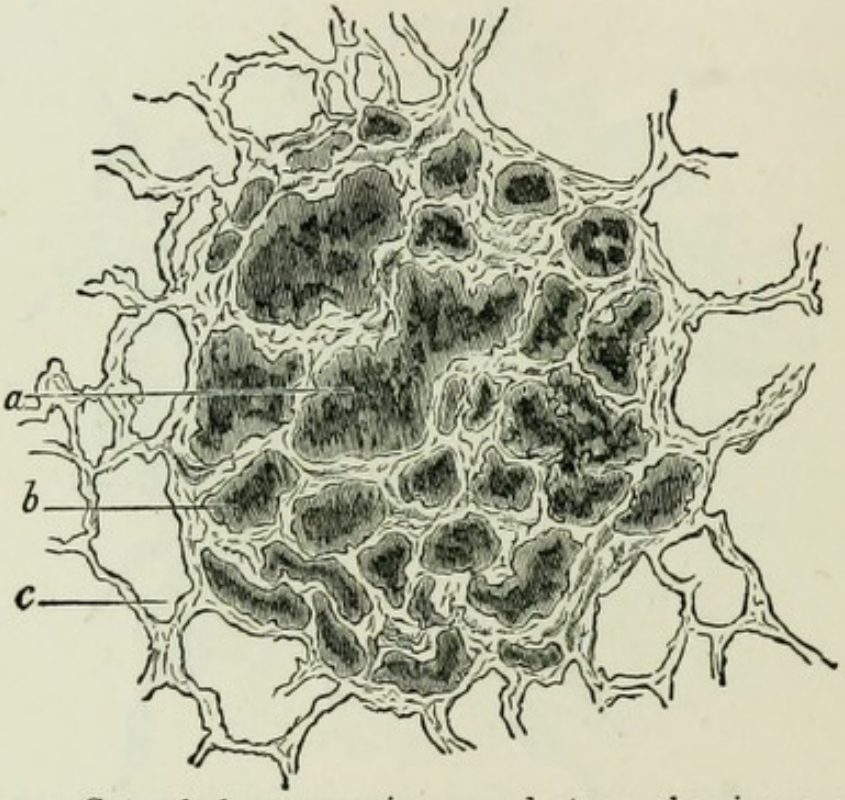

Fig. 19.—Catarrhal pneumonia, second stage, showing a caseous nodule magnified 50 diams. *a*, infundibulum filled with caseous material; *b*, air-vesicle distended with the same; *c*, neighboring air-vesicles comparatively healthy.—*Hamilton.*

the area A are observed the outlines of the air vesicles, with the injected capillary blood-vessels in their walls. The cavities of the air vesicles are moderately distended with catarrhal products. The area covered by B corresponds to a part nearer the center of the nodule, in which the catarrhal cells become packed more closely into the alveolar cavities and the pulmonary capillaries are less numerous and pervious; C

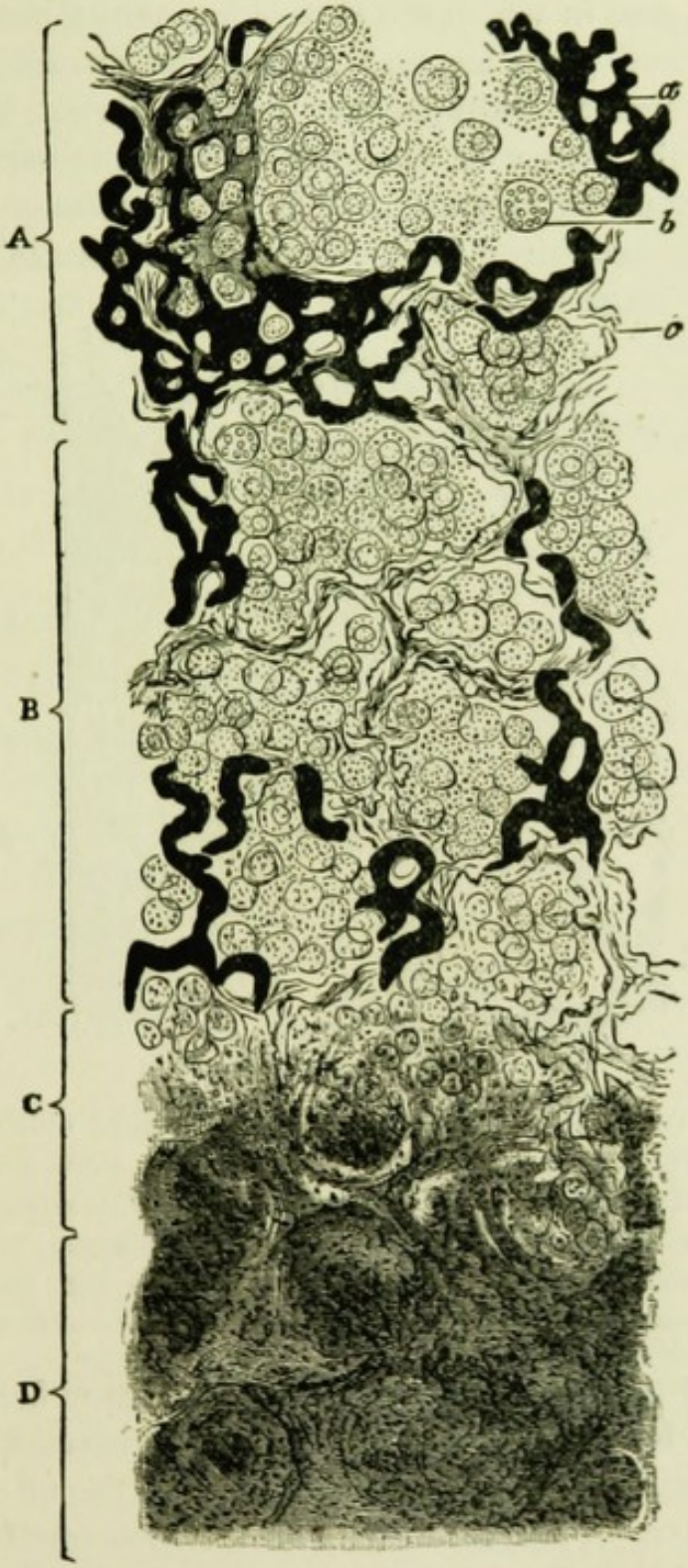

Fig. 20.—Catarrhal pneumonia, second stage, showing a nodule magnified 350 diams. A, B, C, and D represent different areas in the nodule from the periphery towards the center. *a*, injected capillaries of alveolar wall; *b*, catarrhal cells in alveolar cavities; *c*, an alveolar wall.—*Hamilton.*

shows the area in which the nodule becomes a dense, dusky, shrivelled and granular mass and in which the capillaries and the vesiclular walls are lost in the débris. The area D comprises the center of the nodule, which is very compact and in a state of caseation. The original lung tissue can scarcely be recognized in it. Tubercle bacilli are also present in this stage of the disease.

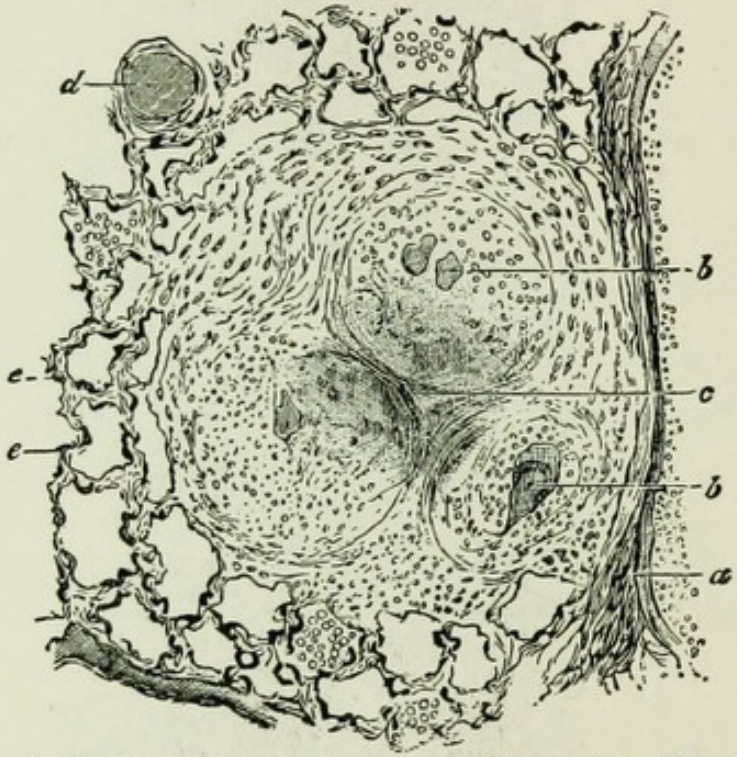

Fig. 21.—Tubercle of lung, two to three weeks old. X50 diams. Source of infection was a caseous peritonitis. *a*, portion of wall of a branch of the pulmonary artery; *b*, giant cells with concentric arrangement of fibrous tissue; *c*, center of tubercle beginning to caseate; *d*, small branches of pulmonary artery seen on transverse section; *e*, injected capillaries of the alveolar walls.—*Hamilton.*

The *post-mortem* appearances of lung tissue belonging to the second form are as follows: There is practically an absence of pleurisy throughout the course of the affection, but the deep layer of the pleura is studded with small tubercles. The same kind of tubercles very often permeate the lungs throughout when the disease-process is acute, or are confined

to the interstitial tissue of certain parts of the lung, as to the peribronchial and perivascular tissue or lobular septa, when this is chronic. These nodules or tubercles are of a gray color, and may become yellow, and have the uniform size of a mustard seed and are of a gelatinous consistency, have a sharply defined border, are devoid of blood-vessels, and either run in lines along the course of a small branch of the pulmonary artery, which is their most common distribution, or they are grouped in small clusters. They do not, like those of the first form, run together and form large nodules. The intermediate lung tissue is vesicular and does not readily collapse when cut into.

When one of these tubercles is viewed under a low power of the microscope it is seen that its borders are sharply marked off from the air vesicles, as is shown in Fig. 21. One of its striking features is its close proximity to a branch of the pulmonary artery *a*. It pushes the pulmonary tissue before it and flattens the air cells in its surroundings. It contains tubercle bacilli, is cellular in character, contains several giant cells and is beginning to caseate in its center *c*. On the other hand, the tubercles, in place of being found in close proximity to the pulmonary capillaries are seen to abound along the course of the lymphatic vessels, and in this condition the infiltration is more likely to be confined to a limited lung area.

The difference between these two forms of pulmonary infiltration is shown in the following side-by-side arrangement of the chief characteristics of each:

First Form.	Second Form.
1. Pleurisy present.	1. Pleurisy absent in early stages.
2. Air-cells are filled with catarrhal material.	2. Air-cells not filled with catarrhal material.
3. Bronchus or infundibulum often in center of infiltration.	3. No bronchus or infundibulum in center of infiltration.
4. Tubercle-nodules composed chiefly of catarrhal cells.	4. Tubercle-nodules are real structures.
5. Tubercle-nodules of irregular size.	5. Tubercle-nodules of size of a mustard seed and are uniformly round.

6. Deep layer of pleura contains no tubercles as a rule.	6. Deep layer of pleura always contains tubercles.
7. Air-vesicles at periphery of nodules comparatively healthy.	7. Air-vesicles at periphery of nodules are compressed.
8. Disease locates itself in spots throughout one lung.	8. Tubercles of the same size and kind that are found in the deep layer of the pleura, permeate both lungs throughout, or are confined to small areas in different parts of a lung.
9. No regularity in the distribution of the tubercle-nodules.	9. The tubercle-nodules are distributed in lines or in clusters around the pulmonary blood-vessels, bronchioles, or along the course of the lymphatic vessels.
10. Frequently coalesce to form larger nodules.	10. The tubercle-nodules preserve their individuality and do not coalesce.
11. Tubercle bacilli present.	11. Tubercle bacilli present.

A true recognition of the histologic differences which obtain in the various destructive processes of the phthisical lung as above outlined is a very important consideration, inasmuch as it demonstrates that there is not only no unity in these changes, but that they advance on separate and widely divergent lines. Indeed it appears that they frequently stand related one to the other as cause and effect. This dual nature of phthisical involution of the lung has long been recognized, but nowhere has it been so explicitly defined and interpreted as it has been by Professor Hamilton in the work already referred to and whose ideas will be closely followed here. Laennec* recognized all deposits in the lungs as tubercular, but subdivided them into gray and yellow tubercles. Rokitansky† divided pulmonary tubercles into interstitial tubercle granulation and tubercle infiltration. The first consisted of round gray, feebly transparent miliary bodies which are deposited in the parenchyma of the lung; and the second he regarded as a pneumonic-tubercular product. Virchow‡ demonstrated that Laennec's gray tubercle was composed of connective-tissue growths, and that his yellow tubercle was a caseating catarrhal pneumonic nodule. He called the former a gen-

* *Op. cit.*

† "Handbuch der allgemeinen pathologischen Anatomie," Wien., 1846.

‡ "Die krankhaften Geschwülste."

uine tubercle, and the latter catarrhal pneumonia. Villemin* discovered that by inoculating rabbits with cheesy material an eruption of miliary tubercles can be produced which are in all respects similar to the genuine tubercle of Virchow. Cohnheim* laid down the dictum that "all is tubercular which by transference to properly constituted animals is capable of inducing tuberculosis, and nothing is tuberculous unless it has this capability." Koch† discovered a specific bacillus in tubercular deposits which, in being inoculated in animals, produces an eruption of tubercles. He found the tubercle bacillus in miliary tuberculosis, in cheesy bronchopneumonia, in tubercle of the brain, in intestinal tuberculosis, etc.

From the above it appears that Laennec's view was, in the main, correct, *viz.*, that there are two kinds of tubercle in the lung, the one is the gray miliary tubercle and the other is the yellow nodule undergoing caseation. In accepting this definition of tubercle it must be borne in mind that the latter formation is merely a nodular aggregation of catarrhal cells, and is not a tubercle at all, according to Virchow's meaning. The former is a connective tissue growth, of the size of a mustard seed, a true tubercle, and is found in the phthisical degeneration which has been described above as the second form (p. 259), while the latter is nothing but a rounded nodule of varying size, composed chiefly of epithelial cells in a state of cheesy metamorphosis, which is neither by nature nor by structure a true tubercle, and which accompanies the phthisical changes that have been noted in the first form (p. 266). Now, after having digressed somewhat in order to define the nature of tubercle and the morbid changes with which it is and with which it is not associated, an effort will be made to trace the clinical development of pulmonary

* "Cause et nature de la tuberculose," *Gaz. hebd.*, 2 Ser. ii (xii) 50, 1865.

† "Die Tuberculose v. Standpunkte d. Infectionslehre," 1880.

‡ *Berliner klin. Wochenschrift*, April 10, 1882.

consumption in its varied pathologic forms and ramifications.

It will be recalled that the pneumonia patches already spoken of were said to be the result of an accumulation of catarrhal material within the air vesicles. As time advances this accumulation becomes so dense that it compresses the blood-vessels within itself as well as those in its periphery, and that it cuts itself off from all sources of nourishment. While this process of condensation is going on, and probably antedating it, another change of equal importance in point of destructiveness is in operation, *viz.*, a thickening of the intima of the blood-vessels which are lying within the area of infiltration, and in consequence an occlusion of their lumen and finally an obliteration of the blood supply channels. This interesting feature will be discussed more fully when the integrity of the nerve supply in relation to the affected lung-area is considered (see p. 284). From this it will be seen that there are two agencies at work which tend to isolate the seat of infiltration from its blood supply. It will also be readily understood that after such isolation has occurred the material included in this detached territory must undergo retrograde metamorphosis of some kind, and its usual, if not its constant, termination is in caseation. This process of cheesy softening begins in the center of the infiltrated mass, and spreads outwards until it sets up communication with a bronchial tube when its contents are discharged and a cavity in the lung remains, as has already been noted.

Caseation marks the beginning of a new epoch in the life-history of pulmonary consumption. From the very beginning of this process and even after excavation has occurred tubercle bacilli, which almost invariably show their presence at this time, are absorbed by the lymphatic vessels and are distributed along their course through the interlobar and interlobular septa, and the deep-seated layer of the pleura on their way to the bronchial glands at the root of the lungs. This virus does not seem to be taken up by the pulmonary

blood-vessels, owing to the reason, in all probability, that the latter are partially or completely occluded by thickening of their internal coat.

What happens, then, after the tubercular virus is absorbed by the lymphatics? At this stage rows of small

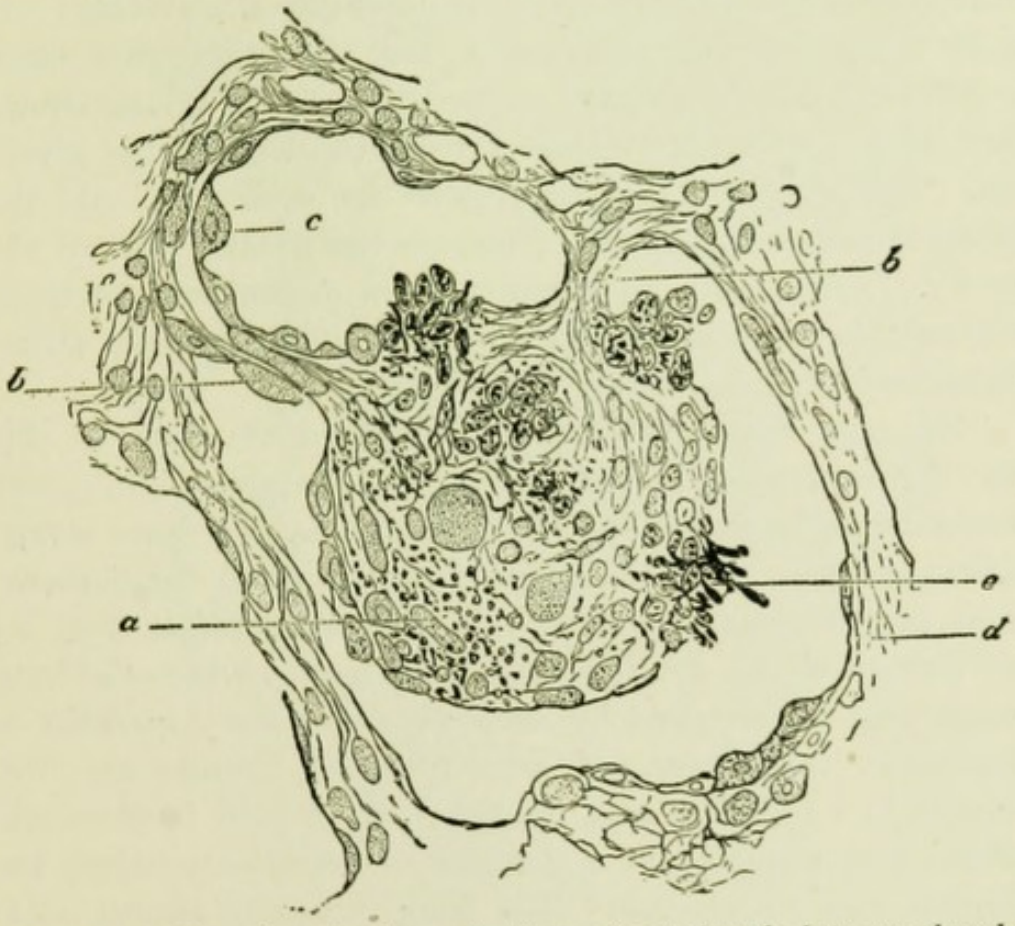

Fig. 22.—Showing the invagination of a tubercle into an alveolar cavity. *a*, the tubercle in an early stage of development; *b*, *b*, the alveolar wall; *c*, alveolar epithelium in a neighboring cavity; *d*, the continuation of the alveolar wall; *e*, pigment particles, originally situated in the fibrous tissue of the alveolar wall, but which have now been carried forwards, and form part of the tubercle-nodule.—*Hamilton.*

nodules are observed in the alveolar walls, the interlobular and interlobular septa, as well as in the deep layer of the pleura, which on investigation will be found to be genuine tubercle-growths within the lymphatic vessels, which never get larger than a millet or a mustard

seed, and which are the irritation-products of the virus on the walls of the lymphatic channels. Professor Hamilton (*op. cit.*, p. 203) in describing the pathology of this form of tubercle says: "The first thing observable is a little swelling on one side of an air-vesicle. This increases in dimensions, and then invaginates itself into the alveolus. It is by this process of invagination into the air-sac that space is afforded for the growth of the tubercle. Fig. 22 shows how such a tubercle originates from the wall of the alveolus. The walls of the air-vesicle are seen at *d*, and the tubercle nodule is noticed to occupy the greater part of the cavity. The nodule, as it increases in dimensions, becomes polypus-like by drawing after it some of the alveolar structures, and these constitute a pedicle.

"Not only does an isolated tubercle nodule grow in this way by invagination, but, when one has so arisen, many secondary outgrowths may be produced by the same invaginating process from the original mass. Such conglomerations of giant-cell systems as those represented in Fig. 23 are formed in this manner, and the extent to which the lung tissue may be involved by such supernumerary growths is sometimes very great. A few tubercle nodules are first formed in a localized area, and then from their borders offshoots are projected by a process of invagination into the neighboring air-vesicles. The lung becomes almost solid at such parts, from the immense mass of tubercles which are thus called into existence. In the course of time the tubercle outgrowth comes to fill the air-sac, and their walls become contiguous."

In this condition we have two processes of disintegration going on. First, that which begins by filling and packing the alveoli with catarrhal products, and ends by the latter undergoing caseation and by the formation of cavities; and, second, the absorption of tubercular virus generated during caseation by the lymphatic vessels, and the production of a

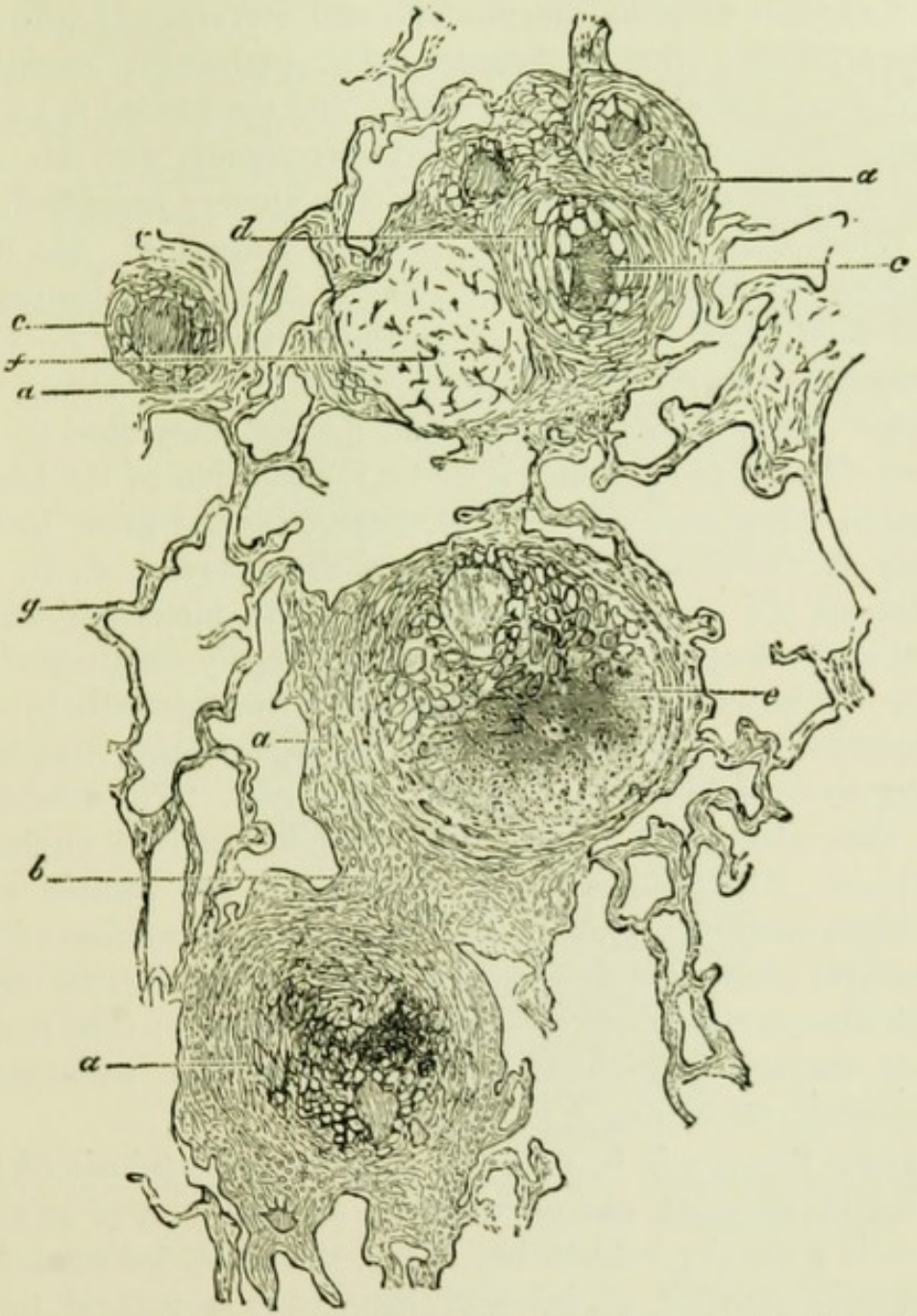

Fig. 23. — Showing tubercle of the lung. *a*, *a*, *a*, *a*, four tubercles; *b*, thickened interstitial tissue uniting two tubercles; *c*, giant-cells; *d*, giant-cell reticulum; *e*, center of tubercle caseating; *f*, a giant-cell system which has become converted into a mass of fibrous tissue.—*Hamilton.*

crop of tubercles along the course of the latter. The first is a chronic catarrhal pneumonia, and corresponds with the characteristics that distinguish that pathologic condition laid down above, as the first form; and the second is a genuine pulmonary tuberculosis, and corresponds with the second form of pathologic characteristics already described.

What, then, is the destiny of these tubercles? Do they undergo caseation like the nodules of catarrhal pneumonia, and thus become sources of further infection, or do they remain as tubercles? Hamilton holds that they rarely undergo cheesy metamorphosis, but, instead, they tend to become fibrous and produce a cirrhotic condition of the lungs. Indeed when one reflects that these tubercles grow in the body of the fibrous framework of the lungs, *i. e.*, in the walls of the alveoli, the interlobular and interlobar septa, and in the deep layer of the pleura, it is easily comprehended how the latter structures are incited to overgrowth by the stimulus which is derived from the germinating tubercles lying in close proximity. Hamilton* says: "The tubercles in such places are formed first, and a localized cirrhosis follows. An organ which is cirrhotic in one limited area to begin with may thus, by the fibrous transformation of the tubercles which are developed from it, become wholly beset with fibrous tissue, the tubercles in the course of time losing their characteristic shape and giving rise to a widespread interstitial thickening."

From this it will be seen that genuine tuberculosis of the lungs is, after all, not such a fatal and destructive process as it is generally held to be. Flint, the elder, believed that a certain number of cases phthisis have a natural tendency to recovery, and the above statements strongly corroborate this opinion, which was solely based on clinical observation. True chronic tuberculosis, then, instead of being destructive, really tends to conservation of the lungs and in

* *Op. cit.*, p. 209.

time brings about that much desired effect, *viz.*, fibrosis of these organs.

While it is true that chronic tuberculosis of the lungs is a conservative procedure in itself, it is also true that its indirect effects are not always so favorable. As has already been stated, the growth of tubercle causes a thickening and induration of the interstitial tissue of the lung, *i. e.*, of the interalveolar, interlobular and interlobar septa, and pleura,

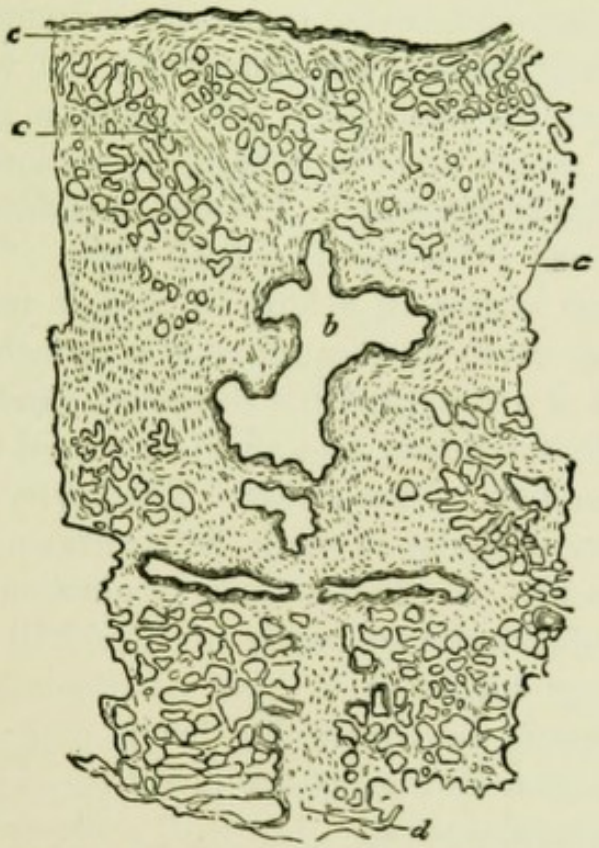

Fig. 24.—Chronic interstitial pneumonia. 2 diams. *a*, thickened pleura; *b*, bronchiectatic cavity; *c*, thickened lobular septum; *d*, thickened septum running inwards.—*Hamilton.*

and this implies a simultaneous contraction of this tissue, which leads to some well-marked changes in the bronchial tubes. It is well shown by Hamilton that the interstitial tissue of the lung radiates somewhat from the bronchi, as the spokes of a wheel do from the hub, and that when cirrhosis of a lung, or a part of a lung, takes place the shortening

of the interstitial tissue draws on the bronchial tube and distends its cavity in a somewhat uneven and irregular manner and produces what is known as bronchiectasy, as is shown in Fig. 24. The dilatation of the bronchial tubes would be less marked and would, perhaps, not occur at all were it not for the adhesions which exist almost invariably between the two pleural surfaces at this time. Independent of this the tube would be the fixed point, and any shortening of the interstitial tissue under these circumstances would draw the lung-tissue toward the tube; but when the costal wall becomes the fixed point the condition is reversed and the tube is drawn upon in different directions, and its caliber is gradually distended. On the mechanism of bronchiectatic cavities Professor Hamilton says: "Fig. 24 represents a large section of a lung in chronic interstitial pneumonia. The cavity in the center is a bronchiectatic cavity, and around it are noticed the thickened bands of cicatricial tissue, while the pleura, also much increased in bulk, is seen at the upper part of the figure. The cavity, it will be noticed, is very irregular in shape, being drawn out into angular projections. If these angular projections be looked at it will be observed that bands of cicatricial tissue run off into the lung substance in lines corresponding with each. If we represent the contracting bands diagrammatically in the scheme seen in Fig. 25 the direction of the contraction will be better understood. Let *P*, *P* represent the thickened pleura, *B* the bronchiectatic cavity, and *F*, *F* the fixed points in the pleura on the one side, and at corresponding parts of the pleura on the other. If the contracting bands of cicatricial tissue shorten towards their central points the traction will be exerted in the direction of the arrows, and the conclusion is irresistible that the bronchus, being the weaker of the two attached points, must become dilated."

Another point of importance in this connection is that bronchiectasy may become a new source of tubercular in-

fection. A cavity of this kind gives the opportunity for the accumulation and caseation of catarrhal material, the virus of which is absorbed by the lymphatic vessels and produces fresh tuberculization and fibrosis in the surrounding area.

That which is true of the lymphatic vessels as carriers

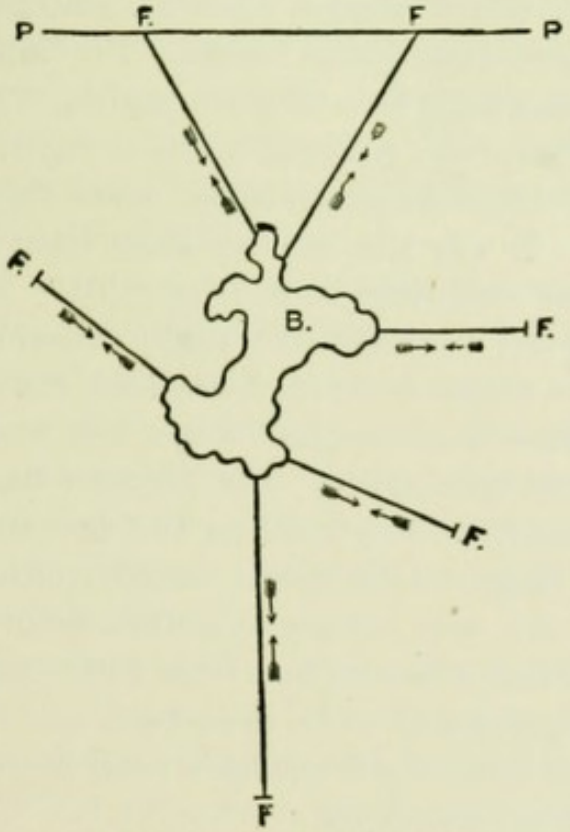

Fig. 25.—Scheme of Fig 24. P, P, pleura; F, F, attached points in pleura and surrounding parts; B, bronchiectatic cavity.—*Hamilton.*

of tubercle virus is equally true of the blood-vessels when the proper conditions prevail, with the exception that the latter carry on this process on a more extended scale. One reason why the blood-vessels of the lung do not always take an active part in the diffusion of tubercle bacilli in pulmonary phthisis, as has been noted already, is that the lumen of those which are involved in the pulmonary infiltration is occluded either partially or wholly, as already observed, and are, therefore, in a great measure incapable of absorption. But when the focus of caseation lies outside of the lungs it seems that sometimes the general circula-

tion becomes an active participant in the absorption and distribution of the tubercular virus. Professor Hamilton* details an instructive example of this kind of tubercular diffusion, which, on account of its scientific interest, will be reproduced here.

"The subject was a woman, aged 20, who gave birth to a child thirty-three days before death. Previous to this she had, from all accounts, been in good health. On the seventh day after delivery she suffered from a rigor, followed by considerable fever, which continued from this time up till that of death. It was also evident from the symptoms that the patient was suffering from peritonitis. The only pulmonary symptoms were those of slight bronchitis. She rallied to a certain extent at one period of the disease, but about seven days before death became worse and finally died with signs of cerebral meningitis. The dates of the case are important, inasmuch as they bear on the age of the tubercles found in the lungs. Her illness dated from a week after delivery, and this was twenty-five days before the date of her death; so that a month had been sufficient to induce all the morbid appearances to be described, and the tubercular growths in the lung could not have been more than from a fortnight to three weeks old.

"After death there was found to be extensive peritonitis, of quite recent and also of somewhat older date. In many places, more especially behind the uterus, the peritonitic lymphy effusion had become caseous, and here and there this had undergone softening. Covering the peritoneum, more especially in regions adjacent to the softening caseous effusion, there were large numbers of gray tubercles in the peritoneum, running in lines along the course of the lymphatic vessels leading to the under surface of the diaphragm. Nearly all the organs showed tubercles of recent origin, and there was extensive cerebral tubercular menin-

* *Op. cit.*, p. 166.

gitis. The lungs were tuberculous throughout. There was no evidence of recent pleurisy, but the pleura was beset with tubercle nodules situated in its deeper layer, and similar nodules were distributed in immense numbers through both lungs. They were round, had a sharply-defined border, of a gray color, had a gelatinous aspect, and were all about the size of a mustard seed. They either ran in lines along the course of a small branch of the pulmonary artery or they were aggregated in little clusters. The former was the commoner of the two arrangements. There was no coalescence between the nodules.

"In this case caseation ensued in the peritonitic effusion; this softened, the caseous débris was absorbed and gave rise to the formation of tubercle in various organs. The tubercles in the peritoneum were evidently of local formation, and had their origin in the lymphatics. In the other organs there is every reason to believe that the blood-vessels were the means of transmitting the caseous products, and that the tubercles were formed within them."

A microscopic examination of a tubercle that is propagated through the blood-vessels is shown to be similar to one that is propagated through the lymphatic vessels, except that the former appears to be formed either from the endothelium of the capillary blood-vessels or from the connective tissue elements of the alveolar wall, while the latter springs from the lymphatic vessels. In any event it is a nodular growth, originating in the connective tissue of the alveolar wall, and which invaginates itself into the alveolar cavity.

It has been conclusively shown in the foregoing pages that caseation is an important factor in the genesis and propagation of pulmonary phthisis, and there is no doubt that the latter frequently arises from this source when it is attributed to other causes. The instances which have been given of caseation occurring in connection with chronic catarrhal

pneumonia and bronchiectasy are the most common and most prolific sources of pulmonary tuberculosis, while caseation outside of the lungs, as in the case just quoted from Hamilton, is an instance where the disease originated in the pelvic cavity and is of more rare occurrence. Other cases of tuberculosis arise from caseous glands, strumous joints, caseation of testicle, etc. Indeed this kind of degeneration precedes and accompanies tuberculosis so often that it is regarded as an almost constant precursor of this disease. Buhl* makes the statement that in 384 cases of tuberculosis caseation was absent only in 9 per cent., and intimates that if a thorough examination were made an old, cheesy node could be found in every case of miliary tuberculosis.

It has been noted already that the tubercle bacilli on being carried along by the lymphatic vessels produce an irritation in the interstitial connective tissue of the lung, which causes a thickening and induration of the same. Now it is a well known fact that stone-masons and other persons who work in stone-dust are prone to fall victims to a peculiar disease known as stone-cutter's consumption. When the lung of a person who dies of this disease is examined it will, according to Hamilton,† be found that little nodules about the size of a mustard seed are distributed over the deep layer of the pleura and throughout the interstitial tissue of the lungs, in the course of the lymphatic vessels. They are hard, with a sharply defined outline, have the appearance of tubercles, and are found to consist in great part of dense cicatricial tissue, with particles of stone-dust lying in their center. They are apparently produced by stone-dust, which is inhaled and absorbed and carried along by the lymphatic vessels.

The essentials of the pathology of phthisis pulmonalis, so

* "Inflammation of the Lungs; Tuberculosis and Consumption," 1874, p. 108.

† *Op. cit.*, p. 202.

far as these have been traced here, can therefore be summed up in the first place, as a chronic catarrhal pneumonia, accompanied by obliteration of the pulmonary blood-vessels and followed by caseation and excavation of the involved lung area, during the progress of which a virus is engendered that, on being carried along the lymphatic vessels, produces an eruption of tubercles in limited areas of lung tissue. This process of tuberculization in turn leads to thickening, hardening and contraction of the interstitial tissue of the lungs and this gives rise to bronchiectasy—another opportunity for the caseation of accumulated products and for further tuberculization. Tubercles thus formed show a greater tendency to fibrosis than to degeneration. In the second place, if the center of caseation is located outside of the lungs, the virus for the production of tubercle may be carried to every important organ in the body through the blood-vessels and produce in the lungs a disseminated crop of tubercles.

Such, then, being the main pathologic condition of this disease, what is the probable nature of the mechanism that underlies and maintains it? The author believes that the nervous system is largely responsible for it, as will be seen from the following evidence: It has previously been shown that pressure on, or division, or disease of the vagi are followed by pulmonary disorders, and an effort will now be made to show the more minute effects of vagus experimentation on the pulmonary organs. After division of the vagi in animals the breathing becomes prolonged, infrequent and laborious, and after death the lungs are larger than normal and do not collapse when the thorax is opened; the cut surfaces show red and intensely congested areas in a remarkably short time, which sink in water, and the bronchial tube contain frothy and bloody mucus, and their mucous surface is red and congested. In from *four* to *ten* hours after division the microscopic appearances are as follows

Pulmonary capillaries are distended, the interalveolar septa are infiltrated, and the alveoli are filled with red blood corpuscles, large granular, nucleated cells and coagulated mucus. The large cells are metamorphosed, alveolar, epithelial cells. In from *ten* to *fourteen* hours the alveoli are filled more densely, there are a large number of lymph cells in the peribronchial and perivascular tissues, the interalveolar capillaries contain an abnormally large quantity of white blood corpuscles, and the alveolar contents are undergoing fatty degeneration.*

These pulmonary manifestations are explained by various observers in the two following ways: First, by the aspiration of food particles and mucus through the imperfectly closed or paralyzed glottis into the lungs. These foreign bodies are supposed to act as local irritants to the pulmonary tissue and to bring about the above-described phenomena, which are known under the name of "Schluck" pneumonia. Second, through paresis of the lungs, which is occasioned by division of their important nerve supply. The former view received the support of Traube,† Frey‡ and Friedlän-

* These results are based on the following named experimental researches:

Friedländer: "Untersuchungen über Lungen entzündungen nebst Bemerkungen über das normale Lungenepithel," *Centralblatt f. d. med. Wis.*, 1873, S. 536.

Genzmer: "Gründe für die pathologische Veränderungen der Lungen nach Doppelseitiger Vagusdurchschneidung," *Pflüger's Archiv*, Bd. 8, 1874, S. 101.

Dreschfeld: "Experimentelle Untersuchungen über die Pathologie der Pneumonie," *Virchow's Jahrbericht*, 1876, ii, S. 2.

Gärtner: *Allgemeine Wien. med. Zeitung*, 1885, S. 38 u. 50.

Schiff: *Archiv f. physiolog. Heilk.*, viii, S. 182, 1849.

† "Die ursachen u. die Beschaffenheit derjenigen Veränderungen welche das Lungenparenchym nach Durchschneidung der Nervi Vagi erleidet," *Gesammelte Beiträge zur Pathologie und Physiologie* i, S. 1; Berlin, 1871.

‡ "Die pathologischen Lungerveränderungen nach Lähmung der Nervi Vagi," Leipsic, 1877.

der,* while the latter was accepted by Schiff,† Ansperger,‡ Gärtner,§ Dreschfeld,‖ Genzmer,¶ and others.

By the adherents of the "Schluck" pneumonia theory it is claimed that a pneumonia similar to that which follows vagotomy can also be produced by section of the recurrent laryngeal nerves. This produces direct paralysis of the vocal cords and facilitates the entrance of food particles into the air passages. That a pneumonia can be produced by section of the recurrents cannot be denied, but whether this is due to the foreign particles which are thus supposed to gain easier access to the lungs is exceedingly questionable. It is much more likely that injury of the recurrent nerves jeopardizes the integrity of the vagi, although to a less degree than if the injury is directly inflicted on the vagi, and thus the so-called recurrent-pneumonia is really brought about through the instrumentality of the vagi. This seems to be confirmed by the following facts:

1. Recurrent pneumonia develops more slowly than vagus pneumonia.

2. Section of both recurrents is not always fatal, while double vagotomy always is.

3. Division of the recurrent in dogs is not followed by death.

4. The injection of from two to four c.cm. of mouth fluid into the trachea of animals (the amount which Traube believed to be aspirated and sufficient to incite pneumonia after vagotomy) produces no disturbance in the lungs, provided the vagi are uninjured.

* "Experimentelle Untersuchungen über die Chronische pneumonie und die Lungenschwindsucht," *Virchow Archiv*, Bd. 68, S. 325.

† *Loc. cit.*

‡ *Virchow's Archiv*, Bd. 9, S. 197 u. 437.

§ *Loc. cit.*

‖ *Loc. cit.*

¶ *Loc. cit.*

5. Ansperger has shown that if, after vagotomy, a tube is inserted into the trachea and the entrance of foreign substances into the bronchi thus prevented the pulmonary changes proceeded as before.

6. In physiological laboratories division of the recurrents in dogs is often resorted to for the purpose of preventing their bellowing, yet they live on without more than temporary inconvenience.

Furthermore, Friedländer has discovered the interesting fact in animals, whose recurrent nerves had been divided and killed in forty-eight hours, that besides the morbid changes in the bronchi and alveoli which follow such section there is also a thickening of the arteria intima, which consists of a proliferation of small, round cells between the elastic coat of the intima and the endothelial coat. This proliferation is more marked in the arterial than in the bronchial wall in these cases, and one can, therefore, speak of a periarteritis as well as of a peribronchitis under these conditions. This adventitious proliferation interferes materially with the arterial stream, for it leads to a narrowing of the lumen of the artery. It first affects the largest and finally the smallest vessels.

Friedländer says* this narrowing of the arterial lumen is of special interest, because a similar affection known as arteritis obliterans plays a prominent rôle in many other pathological processes, particularly in the large series of interstitial inflammatory conditions, as well as in pulmonary phthisis. That such a narrowing of the arterial lumen is detrimental to the nutrition of the artery and its surroundings is very clear, and that such a condition is a further causative factor in the caseation of pneumonia may also be easily understood.

This observation of Friedländer that section of the recurrent nerves causes obliteration of the pulmonary arteries is

* *Op. cit.*, S. 357.

a most significant fact relative to the genesis of pulmonary disease through the instrumentality of the nervous system, for, as has previously been shown, such narrowing and occlusion are common characteristics of chronic catarrhal and interstitial pneumonia in man. On this subject Hamilton* says: "In many of the smaller arteries, however, there is a lesion, which I have always found associated with chronic interstitial pneumonia, and which, perhaps, might account for many anomalous appearances seen in them. It is that extremely important disease of the inner coat of the artery, called 'arteritis obliterans' by Heubner and Friedländer, and which is referred to by the latter as ensuing in

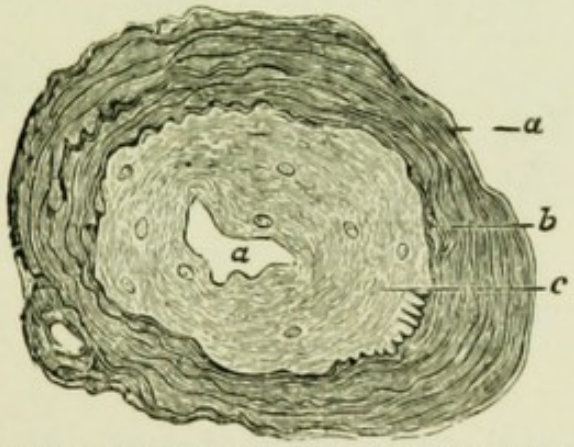

Fig. 26.—Partially obliterated artery in chronic interstitial pneumonia. *a*, Contracted lumen; *b*, elastic laminæ; *c*, thickened intima.—*Hamilton.*

the lungs of animals in which the recurrent laryngeal nerves had been divided. It is of so frequent occurrence and generally so widely spread that I cannot help believing that it plays a most important rôle in bronchitis whenever there is any consecutive interstitial pneumonia present." In Fig. 26 is seen the thickened intima of one of the branches of the pulmonary artery.

Professor Hamilton further states that as a result of this obliteration two changes are conspicuous, *viz.*, caseation and softening of the pulmonary tracts supplied by the affected

* *Op. cit.*, p. 76.

vessels, and dilatation of the capillaries in the surroundings.

Moreover, in discussing the influence of arterial occlusion on the formation of cavities in chronic catarrhal pneumonia Professor Hamilton says: "It sometimes happens, however, that a considerable portion of a catarrhal lung caseates very rapidly and breaks down almost like a slough. In such a case the obliterated condition of the blood-vessels leading to the part, such as that shown in Fig. 27, is ap-

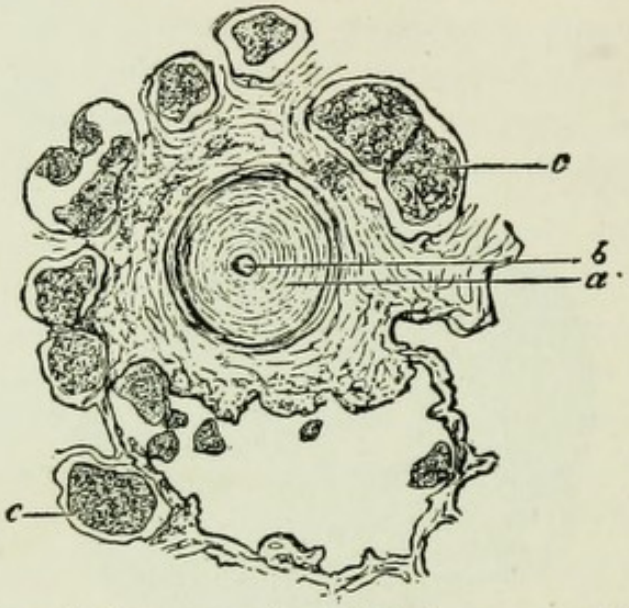

Fig. 27.—Catarrhal pneumonia, third stage. x50 diams. Shows the obliteration which occurs in the branches of the pulmonary artery. *a*, thickened *tunica intima* of a branch of the pulmonary artery leading up to a cavity; *b*, the narrow lumen of the same; *c*, air-vesicles filled with caseous catarrhal secretion and beginning to disintegrate.—*Hamilton.*

parently the cause. As has been demonstrated by Friedländer and others, an obliterative affection of the branches of the pulmonary artery, such as that seen in syphilis, is of common occurrences in phthisical lungs. The above figure represents a portion of a lung taken from the edge of a phthisical cavity, and in its center is shown a transverse section of a small branch of the pulmonary artery leading up towards the cavity . . . . around the obliterated

artery are several air-vesicles containing caseous catarrhal products, all in a more or less disintegrated condition. Now, when this obliterated state of the arterial branches, supplied to an already infiltrated portion of lung, becomes general over a wide area it can easily be perceived how the diminished arterial supply will deleteriously act upon it, and tend to cause necrosis and disintegration *en masse*. This undoubtedly accounts for the presence of those large, rapidly formed cavities seen in certain phthisical lungs."

From the pathologic data which have been given above, it appears that the condition which is called forth in the lungs, through experimental injury to the vagi and recurrent nerves, may be summed up as follows: Dyspnœa, bronchitis, pulmonary congestion, infiltration of alveoli with nucleated epithelial cells, alveolar contents undergoing fatty degeneration, thickening of the arteria intima, and obliteration of the arterial caliber. On the other hand, an almost identical picture is found in the human pneumonic lung, except that in the latter event the process is more chronic, and instead of undergoing fatty degeneration the infiltration becomes caseous, it extends to the limits of excavation, and ends in tuberculization and bronchiectasy if life is prolonged to a sufficient length.

CHAPTER XIII.

GENERAL SYMPTOMS OF PULMONARY CONSUMPTION.

WHILE it is unquestionable that the symptoms of pulmonary consumption vary somewhat in its different types, it is also true that a certain line of general symptoms is characteristic of the disease throughout, and these will be considered here in the following order:

EXHAUSTION OF PHYSIOLOGIC ENERGY. Exhaustion of energy is one of the earliest symptoms of pulmonary consumption, and any agency or influence that has the power of aggravating this tendency also has the potency of provoking this disease. As evidence of such exhaustion the face looks pale and worn, the limbs feel heavy and there is a consciousness that they cannot be handled as readily as they once were; the former vim has, in a great measure, disappeared; the voice is weak and husky at times, the breathing is quick, and the heart's action is easily excited; tiredness is present more or less all the time, and is as marked in the morning on rising as it was in the evening on retiring; there is nervousness and irritability of temper, and altogether there is present an air of depreciation and debility, which borders on the brink of physiologic bankruptcy. It is a state of things which can only terminate in complete dissolution if it continues, and it may be compared to the situation which stares the individual in the face who is in danger of financial failure. With the consumptive it is a problem of spending too much physiologic capital, while with the latter it is one of spending too much moneyed capital. Both have lost and wasted more resources than they can afford, and, unless changed, the career of both will end in disaster and ruin.

Physiologic energy is largely a problem of heredity and nutrition, and both of the latter are possessed in varying quantities by different persons. Those who inherit a large store of energy and maintain good nutrition throughout life are probably entirely exempt from the disease under consideration, while those who dissipate their strength may fall an easy prey to it. On the other hand the stock of energy may be small, but the nutritive processes may be fairly good, and individuals of this kind may, by husbanding their powers and by exercising due caution, escape the disease, yet it is a fact that from this class of individuals the majority of consumptives are recruited. This explains why the weaker, as, for instance, the youngest and then the oldest of a family of children, regardless of a family taint of consumption, are more liable to this disease than the intermediate ones; why those who inherit an unstable nervous organization like the feeble-minded, the mute, the insane, the hysterics, the epileptics, and the members of the families from which these respectively spring are, as we have seen, so very prone to die of consumption. It is essentially a want of tone and stability in the nervous system that constitutes the foundation of a lack of physiologic energy in these cases.

Fever. What is the essential nature of fever? Does it make for health or for disease? Is it a process of evolution, or of dissolution? Does it build up or destroy? Is it an intrinsic evil, or is it a thing to be welcomed and encouraged within certain limitations, in the hope that it will enable nature to fight and to overcome disease, or does it make the last state of the body worse than the first? The only rational answers that can be given to these questions are to be obtained through a study of the production of the normal temperature of the body, and this leads to the following questions: Where and how is the normal heat of the body generated and maintained? Physiology teaches that the heat of the body is almost exclusively produced through

oxidation, and that four-fifths of this amount comes from oxidation occurring in the muscles.

While heat is thus being generated there is going on at the same time another process in active operation, *viz.*, that of heat-loss, or heat-dissipation. This serves as a heat-leveller and tends to limit heat-accumulation in the body. From an estimation of these two factors it is evident that the degree of temperature present in the body at any moment is dependent on how much is produced and how much is lost at that time, or, in other words, the degree of body temperature at any time is the balance between heat-production and heat-dissipation.

It is evident, therefore, that in order to preserve heat equilibrium in the body neither heat-production nor heat-loss must go on without restraint, and that both must be adjusted in such a way that no more heat is produced or lost than is necessary to carry out this purpose. This function is supplied by a special nervous apparatus known as the thermotaxic mechanism, which regulates both heat-production and heat-dissipation. When the function of this mechanism becomes deranged, or is overthrown, either heat-production or heat-dissipation, or both, become deranged and fever or hyperpyrexia, or in some instances a subnormal temperature, supervenes.

It has been well established by Ott, Aronsohn and Sachs, Eulenberg and Landois that the thermotaxic mechanism exercises its function through a number of heat centers, which are located in the cortex, base of the brain and cord; for the above-named experimenters have shown that electric or mechanic irritation of these centers produces a rise of body temperature which lasts for hours.

From the above general remarks on the physiology of pyrexia it is clear that the neurotic nature of phthisis explains much that otherwise remains obscure in the fever of this disease. It makes clear why fever is in many instances

a precursor, or at least a premonitory symptom, of this disease, as has been shown by Sydney Ringer*; why it is its almost constant concomitant, and why, as is frequently the case, after a long period of absence during convalescence or in a state of recovery it is suddenly recalled by some indiscretion performed, or worry, or grief undergone by the patient at the time.

On the other hand, there is good reason for believing that in cases of phthisis where there is rapid destruction of lung tissue the fever is due directly to the absorption of septic material, but this is hardly true of the slowly progressive cases.

Loss of Weight. Loss of flesh is one of the earliest and most constant symptoms of pulmonary consumption. A great deal of this is undoubtedly due to an impaired state of the appetite, yet this fails to account wholly for the depreciation in this direction. There are many cases of incipient phthisis that show no perceptible morbid changes in the lungs, and apparently have no symptoms of dyspepsia, yet they undergo a gradual loss in weight and strength; while there are others which, during the course of their disease, retain a fairly good appetite and yet become flabby and emaciate.

It is quite probable that defective nerve tone has some share in bringing about the loss of tissue in this disease, for in spite of the fact that no distinct trophic nerves have as yet been made out it is quite clear from the many experimental and clinical data which the literature of medicine affords, that the nutrition of that part of the body suffers when its supplying nerve is divided or diseased.

Among the most notable experimental instances of this kind is the division of the tri-facial nerve. Section of this nerve within the cranium is followed by loss of sensation

* "On the Temperature of the Body as a Means of Diagnosis in Phthisis and Tuberculosis," 1865.

of that part of the face to which its fibres are distributed, the cornea becomes cloudy, the nasal chambers are inflamed and ulcers appear on the lips and gums. Durdufi in his "Experimental Study on Trophic Nerves"* demonstrates that unilateral division of the vagi and sympathetic nerves in dogs four months old leads to cloudiness of the cornea on the same side in eight days, and of the opposite cornea in fourteen days. In all the dogs he experimented on, about 30 in number, he found intense engorgement of the lung on the side in which the vagus was divided, and he lays special stress on the fact that in none of the vagotomized animals was there found the least amount of food or of other foreign matter in the respiratory passages. He believes that both the vagi and sympathetic nerves wield a trophic influence, and attributes the changes in the cornea to section of the sympathetic and those in the lungs to vagotomy.

Clinical evidence is also conclusive on this point. For example, it is well known that in neuralgia, the limb, or area of tissue to which the affected nerve is distributed, emaciates and loses its fat. Paget† relates a case in which ulceration of the thumb and middle finger was produced by compression of the median nerve by callus thrown out to repair a fracture. Packard‡ reports an instance in which choreic movements, nervous irritability and general loss of flesh and strength were entirely relieved by exsection of a nerve filament of the thumb, which had been injured by the impaction of a splinter of wood. Sloughing of the sacrum and heel frequently follows injury to the spine. Clouston§ states that thinness is the almost constant accompaniment of melancholia, and that fattening of the patient is its natural cure,

* *Centralblatt f. allgemeine Path. u. patholog. Anatomie*, V, No. 12. *Centralblatt f. d. med. Wis.*, 1895, p. 290.

† Lectures on "Nutrition."

‡ *American Jour. of the Medical Sci.*, April, 1870.

§ "Mental Diseases," p. 469.

and he furthermore says that this depraved or weakened trophic energy speedily tends to end in pulmonary phthisis. It seems, therefore, that phthisis and melancholia run a parallel course in this respect; emaciation is their common enemy, while fat redeems them both. Evidence of this character shows that a causative relationship exists between disease of the nervous system and disorders of nutrition, and when this is taken in connection with the fact that, as has been shown, serious structural and functional disturbances of the nervous system exist in pulmonary phthisis it is quite probable that the emaciation which occurs in this disease is largely traceable to defective nerve influence.

Hoarseness and Aphonia. Hoarseness is sometimes a premonitory symptom of pulmonary consumption, and frequently accompanies this disease throughout its course and before its fatal termination is displaced by aphonia. It appears that in many of these instances, and especially in the female sex, both the hoarseness and aphonia are of an ephemeral and, therefore, of a functional character. The author has on a number of occasions observed hoarseness amounting to partial aphonia, dependent on paralysis of one or of both vocal cords, which was accompanied simultaneously by pulmonary phthisis in the incipient period and which was entirely relieved by medication addressed solely to the nervous system. This indicates that at least in some cases both of these symptoms are dependent on impairment of the vocal nerve supply and in all probability on defective pneumogastric innervation. Sir Morell Mackenzie,* in discussing aphonia in its relation to pulmonary consumption, says that debility and hysteria are undoubtedly the most frequent causes of aphonia and that the latter, which is common in the second and third stages of phthisis, is often erroneously ascribed to structural changes in the larynx. In thirty-seven cases

* "Use of the Laryngoscope in Diseases of the Throat," second edition, p. 185; 1869.

of phthisis, in which the voice was affected, he found that in eleven the affection was purely functional, in twelve there was thickening of the mucous membrane and in fourteen there was congestion. He believes that in the cases of functional aphonia the nerve force is feebly or imperfectly evolved, or it is not directed in the proper channel. Reference has already been made to the frequent association of laryngeal disorders with nerve disease, and especially with locomotor ataxia (p. 128), and a few abstracts of interesting cases will be quoted herewith which show a similar intimacy between the former and pulmonary derangement.

Case 212. Reported by Dr. Lyon* to the Hunterian Society, which had fibrosis of the left lung with paralysis of the left vocal cord, and with contraction of left pupil.

Case 213. Bäumler† describes the case of a male, 61 years old, who, for four months previous to his admission, had been suffering from hoarseness and dyspnœa. It was found that he had consolidation of both apices and pleurisy. A laryngoscopic examination showed a normal mucous membrane and almost complete immobility of the left vocal cord. Death in a week. Section showed induration of both apices, peribronchial nodules in lower lobes and bronchiectasy in same area. The left recurrent laryngeal nerve was found overgrown by an enlarged bronchial gland.

Case 214. Krauss‡ gives the history of a male, aged 45, syphilitic, who became affected with neuralgic pains at the age of 35, which were followed by ataxia and difficult respiration, the latter being due to crico-arytænoid paralysis. Death. Section: Slight degeneration of sciatic nerve and marked degeneration in vagi and in recurrent laryngeal nerves.

COUGH. This is a reflex symptom. It depends on irrita-

* *Lancet*, April 24, 1897.
† *Deutsches Archiv f. klin. Med.*, Bd. 37, p. 231.
‡ *Centralblatt f. Nervenheilkunde*, Bd. 9, p. 715.

tion of the sensory fibres of the pneumogastric nerves, the impulse from which, being transmitted to the ganglia or to the root of the latter, is referred back to the lungs through the motor filaments of the same nerve and there manifests itself in the complex phenomenon of expulsive contraction, which we know as cough. The cough of pulmonary phthisis varies with the stage of the disease. In its incipiency the cough may be trivial and, indeed, be absent in some cases, but in others it is one of the earliest and most constant symptoms, although there may be very little or no expectoration. In the advanced stage of the disease cough is more or less persistent, and so long as the catarrhal element predominates it is accompanied by an abundant yellow expectoration. After cavities are formed the expectoration is generally of a grayish color and of tough, fibrous consistency.

Cough being a reflex nervous movement it necessarily follows, from a fundamental physiologic law, that the degree and amount of cough in any case correspond with the irritability or impressibility of the nerve-supply of the lungs. This applies more forcibly, perhaps, to the cough of phthisis than to that of any other form of lung disease and explains why the cough of this affection not only varies very much in different individuals, but why it varies in the same individual under different bodily positions and conditions. In the very nervous phthisical individual the cough may be one of the most prominent and distressing symptoms from the beginning to the termination of the disease, while in the insane, for instance, in whom the sensibility of the nervous system is in a great measure subdued, phthisis goes through its various stages without much or any cough, and often very little expectoration. Then, too, phthisical patients cough more when they are tired than when they are fully rested; and again, as a rule, their cough is easier when they sit or stand than when they lie down. The latter event is probably due to the fact that the lying position allows more blood to

gravitate to the apices, which are oftenest involved, and the increased fulness of blood thus induces greater irritability in the nerve filaments of these parts. The same reason also explains, perhaps, why these patients generally lie much easier on the healthy side of the chest than on the side which is affected. Moreover, sleep is an obtunder of nervous irritability, hence the cough of the phthisical, after they have once fallen asleep, is comparatively quiet during the night, although it is always worse in the morning on account of an accumulation of material in the lungs during the sleeping hours.

It is well known that cough may be excited by pressure of aneurism, or other tumors on the vagus. Occasionally the bronchial or cervical glands become enlarged in consequence of measles, diphtheria, whooping-cough, bronchitis, etc., and compress the vagi and engender a cough of a very distressing character. A case of this kind is reported on page 46. Sometimes the pleural membrane thickens in the apex, impinges on the vagus and causes a similar cough.

Dyspnœa. In considering dyspnœa as a symptom no reference is made to that form which accompanies a genuine attack of asthma, nor to what is known as suffocative catarrh, but to that form which manifests itself as an oppression of, or tightness, in the chest, and which is probably as much a neurotic condition as a genuine paroxysm of asthma. According to the author's observation this symptom presents itself as an exhausted state of respiration and prevails chiefly in the alcoholic and fibroid varieties of phthisis, and in what is known as miner's asthma. It differs from true asthma in that it is greatly aggravated by physical exercise, and sometimes very much relieved by rest. The following cases illustrate the close relationship that exists between a defective nerve supply and dyspnœa:

Case 215. Wallis* reports the history of a male patient,

* *Hygeia*, xlvi, p. 545.

aged 33, who for a number of years had been suffering with cough and hoarseness, was taken ill with pleurisy and bronchitis, associated with fever. After the pleurisy disappeared he continued to cough and become short of breath, which grew into a strongly marked dyspnœa. There were dulness and the absence of respiratory sounds in the anterior portion of the left lung. The dyspnœa increased, œdema set in, and he died. Section: Neuromatous swellings of the superficial nerves of the arms and legs, as well as of the deep-seated nerves. Both vagi were hypertrophied. These nerves in the region of the thyroid gland were as large as an ordinary sciatic nerve, and at intervals had nodular swellings on them. The right lung was œdematous and partly consolidated, while the left was dry and had undergone fibroid degeneration.

Case 216. Andral* observed persistent dyspnœa in a patient without perceptible cause. Section showed that both vagi were surrounded and compressed by enlarged lymphatic glands.

Hemoptysis. Hemoptysis is such a constant concomitant of the disease under consideration that it will not be regarded as a mere symptom, and will be discussed as a separate disease in succeeding pages.†

Nervousness. In a large number of phthisical patients nervousness constitutes an early and an important symptom. It is usually associated with tiredness, and sometimes with persistent sleeplessness. All degrees of nervousness, from slight excitability to twitching of the extremities, are present. Out of 1,000 consumptives Dr. Edward Smith‡ found that 71 per cent., or nearly three-fourths, were of a nervous or super-sensitive temperament. An examination of the nervous phenomena of fifty-one cases of pulmonary tuberculosis

* Cited by Longet, *Supra*, p. 312.

† See Chapter XIX., p. 391.

‡ "Consumption: Its Early and Remediable Stages," Philadelphia, 1865, p. 44.

(37 males and 14 females) Dr. Weil* found marked nervous trouble in 37 per cent. of the males and 42 per cent. of the females. The predominating complication, which was met in all cases, was muscular hyperæsthesia; in some cases this change passed unnoticed until the muscles were pressed or pinched, and in others it was so severe as to prevent movement. In most instances it was unilateral, and by preference affected the muscles of the trunk, arm, hip-joint and neck, and in these muscles faradic excitability was increased. This muscular hyperæsthesia was generally accompanied by hyperæsthesia of deeper tissues, the bones, joints and tendons. Cutaneous sensory disorders were not so common. The pharyngeal reflexes were absent nine times. Concentric narrowing of the field of vision was noticed in eleven out of seventeen cases—always seeming to follow the development of cutaneous sensory disorders, and by preference appearing on the same side as the cutaneous anomaly. The tendon reflexes were not considerably altered. Spontaneous pains were not infrequent, such as neuralgia, sciatica, and apical pulmonary pains, with which a deep-seated hemi-hyperæsthesia generally existed. Motor troubles occurred only in two cases; in one there was paresis of the arm and leg of one side, accompanied by convulsive movements and deep-seated hemi-hyperæsthesia; in the other paralysis of the left upper extremity.

He concludes that four out of every ten tubercular subjects are thus affected; that the symptoms usually appear during the early months of the evolution of tubercle, and run a parallel course; that they have no connection with hysteria, and that they arise probably from a centripetal irritation, of which the point of departure is in the sensory nerves of the bronchi and lungs.

Thoracic Pain. Pain and tenderness in the chest-wall

* "Nervous Complications in Tuberculosis," *Revue de Médecin*, Paris, June, 1893. *Medical Chronicle*, September, 1893, p. 405.

are also common symptoms of pulmonary consumption. Patients of this kind frequently aver that they feel weaker and more tender on one side than on the other, and on investigation it is often, though not invariably, found that the tenderness is situated on the same side as the affected lung. More than this, it often happens that the whole half of the head, or neck, chest and abdomen, on the side of the affected lung, is more or less hyperæsthetic, or comprises painful spots, while the other half is comparatively normal. Consumptive patients, especially of the female sex, are apt to have irritable or sensitive spots along the thoracic portion of the spine.

Loss of Appetite. Failure to eat is one of the most annoying symptoms of pulmonary consumption inasmuch as it seriously interferes with the efforts of feeding and sustaining the patient. It has been attributed to various causes, chiefly among which are defective gastric secretion and motion, but in all probability the real difficulty lies deeper and is closely dependent on the integrity of the abdominal nerve supply.

This statement is confirmed by the following facts: The stomach, intestines and all the important organs of digestion are innervated by the same nerve that supplies the respiratory organs; section or irritation of the vagi produces pulmonary deterioration, as has been previously shown; and, according to Bernard,* causes the walls of the stomach to dilate and the gastric mucous membrane to become pale and flaccid, arrests the secretion of gastric juice, deranges the secretory function of the liver and paralyzes the small intestine. According to Leubuscher† and Schäfer, Pawlow and Schumova, and Kiehl section of both vagi below the recurrent laryngeal nerves produces loss of appetite, wasting and death in animals. No free hydrochloric acid was found in

* Flint's "Text-Book of Physiology," p. 252.

† "The Vagus and the Secretion of acid by the Stomach," *British Med. Jour.*, Epitome, Sept. 15, 1894.

the stomach of such animals, and the gastric juice possessed little digestive action. Clinical instances are not wanting to show that vagus disorder is the cause of a depraved appetite, as is attested by the following cases:

Case 217. Longet* contributes an observation of Johnson, where, in a case of softening of the oblongata and compression of the root of the left vagus by an aneurismal enlargement of the left vertebral artery, complete loss of hunger and thirst occurred.

Case 218. Brignardi† found in the obduction of a woman, who died of phthisis and who had a voracious appetite during the latter part of her life, that both vagi were affected with red neuromatous swellings.

Case 219. Swan‡ saw a patient who, in spite of perceptible integrity of the lungs, suffered from dyspnœa. Her appetite became voracious without being fully able to satisfy it; she vomited undigested food which had been eaten several hours before, and gradually emaciated and died. At the post-mortem examination it was found that both vagi had atrophied.

Furthermore, it may be said that division of the vagi in animals frequently produces an unnatural appetite or abolishes the power of eating altogether. It is possible, too, that disorder of the higher nerve centers may disturb the gastric function, for Ferrier located a hunger-sense center in the inferior surface of the brain, and disease of the latter may lie at the foundation of that condition which is known as anorexia nervosa, anorexia gravis, anorexia mentalis, etc. Soltman§ records a case of cerebral anorexia which was entirely relieved by the application of faradism to the head—one pole to the neck and the other to the temple.

* "Anatomie und Physiologie des Nervensystem," Bd. 2, S. 313.

† Longet, *Supra*, Bd. 2, S. 313.

‡ "Treatise on Diseases and Injuries of Nerves," London, 1834, p. 170.

§ *Centralblatt f. d. med. Wis.*, 1895, p. 8.

In any event it is quite clear that disease of the nervous system and loss of appetite are closely allied, and that, therefore, the dyspepsia of pulmonary consumption may, in common with many other symptoms, be regarded as one of the sign-posts which discloses the state of the nervous system. It is a well-recognized clinical fact that the prospects of a phthisical patient, who retains or recovers his appetite, are infinitely brighter than they are under opposite circumstances. This is not alone due to the fact that he eats and assimilates more, but because his innervation is in a fairly good condition.

VOMITING. Vomiting in pulmonary consumption is generally attributed to violent coughing, and is hence regarded as a reflex act. It is undoubtedly true that vomiting is called forth by cough, yet it does not necessarily follow that the latter is the ultimate cause of the former. It is quite probable, however, that the real cause of the difficulty lies in the nerve supply of the lungs and the stomach. The respiratory and the vomiting centers have close anatomical relations in the oblongata,* and if it is true that the respiratory nerves are impaired in the disease under consideration it is natural that the irritability of one center should readily extend to the other, and common disorder follow.

Vomiting may, therefore, be regarded as a reflex index of the irritability of the respiratory and vomiting centers in pulmonary consumption.

DIARRHŒA. Diarrhœa in phthisis is always an unwelcome feature, and when it assumes a form more or less chronic it becomes an inveterate and implacable enemy. Neither is it a rare complication, for it seems that the great majority of consumptives that come under running observation display a tendency towards diarrhœa at one time or another during their illness. Louis† states that out of one hundred and

* Foster's "Text-Book of Physiology," p. 237.

† "On Phthisis," p. 195.

twelve cases which came under his observation only five were free from diarrhœa.

The morbid condition of the bowels varies in different stages of the affection. It may be said, however, that when the diarrhœa is of long duration ulceration of the intestinal tract is almost uniformly present. In cases of more recent standing the mucous membrane is thick, soft and reddened, and in some there is scarcely any appreciable morbid alteration. Louis observes that diarrhœa is less copious in individuals in whom ulceration alone existed than when softening was present; and he expresses* the belief that probably the marked intestinal lesions originated towards the close of life "and that previously to this period the diarrhœa resulted from a simple alteration of secretion."

As a rule the stools are copious, offensive and serous, and of a yellowish color and free from blood or mucus. In extensive ulceration the bowels may move from ten to fifteen times a day, and when the rectum becomes involved, as it does in the final stage of the disease, tenesmus and blood accompany the passages.

It is of great interest to notice in this connection that the chronic insane are subject to a diarrhœa very similar to that which affects the phthisical. In a very instructive contribution to this topic Dr. Cowen† states that two varieties of diarrhœa occur among the insane—the more serious one being an advanced stage of the middle variety. First there is a very watery alvine and offensive flux, very frequently repeated, without passage of blood or an excess of mucus, and not often fatal; and, second, a frequent diarrhœa, often accompanied by tenesmus—the stools often containing blood and much mucus. This is usually fatal. In 1894 thirty

* *Op. cit.*, p. 197.

† "Trophic Intestinal Affections in the Insane." Thomas Philip Cowen, M.D., B.S., London, Assistant Medical Officer, County Asylum, Prestwich, Manchester. *Lancet*, March 16, 1895, p. 669.

cases of this kind proved fatal in Prestwich Asylum. Section shows an inflammatory affection of the ileum or colon, and often marked ulceration of the mucous membrane. The disorder shows itself chiefly in those afflicted with general paralysis, stuporous melancholia or dementia of an incurable type.

Dr. Cowen expresses the belief "that these intestinal lesions form a part of the general degenerative process—and that they owe their origin to a nervous perversion. The reasons are: (1) The rarity of such lesions in the sane; (2) their comparative frequency in the insane; (3) negative evidence as to causation; (4) their association with other trophic lesions, and (5) their association with disease of the central nervous system."

This author furthermore says: "It is not at all unusual in the degenerate insane, especially in general paralysis of the insane, for trophic lesions to occur, *e. g.*, atrophy of skin, muscles and bones, acute sloughing of tissue, as seen in acute bedsores and herpetic and bulbous eruptions, and a peculiar low form of pneumonia, which is probably of nervous origin. Some of these lesions occur in the same patients in whom the intestinal affections arise later. In fact one or the other of these tropho-neuroses is always present. Dr. Hale White describes a case of intense colitis associated with double descending lateral sclerosis. Dr. Ackland raises the question whether in diseases of the spinal cord we may not get ulceration of the intestine comparable to other trophic lesions, as acute bedsores. He records two cases of disease of the spinal cord, in both of which small ulcers were found in various parts of the bowel. Curiously enough a short time ago a similar case arose in this asylum. A localized phlegmonous inflammation of the large intestine occurred in association with a transverse myelitis in the dorsal region. In this case there was no local cause for the affection of the gut, and the only explanation valid was that the lesion was a trophic one.

"The causation of these two varieties of diarrhœa may be explained as follows: (1) The watery alvine flux frequently seen in general paralytics is due apparently to centric irritation of the vagus nerve. Buzzard regards a similar condition met with in certain cases of tabes as dependent on irritation of the vagal nucleus in the oblongata. In these cases the flux is probably the result of paralysis of the splanchnics—the vasomotor nerves of the intestines—and to the resulting transudation of fluid from the blood-vessels into the bowel; (2) the ulceration is probably due to the same influence, *i. e.*, to a further extension of irritation of the nuclei in the oblongata."

From the facts set forth above it appears that a striking resemblance obtains between the diarrhœa of the insane and that of the phthisical, in the following respects: (1) The stools are copious, watery and offensive; (2) early in the affection they are free from blood and mucus; (3) late they are bloody and are accompanied by tenesmus; (4) early the mucous membrane is congested and inflamed, and (5) late there is ulceration of the mucous membrane.

Now, when the marked analogy between these two symptoms is considered in connection with the fact that in the one case the diarrhœa is certainly brought on by disordered innervation of the splanchnic distribution of the vagus, it greatly strengthens the belief that the diarrhœa of consumption is likewise, in great part, due to a weakened state of the same set of nerves.

SWEATING. This is one of the common symptoms of pulmonary consumption. In some cases it is more marked than in others, but in the majority of instances it is constant throughout the disease until the impending termination of the latter, when it becomes aggravated. It is sometimes regarded as a dependency of fever; and there is undoubtedly some connection between these two symptoms, but not as cause and

effect. There is proof sufficient that perspiration can take place without the presence of fever. This is shown by the copious sweating which accompanies the death agony, by the sweating which occurs during and for some time after the crisis of pneumonia, and of other diseases, and by the profuse perspiration which follows the anguish of mental torture; in all of which there is an absence of fever.

Now we have the most direct experimental evidence* to show that stimulation of certain nerves calls the sudoriparous glands into action independent of any vascular widening, or in the absence of fever. Thus, in the dog and cat, increased sweating takes place when the sciatic nerve is stimulated, even though the aorta is divided or ligatured. These nerve-fibres are probably co-ordinated by a center or centers, located in the spinal cord, and irritation of these centers or nerves excites the function of the sweat-glands.

When the neurotic nature of phthisis is considered in connection with the experimental fact that the secretion of sweat is under the domination of the nervous system, it seems very probable that the latter is involved in producing the exhaustive sweats of the disease under consideration.

Œdema. Œdema of the extremities shows itself to its fullest extent towards the end of consumption, and is generally regarded as the sequence of cardiac or constitutional exhaustion. It appears probable, however, that in many cases at least the œdema is only the final culmination of a process which had been less obviously active throughout the whole course of the disease, and that defective innervation is in part responsible for its development.

Dropsy is an accumulation of serous fluid in the lymph-spaces of the body. In health these spaces are fed with lymph derived from the capillary arteries, which is absorbed again by the venous radicles and lymphatics. Either the lymph-

* Foster's *op. cit.*, p. 315.

atics or venous radicles, according to Brunton,* are capable of carrying away all the lymph that is poured into the lymph-spaces by the arterioles, so that in case there is an obstruction in either of the former channels the other takes on a compensatory action and performs the work of both. Ranvier, Cohnheim and Brunton showed, however, that ligature of the vena cava alone does not always produce œdema in the lower limbs of the dog or cat, but the case is generally different when the sciatic nerve of one side is divided at the same time. Œdema usually follows in this, but not in the opposite leg, although the venous circulation is equally obstructed in both legs. Ranvier furthermore demonstrated that this œdema depends on paralysis of the vasomotor and not on that of the motor nerves, for by dividing the roots of the motor nerves of one limb in the spinal canal before they had joined the sympathetic branches, and the sciatic nerve—including the motor and vasomotor nerves—in the other, the limb in which the motor roots were alone divided became paralyzed, but did not swell while the other limb became very œdematous. From the results of these observations we must conclude that the vasomotor nervous system plays a most important part in the production of œdema, while at the same time it must not be overlooked that the state of the blood and the blood-vessels, the impaired sucking power of inspiration and of the cardiac diastole, which prevail more or less in pulmonary consumption, play a subsidiary rôle in bringing on this symptom in this disease.

Disorder of the Heart. Organic disease of the heart appears to be a rare complication of pulmonary consumption, and it is held by some very able authorities that if they co-exist the former rather conserves than aggravates the latter. There is no question, however, that the heart in phthisis varies from the normal state of that organ and that

* "On the Pathology of Dropsy," *London Practitioner*, vol. xxxi, p. 177.

its function is markedly compromised in this disease. Rokitansky* says that the heart of the phthisical is small, and that the structure of the arterial walls is frail.

Moreover, it has already been shown on page 463 that the heart of phthisis in regard to size corresponds with that of insanity. Not only is the heart small in phthisis, but sufferers from this disease are liable to irritability of the heart and of the circulation. Indeed a quick and an excitable pulse is frequently one of the early landmarks of phthisis, and it may be laid down as a rule that this disease never progresses favorably while the pulse-rate is persistently above a hundred. In Dr. Brehmer's† collection of 500 cases of phthisis 60 per cent. of them had an irritable pulse and cardiac palpitation.

* "Lehrbuch der pathologischen Anatomie," 1858, Bd. 1, p. 303.

† "Die Aetiologie der chronischen Lungenschwindsucht," von Dr Herrmann Brehmer, sen., Berlin, 1885.

CHAPTER XIV.

CLINICAL TYPES OF PULMONARY CONSUMPTION.

WHILE it is true that pulmonary consumption is a wasting disease of the whole body, in the pathological development of which the lungs are conspicuously concerned, it must be borne in mind, as has already been stated, that the morbid changes which the lungs undergo are by no means uniform and that at least three clinical types of the disease can be outlined. It must be understood, however, that these types are not always separated by hard and fast lines throughout, but that one may run into another and that two of them may exist side by side in the same lung. These clinical types of consumption are (1) acute tuberculosis, (2) chronic catarrhal phthisis, and (3) chronic tuberculosis or fibroid phthisis.

ACUTE TUBERCULOSIS. This type of the disease, also called galloping consumption, is very rapid in its course and in order to describe it fully a clinical example will here be introduced. A female, aged 25, whose father and mother were well, but who lost a sister from consumption two years previously, was first seen August 22, 1890. Three months before she spat some blood, and then coughed, had copious muco-purulent expectoration, fever, night-sweats, chills and a very poor appetite. There was some dulness in left apex, and subcrepitant râles were distributed in spots over the whole lung on the left side, while the right lung was practically free from adventitious sounds. Her morning temperature was 103° F., and her pulse 120. On September 8th her temperature rose to 104° F., and a fine rash, which lasted

for five days and which reminded one of belladonna poisoning, appeared over the whole lung and was especially well marked over the face. She menstruated at this time and her abdomen was very tender. She became progressively worse and died October 22d following. Post-mortem examination showed a diffuse eruption of miliary tubercles over the whole of left lung, especially marked in the upper half, and a bronchial catarrh of right lung.

Symptoms. As has already been stated tuberculosis is the result of an infective process, dependent on a toxine—the tubercle bacillus—which circulates either in the blood-vessels or in the lymphatics (generally in the former in acute tuberculosis) and which emanates from a caseous center. The symptoms of this type vary and are governed largely by the degree and intensity of infection which prevails in other organs besides the lungs. If, for example, the peritoneum or the serous coverings of the brain are much involved simultaneously the symptoms arising from this condition produce a clinical picture different from that which is presented by the pulmonary symptoms alone. As a rule it may be said that the lungs are the favorite seat of acute tuberculosis, and that its invasion is rather sudden and is accompanied by rigors, fever, headache and sweating. Cough is persistent from the beginning and is accompanied by copious expectoration, which is generally of a mucous consistency and light in color. Dyspnœa is generally very marked, the respiration frequently rising to fifty and seventy a minute. The pulse is rapid from the onset and the circulation is feeble throughout; the finger-nails, nose and lips have a cyanotic appearance. The fever is usually high, but may assume a remittent character. The sweating is very profuse during the remission of fever, but the skin may be moist in the presence of the latter. Hemoptysis is often wanting, but epistaxis is a frequent concomitant. There is total anorexia, sometimes vomiting and the tongue is dry

and coated, sordes collect on the teeth and diarrhœa follows. Delirium and picking at bed-clothes make their appearance early in the disease. When the brain-disturbance is marked an erythematous rash often shows itself on the body, the delirium is of a low, muttering character, liable to change into stupor and coma, and end possibly in convulsions. When the tubercular invasion affects the abdomen chiefly there are marked tympanites and diarrhœa, and the type presents a striking analogy to typhoid fever, with which it is doubtless at times confounded. The duration of acute pulmonary tuberculosis, which invariably terminates in death, depends on the extent and intensity of the infection. If this is very pronounced and general the patient may not live longer than two or three days. In the majority of instances it lasts from four to five weeks, and may endure for three or four months. The degree of fever is a reliable index of the degree and intensity of the infection.

Physical Signs. These do not always yield satisfactory results. A lung may be rather extensively infiltrated with tubercles without giving rise to the usual physical signs. The percussion-note resembles that of bronchitis more than that of pneumonia, and is frequently hyper-resonant or tympanitic. This is in part due to the emphysema which accompanies the process of acute tuberculization, and, at least in the upper anterior part of the lungs, to the large bronchial tubes which underlie the affected lung area.

The respiratory sounds are either rough or weak, and crepitant, and subcrepitant râles, mingled with sibilant and sonorous râles are generally present. Sometimes the latter are displaced by coarse, bubbling râles. Friction sounds are also commonly heard. A diagnostic point of some value is the disproportion that exists between the physical signs and the dyspnœa.

Cause. A caseous source of infection (see p. 280).

Chronic Catarrhal Phthisis. This is the most com-

mon type of phthisis, and is also known as caseous phthisis, catarrhal pneumonic phthisis, and chronic bronchopneumonia. It frequently follows an unresolved acute catarrhal pneumonia, bronchitis, whooping-cough, influenza, etc. It is essentially a catarrhal affection of the alveoli and its beginning, course and termination are well illustrated in the following example: Female, aged 30, single, and a teacher, was first seen August, 1894. Father and mother well. Patient is oldest of a family of five, and rest are all well. An uncle and a cousin on the mother's side died of consumption, an aunt and a cousin on the father's side have asthma. Patient resembles her father. At the time of her first visit she gave the following account of her symptoms: Had grip three years before and been sick ever since, but began to cough severely and expectorate copiously in April, 1893, when she had hemoptysis. Color of expectoration was yellow and green. Her appetite was poor, her bowels loose, and she menstruated regularly; slept poorly, tired easily and was very nervous, having had frequent attacks of hysteria. Had a great deal of dyspnœa and cardiac palpitation. There was œdema of lower extremities, but urine was normal. Pulse 106, respiration 18, and evening temperature 100° F. There was dulness in the right apex extending to third intercostal space and to middle of interscapular region. Amphoric breathing below clavicle, and some moist râles distributed over whole lung anteriorly and posteriorly. Left lung and heart normal.

This patient became worse and died in June, 1895. No post-mortem was made.

Symptoms. The disease is generally ushered in by a chill, which may be traced to a cold, although on inquiry it is found that the state of health had been below par for some time previously. Grip is also occasionally responsible for its beginning. The cough is marked from the outset and the expectoration is yellow at first, but later becomes greenish in

color. Tubercle bacilli are found in the sputum. Hemoptysis is frequent. Louis found it in 57 out of 87 cases. Dyspnœa appears on exertion, and the pulse ranges around 100. Moderate fever and night-sweats are present in the early history of the disease, but become pronounced towards the end. The appetite is poor and the bowels may be regular at first, but later show a tendency to diarrhœa. The menstrual function persists until the disease is well advanced. As the disease progresses the symptoms become aggravated. The cough is more troublesome, and often induces vomiting. The pulse becomes quick, the temperature rises, night-sweats are more profuse and hectic symptoms supervene.

Physical Signs. These are more decisive than those which are found in acute pulmonary tuberculosis. The disease usually begins in an apex, and the respiratory movements of the affected side are lessened. Vocal resonance and fremitus are increased, and impaired percussion resonance or dulness is generally present. Auscultation elicits some and perhaps most of the following-named signs: Prolonged expiration, bronchial breathing, crepitation, sibilant râles and an occasional click towards the end of inspiration. The respiratory sound is harsh, or diminished in volume. In the advanced stage of the disease large mucous râles, amphoric breathing, pectoriloquy and the cracked-pot sound show themselves and demonstrate that excavation has occurred or is going on.

Prognosis. This type of phthisis offers a hopeful outlook if the patient can be placed under suitable conditions for treatment provided the disease is limited to one apex, or to one lung, and if the constitutional disturbance has not been too extensive or too pronounced.

Fibroid Phthisis. This type is essentially a slow form of tuberculosis, and has also been described by various authors under the titles of chronic interstitial pneumonia, chronic pulmonary tuberculosis and pulmonary cirrhosis.

Like acute pulmonary tuberculosis it has its primary seat in a caseous center, but with the important difference, that in acute pulmonary tuberculosis the caseous center, as a rule, lies outside of the lungs, while in the fibroid type this is located within the lungs. It will be seen, therefore, that it is a secondary disease of an infectious character, and is dependent on the liquefaction of catarrhal material resulting from a chronic pneumonic process. The pathology of this procedure has been described already, and hence it is not necessary to say more on the subject except, in recapitulation, that the infectious material is derived either from catarrhal products which are undergoing degeneration in alveoli or bronchi in the course of chronic catarrhal phthisis, or from caseous masses which result from the obliteration of small pulmonary arteries during the course of the same disease; that the infectious material is absorbed chiefly by the lymphatics and not by the blood-vessels, and that the whole morbid procedure has a tendency to thicken and to shrink the interlobular and interlobar septa, and to produce chronic pleurisy, contraction of the lungs and to generate bronchiectasy.

Both acute pulmonary tuberculosis and fibroid phthisis being regarded as the product of infection, the interesting and important question arises here as to why one is an acute and the other a chronic process? Why in the one instance the infection is carried by the blood-vessels and induces a more or less widespread tuberculosis throughout the body, while in the other the infection is conveyed by the lymphatics and a local tuberculosis is the result? What are the conditions which determine the manner of absorption of the virus? And why is the latter placed out of the reach of one, and not of the other? Professor Hamilton* gives the following lucid and scientific interpretation of this phenomenon: "When the caseous softening occurs gradually there seems to be much

* *Op. cit.*, p. 199.

more liability to the débris being absorbed by the lymphatics than by the blood-vessels. There is usually a non-vascular area around a chronic caseous deposit, which apparently prevents the softened mass being removed by the blood-channels, while the lymphatic radicles are capable of taking up a small quantity of it. I have seen a lymphatic gland in an instance of general tuberculosis which had suddenly caseated and softened, and in which the caseous matter had been rapidly absorbed. The wall of the cavity which resulted was covered by a plexus of congested blood-vessels, and no doubt these had been the means of removing the contents of the cavity and of distributing them generally throughout the body. In a chronic softening there is not any such vascular plexus to be seen on the wall of the softening part, but a hard layer of caseous substance intervenes between the surrounding blood-vessels and the fluid contents at its center. This apparently prevents the blood-vessels taking up the débris in any quantity."

According to Dr. Hamilton, then, the degree of activity in the caseating process is the main determining factor in the production of acute and chronic pulmonary tuberculosis. If the process is active the pulmonary capillaries come in direct contact with the infecting virus, absorb it and produce general tuberculosis; while if the process is slow or gradual the virus is isolated from the blood-vessels, but is absorbed by the lymphatics and a local tuberculosis is brought about. The comparative slowness of the lymphatic circulation is another factor that tends to retard the process of infection when this is carried on by the lymphatics alone, and must be borne in mind when considering the conservative nature of pulmonary fibrosis.

The following is a typical example of pulmonary fibrosis: Male, aged 30, was admitted August 1, 1896. Had been coughing and expectorating for about four years, also had hemoptysis, night-sweats and dyspnœa, and suffered from

diarrhœa. On admission cough was very troublesome, and the expectoration was copious and of a yellow color. Appetite was variable, bowels regular and breathing very short. The morning and evening temperature was 98° and 100° F.. pulse 100 and respiration 40. There was marked depression in upper anterior part of left chest, and dulness over the whole of this side, especially in the lower half. There was amphoric breathing directly beow the clavicle. Death occurred on the 15th of the following month. Post-mortem examination showed marked emaciation of the body and the left side of the chest was very much contracted. The left lung had shrunk to one-third of its natural size, was adherent everywhere to the chest walls, and its pleura and interlobular septa contained whitish bands of fibrous tissue. There was an old, shrunken cavity in the upper part of this lung, which communicated with some bronchial tubes. The right lung was large, and its edges were emphysematous. It contained some cheesy masses, around which were located some recent miliary tubercles.

Symptoms. It comes on insidiously, with a slight cough and mucous or yellow expectoration, the beginning of which may be a slight bronchitis. The cough goes and comes and is usually aggravated in winter. The appetite may be fair, but emaciation occurs and the patient has a haggard look. Hemoptysis is rather the exception in the early part of this type, although it occurs later when excavation has taken place. There is but slight elevation of temperature. The shoulders become rounded, and there is retraction of the supra and infra-clavicular spaces, especially on that side of the chest that contains the affected lung. In the course of three or four years there is less tendency to improve during the summer season, the cough is worse, the fever is higher, although this does not rise so high as it does in the catarrhal or acute tubercular type, the expectoration is more profuse, renal trouble supervenes, dropsy of the extremities appears and the final result is impending.

Post-mortem appearances are marked emaciation of the whole body, retraction of the upper part of the chest. There is pronounced shrinking of the affected lung, although the latter is bound to the chest-wall by fibrous pleural adhesions, which are greatly thickened, especially in the apex. The depression above and below the clavicle is caused by the shrinking and traction of the lung substance on the upper part of the thorax. When the lung is removed and percussed some parts of it give rise to a hyper-resonant sound and others produce a dull note. Cavities, ranging in size from a hazelnut to an orange, are usually found in the apex. These cavities* are always bronchiectatic in character and contain cheesy catarrhal products. Tubercle nodules are found in the interlobular septa and in the visceral layer of the pleura.

Physical Signs are the same as those of chronic catarrhal phthisis.

Prognosis. The prognosis of fibroid phthisis, although chiefly a tubercular affection, is, on the whole, good. It is slow in its course and may last an ordinary life-time. Not every case terminates thus favorably. The extent of the pulmonary disintegration is an important factor in its prognosis. If this is limited to one apex, and not accompanied by a great deal of bronchiectasy, and the patient is placed under good hygienic and remedial management the prospects for recovery are very favorable.

Mason's or Grinder's Phthisis. This is another form of fibroid phthisis, which is caused by the continual inhalation of dust incidental to the occupation of stone-cutting, knife and fork grinding, clay working, etc., the pulmonary changes of which are similar to those found in the variety of fibroid degeneration just described. The lungs are deeply pigmented, and fibrosis is general. The pleuræ are thickened and nodules about the size of a millet or mustard seed are

* For an explanation of the formation of bronchiectatic cavities, see p. 275.

scattered through these structures as well as through the framework of the lungs. When these nodules in the lungs of stone-masons are examined microscopically they are, according to Dr. Hamilton,* found to consist chiefly of "dense, concentrically arranged bundles of cicatricial tissue, the particles of stone-dust lying in great numbers at the center of the tumors and in the plasmatic spaces between the bundles. The stone-dust particles are very minute, and are either round or angular. They have a clear center and, when seen in mass, a grayish color. They run in the course of the lymphatic vessels, and are occasionally accompanied by particles of carbon, which have also been inhaled. The cicatricial tissue is undoubtedly caused by the stone-dust irritating the fibrous stroma of the organ. Giant-cells are not found in these nodules, and the reason apparently is that fibrous hyperplasia takes place gradually, so that abundant time is afforded for the nuclei of the stroma being organized. They do not rush into an embryonic existence, as in the case of tubercle, where the irritant is very acute, but pass through the stages of round and spindle cells very slowly until the perfect fibres are produced. With this exception, however, the nodules in the stone-mason's lung are identical with tubercles."

Laryngeal Tuberculosis. This is another type of phthisis in which the laryngeal disturbance is the most prominent factor and, without exception almost, it is a condition which is secondary to grave changes in one or both lungs. The exceptions are, however, exceedingly rare, for Dr. Mackenzie states†: "I have only met with three cases in which, on post-mortem examination, laryngeal phthisis was present without any disease of the lungs. As an almost invariable rule cavities are found in the lungs, or at least breaking down of lung-tissue." Dr. C. Theodore Williams‡ voices the same opinion in the following statement: "I have never seen a

* *Op. cit.*, p. 202.

† Reynolds' "System of Medicine," vol. iii, p. 461.

‡ "Pulmonary Consumption," p. 281.

case which I could clearly identify as laryngeal phthisis, where the lungs were not involved, and generally to a large extent." Like every other tubercular structure, laryngeal tuberculosis is an infectious product and is in all probability dependent on caseation of the lungs for its source of contamination. Its favorite resorts are the arytenoids, the epiglottis, the ventricular bands and the posterior wall of the larynx, and it is primarily characterized by a smooth, pale, gray swelling, which terminates in ulceration.

It has been stated already that the line of demarcation which is here drawn between the three types of phthisis are more or less artificial, and that one may be translated into the other; and yet it has been observed that they have distinguishing characteristics which, for the purpose of facilitating a differential diagnosis, are arranged side by side in the following table:

Acute Tuberculosis.	Chronic Catarrhal Phthisis.	Fibroid Phthisis.
1. Onset, sudden.	Sudden.	Gradual and insidious.
2. Cough, persistent.	Less marked.	Moderate.
3. Expectoration, white, gray and copious.	Yellow.	Not very profuse.
4. Dyspnœa, very marked, with sudden onset (resp. 50 to 70).	Moderate.	May be marked, but its onset is gradual.
5. Chills, very marked.	Medium, may be absent.	Not very pronounced.
6. Fever, very high.	Moderate in the beginning.	Very little, if any.
7. Cyanosis, pronounced.	None.	None.
8. Hemoptysis, not frequent.	Frequent.	Not frequent.
9. Fibrosis in lungs, absent.	Absent in beginning: present later.	Always present.
10. Mental symptoms, delirium and coma.	Absent.	Absent.
11. Other organs involved, general involvement.	Other organs free in beginning.	Not generally involved.
12. Nature of process, infectious.	Non-infectious.	Infectious.
13. Cavities, very seldom.	Excavation in later stage.	Bronchiectasis.
14. Percussion and auscultation signs not satisfactory; more like those of bronchitis.	Satisfactory.	Satisfactory.
15. Contour of chest, unaltered.	Depression over affected area.	Marked retraction.
16. Pleurisy, frequent and extensive.	Not until late.	Prominent from beginning.

CHAPTER XV.

THERAPEUTICS OF PULMONARY CONSUMPTION.

RESTING on the proposition that pulmonary consumption is ultimately a disease of the nervous system, and especially of that part of it which supplies the lungs, the general principles of the treatment of this disease resolve themselves (1) into those therapeutic measures which increase the vital resistance of the general nervous system, and (2) into those which increase the vital resistance of the pulmonary nerve supply.

MEASURES WHICH INCREASE THE VITAL RESISTANCE OF THE GENERAL NERVOUS SYSTEM.

Rest. One of the principal and most important measures that can be employed in fortifying the vital resistance of the nervous system is rest of the body. It has been previously shown (p. 289) that exhaustion of nervous energy or an approach to physiologic bankruptcy is one of the earliest and most constant symptoms of this disease, and that this condition of the consumptive may well be compared to the situation which stares the individual in the face who is in danger of financial failure. For is it not true that both have spent more of their resources than they can afford to lose? Can they expect relief if they continue this career of extravagance? The answer is obvious, but what is the remedy in this emergency? In the case of the financier if he is prudent, and wishes to save himself from disaster, he will either increase his income or reduce his expenses, or do both. In this way he will in time recover sufficiently to be able to compete

with his more fortunate financial neighbors. The same principle applies to the consumptive. He must likewise improve his capital. He must increase his income, or at least diminish his bodily expenditures.

Now of what do the bodily expenditures of the consumptive consist? They consist of the amount of force which is consumed in innervation, respiration, circulation, digestion, secretion, maintenance of temperature and muscular motion. Making an approximate estimate of the total energy which is expended by the body it may be said that one-fifth is applied to mechanical or muscular labor and the remainder leaves in the form of heat. Of the one-fifth by far the greatest part is expended by voluntary muscular action. Thus the muscular energy which is necessary to carry on a day's labor is equivalent to about two hundred and fifty thousand kilogrammeters; which means the lifting of two hundred and fifty thousand kilogrammes to the height of one meter, or about two million pounds, which means the lifting of two million pounds one foot high. Besides this the heart expends about eighty-five thousand kilogrammeters, or about five hundred thousand foot-pounds in its own work during twenty-four hours; and, finally, not an inconsiderable amount is appropriated to the functions of respiration, digestion, etc.

Now if the total amount of energy in the consumptive is inadequate to carry on all the functions of the body properly it seems clear that by putting to rest those organs which are not directly indispensable to life, as the muscular system, for example, the amount of energy thus saved is appropriated to the maintenance of the more vital functions. At any rate practical experience proves that if the muscular system is kept quiet the heart's action is strengthened, the respiration becomes more full and is reduced in frequency, digestion is improved and the organs perform their work to better advantage.

How shall the rest treatment be applied, and how long shall it be continued? Shall the patient be confined to bed constantly, or remain up a part of the time, or all the time? These are questions concerning which no iron-clad rules can be laid down in their practical enforcement. They all hinge on the condition of the patient. His physical state and his power of resistance will indicate what course to pursue. If the disease in the lungs is trivial, if there is only slight constitutional disturbance, moderate fever, a tolerably good appetite and not too great a tendency to become tired, a greater power of resistance is present than if there exists widespread lung mischief, combined with high fever, great constitutional weakness, emaciation and a persistent sense of fatigue and exhaustion. A patient in the latter condition must be placed in complete repose on the back; while it is probable that one in the former condition may be treated successfully with a less rigid observance of the rest principle. It is best, however, if most consumptives are placed in bed at the very outset, and kept there, day and night, until they are able to sit up and walk without becoming fatigued, or provoke a rise of temperature by doing so. If the temperature is continually around 100° F., or is below this point during rest, and rises above it on exercising, or if a slight effort brings on a sense of fatigue, it is very important that absolute rest should be faithfully continued. A patient with a temperature remaining at or below 99° F., without a feeling of tiredness, may get along well without going to bed at all, provided the amount of physical exercise is well regulated. One thing is plain and that is that the rest-treatment must not be abandoned until the temperature remains below 100° F., and until there is a recovery of a measurable degree of strength and flesh. This usually requires from three weeks to two months and sometimes longer.

When a patient is able to sit up great care must be taken not to make the transition too suddenly. The sitting up

must at first be limited to twenty minutes or half an hour, or even a shorter time if it tires too much—once or twice a day. This period is steadily lengthened until he is able to sit up two hours in the forenoon and two in the afternoon. After he is able to do this he must be allowed to walk around the room, in the hall, perhaps down stairs, and out on the porch. He should then spend all his active hours, either sitting or walking, in the open outside air, provided the weather at all permits it. In cool or cold weather his body should be well protected from unfriendly draughts of air.

Exercise, when applied at the proper time, has a most valuable therapeutic influence, but when introduced prematurely it is a fallacy of the worst type. The value of exercise is often based on the assumption that because it gives strength in health it must have the same effect on the invalid. Nothing is farther from the truth than this, as can be illustrated by another example drawn from the field of finance. It goes without saying that money has the power of making money. The banker puts his money on interest, or, in other words, he exercises his capital, and by so doing he increases his financial strength; but the poor man has no money to put on interest, and he struggles along in poor financial health from year to year. This comparison holds good between the man in health and the consumptive. The former has a sufficient amount of reserve physiological capital which he can expend in physical exercise, and physiologic activity brings not only the interest which manifests itself in strength, but also that which builds muscular tissue, hence by doing this he enhances his normal resources; but the latter has no reserve capital whatever, and is, as has already been said, on the brink of physiologic dissolution. In his exhausted state he lives from hand to mouth, for he consumes all the force which he obtains from his food in carrying on the functions which are necessary for his bare existence. Exercise in his case is, therefore, meaningless in so far as get-

ting strength is concerned, and can have no other than a disastrous effect on his already drained and devitalized constitution.

Rest is also a valuable febrifuge. It is well known that in health muscular contraction develops heat, but in the febrile state it greatly aggravates the abnormal temperature. In the consumptive it is remarkable how readily the temperature flies up after a little exercise. On a number of occasions the author has observed that a brisk walk, running upstairs or the swinging of dumb-bells will raise the temperature of such patients from a degree to a degree and a half in the course of five or ten minutes, even though it has been near the normal point before. It seems as though the temperature-rise is one of the most sensitive symptoms of this disease, for in most instances it is the earliest evidence of the beginning of the latter and in many it is the last to entirely disappear.

Rest not only obviates the rise of fever in phthisis, but by quieting the muscular and nervous systems it tends to lower the same; and it is, therefore, such an important measure that it must never be lost sight of in the treatment of this disease. Its value as such is becoming thoroughly recognized by many physicians, and especially by those who are engaged in specially treating this disease in mountain resorts, as will be seen further on.

Rest restores the appetite. It is the prevailing opinion that a consumptive cannot eat so long as he is confined to bed, and in obedience to this influence many of these invalids actually rise early enough to take a brisk walk before breakfast in order to get up an appetite. The results are nearly always disappointing, for very frequently they return utterly fatigued and unable to eat at all. The reason for this is obvious: The little reserve energy which they had was wasted in the walk and when they come to the table there is not enough left to carry on the process of digestion, and the

consequence is a failure of the appetite. For such patients rest is most appropriate, and they are often astonished at the increase of the appetite so soon as they refrain from active exercise. The following case is a very fair illustration of this: A young man, 19 years of age, whose mother died of consumption, began to run down in weight and strength for three months before he was seen by the author. During this time he kept up his studies at school and did considerable exercise in the way of playing ball, walking, dancing, etc. When he came under treatment he was very weak, his respirations were 45 and he had a good deal of fever. There were impaired percussion resonance and a few sibilant râles heard in the right supra-clavicular fossa. His one great apprehension was that he would lose his appetite if he were compelled to lie down. In spite of his protests he was persuaded to go to bed. Up to this time he had steadily declined in every respect, but now he began to improve markedly in appetite, strength and weight, his cough, expectoration and fever diminished, his breathing became better, and in three weeks he had gained thirteen pounds. In the course of a few months he had made an uninterrupted recovery. He was confined to the bed in all about five weeks.

Rest quiets the heart and augments the power of the circulation. The heart of most consumptives is weak and irritable, and beats beyond its normal rate. Placing the body in the recumbent posture allays the irritability of this organ, reduces the number of its pulsations and enhances its power of contraction.

Besides all the direct and indirect benefits which have been shown to accrue from the application of rest in the treatment of pulmonary consumption, it also preserves patients of this class from committing hurtful acts of indiscretion. Its value in this respect is not easily overrated, for many consumptives sacrifice their lives on the altar of imprudence. There are few, if any, invalids who are more prone to com-

mit offences in this direction than consumptives. This is probably due to the fact that they are a hopeful class, and are, therefore, very much disposed to overestimate their strength and ability. The author has known undue and sudden strain, like that produced by shoveling snow, lifting heavy weights, running up stairs, racing after street-cars, or driving a span of spirited horses start serious hemoptysis or prostrate the patients to such a degree that they never recovered from it. Others have recorded cases in which excessive horse-back riding and mountain-climbing have led to similar consequences. It is clear, therefore, that a close supervision of the consumptive's physical movements will save him from many blunders which he would otherwise commit.

Food. The problem of alimentation in pulmonary consumption is a very important one. Nothing gives rise to greater anxiety than the persistent loss of appetite which is so often present in this disease, and nothing taxes the ingenuity of the practitioner more, and nothing demonstrates his ability better than his success in devising ways and means whereby his patients are led to partake sufficient nourishment. Anorexia is often such a prominent symptom that one might be led to suspect that the original seat of the disease has its location in the digestive organs.

Now the task of making a patient eat, and sometimes against his own will, though a very arduous one can usually be carried out successfully if sufficient tact is displayed by both practitioner and nurse. System, promptness and perseverance will accomplish much. A little food at regular intervals, and oft-repeated, will furnish a large amount of nourishment in twenty-four hours and will convince the patient that he has the power of ingesting a great deal more than he thought he had. The author has seen patients who, absolutely refusing all kinds of food, would by persuasion take half a glass of milk, either cold or hot, every hour and a half, which gave them over a quart and a half of milk in

the twenty-four hours, or would take two tablespoonfuls of freshly expressed* beef juice every four hours, which is equivalent to eating a pound of round-steak. A beginning of this kind will restore their confidence in their digestive capacity and so soon as this is effected they will begin to partake of other foods.

Another important feature in a consumptive's dietary is variety. In this particular more depends on a good cook than on the physician or nurse. She must be dextrous and be able to render the bill of fare as tempting and as enticing as possible. The patient must not know in advance what the latter has in store for him, so that he is not allowed sufficient time to manufacture excuses for not eating. He always eats best when he is surprised by some new and inviting dish; and he should also be permitted to eat anything for which he has a craving, unless it is known to be positively injurious.

In cases with cavities, and in others where great irritability of the respiratory tract exists, vomiting occurs frequently in the morning. This is generally due to the violent coughing which is necessary to expel the liquid accumulation of the previous night and may be overcome in most instances by asking the patient not to eat until the paroxysm of cough is over, or if it is necessary to eat, to partake only of milk, soup, milk-punch or liquid food, and these only in tablespoonful doses at short intervals.

Now what kind of foods ought such patients to have? Clearly those which contain the greatest amount of food-energy in the smallest bulk and which at the same time confer the greatest amount of good on the body with the least expenditure of digestive force. Foremost among these are freshly-expressed beef juice and milk of the former, two ounces given alternately with a glassful of fresh milk every

* So far as the author knows the most serviceable beef press in the market is the Osborne No. 1 or No. 2.

two hours. Then comes beef, broiled, roasted, rare or scraped; lamb or mutton, sweat-breads, fat bacon, smoked sausage, cheese, ham, poultry and game, eggs, raw or soft-boiled; soups—oyster, clam, turtle, barley, pea, bean, tomato, celery, etc.; butter, cream, oatmeal, wheaten grits, corn-meal mush, corn-bread, bread, biscuits, milk-punch, whisky, brandy, wine, malted milk, and malt liquors. On the other hand, many other less nutritious foods and condiments, such as fish—fresh, salted or smoked—asparagus, lettuce, celery, green peas, tomatoes, potatoes, coffee, tea, chocolate, oils, spices, pepper, salt, mustard, horseradish, vinegar, etc., must be admitted to the list; as desserts, ice cream, farina, sago, tapioca, apple or milk pudding, custards, baked or stewed apples, and cooked fruits with fresh cream are to be recommended. Meals should be served at regular intervals at least three times a day, and the food should be slowly and thoroughly chewed before it is swallowed. Dinner taken in the middle part of the day should be the heaviest meal, and the one at night should be the lightest.

Feeding through the rectum is a method which deserves considerate attention in the alimentation of this disease. In spite of the theoretical objections that have been urged against this way of administering food there is no question that practically the food which is placed in the lower bowel is digested and absorbed. It is a method which economizes the strength of the stomach and sometimes counteracts the tendency to diarrhœa that exists in many cases. Liquid food, like milk and fresh beef juice, about six ounces of the former and four of the latter, with ten grains of pepsin, is to be given with a hard-rubber syringe, morning and evening. The author has used, with great benefit, from two to four ounces of fresh beef blood given morning and evening in the same way, not, however, during the same period when the milk and beef juice were administered by the bowel. An egg beaten up with milk, with the addition of a little pepsin,

is also of value. Another serviceable plan is to place a teaspoonful of raw, scraped beef-pulp in a large, hollow suppository, with about five grains of pepsin, and introduce this into the rectum three or four times a day.

Fat-Producing Substances.

Fat is one of the most essential constituents of the human body. Its diminution is one of the earliest symptoms of pulmonary consumption, and hence the important question arises as to the source of this proximate principle. Is it derived from the outside as fat, or is it manufactured by the body from other food? There are two great classes of foods, the proteids or albuminoids, and the fatty and starchy foods, and there was a time when the animal body was likened to a steam engine, and it was believed that the proteids furnished the material for the structure of the machine, while the fats and starches were oxidized and gave the necessary force to keep the machine in motion. This view is, however, not altogether tenable at the present day.

The fat of the body is contained in cells, which are composed of protoplasm and possess nuclei. The cells abound in the interstices of loose connective tissue and are found under the skin, especially in the soles of the feet, the palms of the hands, buttocks, female mammary gland, around the synovial capsules of the joints, in the orbits, in the medullary canals of bones, in the surroundings of the kidneys and the omentum, and on the surface of the heart.

When an animal fattens it appears that oil globules are formed within the fat cells. The globules are formed by the cell itself, and at the expense of its own protoplasm, which becomes attenuated, and they increase in number and are deposited within the cells in a mechanical manner. It seems, therefore, that the fat of the body is as much a secre-

tion of the fat cells as pepsin is a secretion of the peptic glands, or as the oily matter of the skin is a secretion of the sebaceous glands or as the fat of milk is the product of the cells of the mammary gland.

From the fact that the protoplasm of the fat-cells undergoes metamorphosis when the oil globules form, it seems quite obvious that other food, beside that of a fatty nature, is used by the body in the manufacture of fat and that in all probability proteid food is used for this purpose. It was shown long ago by Liebig that fatty, starchy and saccharine foods do not form the exclusive supply of fat in the body; for the butter in cow's milk far exceeds the scanty supply of fat in her food, and the wax which is produced by the bees is out of all proportion to the amount of sugar which they consume in their food. The feeding experiments of Lawes* and Gilbert also demonstrate "that for every 100 parts of fat in the food of fattened pigs, 472 were stored up as fat," showing, therefore, that fatty foods only supply one-fourth of the fat which is contained in the body.

No effort is made here to convey the idea that albuminous foods supply the greatest part of the fat of the body, but it is desired to lay special emphasis on the facts that fats and oils do not play the important part as fat-builders which is ascribed to them, and that proteids are of greater value as fat-producers in pulmonary consumption than they are generally believed to be. In fact, evidence is not wanting to show that both fats and carbo-hydrates diminish the metabolism of the body, while a meat diet enhances the same, increases the oxidizing activity of the body, multiples the number of red blood-corpuscles and leads to a rapid consumption of fatty and carbo-hydrate food.

Another important question is as to the influence of rest and exercise on the fattening process in the consumptive. Is physical activity more conducive to fat-building than rest?

* Phil. Trans., 1860.

There is no doubt that in health exercise gives both fat and strength, but it seems to be quite different with the consumptive. The fat which is stored up in health represents so much surplus capital, which is laid up for a rainy day, but the consumptive has no or very little fat, and hence a very small amount of surplus capital. Now it seems quite clear that rest promotes the collection of fat. Swine and cattle, which are prepared for the butcher's knife, are not allowed to run loose, but are closely confined; and the geese of Strassburg, which fatten to enormous proportions in a few weeks, are shut up in tight boxes with just sufficient room to project their necks.

From these facts it is apparent that fats and carbo-hydrates furnish only a partial supply of fat in the body; that the balance is furnished by the proteids; that the invariable plying of consumptives with fats and oils in order to fatten them is erroneous, and that rest is probably more conducive to the fattening-process in the consumptive than exercise.

According to the author's experience consumptives may be divided into three classes so far as the remedial value of oils and fats are concerned. First, those with whom these agents disagree under all circumstances; second, those with whose digestive organs they agree, but fail to give any staying powers; and third, those with whose digestive organs they agree and whose power of resistance is increased and whose weight improves. Oil extracted from the liver of the codfish is generally used in the treatment of phthisis, although linseed oil and emulsified petroleum are also recommended. Cod-liver oil should be given in its pure state, for the probability is that if it fails to agree in this form it will be obnoxious to the stomach in any other. Whisky, brandy or malt liquors will sometimes aid its digestibility. Cod-liver oil is also frequently rubbed into the skin with great benefit, especially in children. Leaf lard, as an external application in this disease, has also been highly spoken

of. When cod-liver oil disagrees it is a good plan to resort to a pertoleum emulsion, which the author has found a valuable remedy.

Influence of Drug Treatment.

Drugs play a very conspicuous part in the successful treatment of pulmonary consumption; but it must be recognized that, like everything else, they have their limitations and shortcomings, and it is principally because insufficient weight is given to the latter factors that disappointment and loss of faith in their efficacy follows their employment. A grievous mistake is made, however, when it is concluded that because our prescriptions do not always yield the expected benefit, medication is useless and remedial efforts are necessarily futile. The truth is that we expect too much of drugs, and hope to accomplish with them what should be done by other means. We are too prone to forget that there are other things than drugs, food and fresh air, and that it is our duty to supervise the whole of the invalid life of those who suffer from this disease and to readjust their lives to a new environment.

Drugs and their application to this disease will be considered under the following heads: Tonics and stimulants, antipyretics, and antirheumatics.

Tonics and Stimulants. Foremost under this heading comes *strychnine*, but before discussing its therapeutic application it is necessary to refer briefly to its physiologic action. Without going into a preliminary explanation it may be said that all drugs stimulate in small and depress in large doses, and strychnine is no exception to this rule. It has an affinity for the nervous system, and, therefore, it stimulates, or depresses and paralyzes this part of the body, according to the size of the dose which is administered. When a small dose is injected the vigor and excitability of

the frog are increased, while a large dose produces tetanus and paralysis. That in large doses it engenders paralysis has been amply proven by the researches of Reichert, and often observed by the author in his own experimental work on this drug. Moreover, in the author's researches* on the influence of drugs on the frog's heart it is shown that strychnine adds force to the ventricular contraction over and above that which it derives from the blood circulating through it. Instead of abstracting force from a nerve, or of disintegrating the latter in small doses, as is believed by some, it augments nerve function and facilitates the transmission of normal impulses.

Figuratively speaking its action may be likened to the form of a pyramid, the ascending line of which represents its physiologic or healthful action, while the descending line represents its toxic or morbid action. Its physiologic, which is its stimulant action, is exhibited in increased vigor and strength while its toxic, which is its paralyzing action, is exhibited in tremor, tetanus and paralysis. By the stimulant-action of strychnine is meant an uplifting and a health-giving influence on the nervous system. It must be remembered in connection with this that health and disease are not separated by a hard and fast line, but that they shade off the one into the other. That which is health to-day may be disease to-morrow, and *vice versa.* The size of each one's territory depends on the degree of bodily resistance. If this is weak on the side of health, disease extends its area; if, on the other hand, it is strong, health will enlarge its domain and crowd out disease. Now let us say, for example, that the state of the nervous system is depressed to a point near the base of the cone, how will strychnine raise this to a point as high as is consistent with a full measure of health? In

* "The Action and Antagonism of Some Drugs on the Frog's Ventricle," by Thomas J. Mays, M.D., *Therapeutic Gazette*, Feb. 16, 1885, p. 73.

answering this it may be said that this drug in stimulant or tonic doses gradually and persistently forces the line of health upward and out of the realm of disease until the apex of the figure is reached. If this point is overstepped tetanus ensues. This constitutes the danger-level of its action, and should never be crossed, although it should be hugged as closely as is consistent with safety. This danger line varies in different individuals and is liable to shift its position in the same person if the drug is taken for a long period. The dose which calls it forth at one time will not bring it out later. A certan degree of strychnine-immunity is established in this way. A time comes, however, in the history of each individual to whom the drug is administered when this line not only refuses to be moved further up, but sinks somewhat toward the base. In other words, the system seems to be saturated with the drug, and very small doses will now readily exhibit the toxic line.

What, then, constitutes the proper dose of strychnine? From what has been said it is evident that this must be a graduated or a shifting one, since the impressibility of the nervous system varies from time to time. The initial dose should, therefore, be a comparatively small one, say $^1/_{32}$ or $^1/_{30}$ of a grain four times a day; this is given for one week, when it is increased to $^1/_{24}$ of a grain for another week; for the following week raise it to $^1/_{20}$ of a grain, and so on, making a slight increase every week until nervousness, restlessness or twitching—signs of the beginning of strychnine intoxication—are observed. When given in this manner these symptoms do not, as a rule, develop until $^1/_{12}$ or $^1/_8$ of a grain, or even a larger dose is administered four times a day. After the desired point has been reached the question arises whether it is better to go on with the largest dose or to go back and start with a smaller dose. On the whole it is good policy to reduce the dose somewhat at this point. If, for example, it is found that $^1/_8$ of a grain is a maximum

dose, it is well to go back to $^1/_{16}$ of a grain, gradually increase the dose again until $^1/_8$ of a grain is reached, and then again return to $^1/_{12}$ or $^1/_{16}$ of a grain. After this ground has been gone over several times in this see-saw fashion it will probably be found that $^1/_8$ of a grain produces dangerous symptoms no longer, and that as much as $^1/_6$ of a grain can be given. When administered in this way it may be given for a long period without detriment in the great majority of phthisical parents. This is shown in the tabular collection of ten cases (as given on the opposite page), in which the number of days the drug was taken, the amount, the largest dose four times daily, the average daily dose, the period during which the largest dose was taken, and the condition of the urine at the termination of the treatment are noted:

TABULAR VIEW SHOWING THE EFFECTS OF LARGE DOSES OF STRYCHNINE.

Patient.	No. of days taken.	Amount taken.—Grains.	Largest dose 4 times daily.—Grain.	Average daily dose.—Grain.	Period during which largest dose was taken.—Days.	Condition of urine at end of treatment.
W.	44	11¼	1-10	¼	30	No examination.
S.	661	181	⅛	¼	30	No examination.
S.	180	44	1-10	⅓+	30	No examination.
M.	227	70½	1-9	⅓	30	Normal.
H.	33	11⅓	1-15	⅓	30	Contained albumin at beginning, which diminished markedly.
N.	330	110⅙	1-7	⅓	60	Normal.
G.	369	172½	⅙	⅓	60	Normal two months after treatment was begun. Died unexpectedly.
L.	600	142	1-7	1-5	93	Normal.
B.	720	242	1-7	⅓	120	Normal.
J.	75	20	1-9	¼	19	On admission contained albumin; disappeared entirely during treatment.

Altogether the ten patients given in the above table took about two ounces of strychnine during the time they were under observation, and in doses ranging from $^1/_{12}$ to $^1/_{16}$ of a grain four times a day. One patient took $^1/_6$ of a grain uninterruptedly for two months, and another took $^1/_7$ of a

grain four times a day for 93 days, while another took the same dose with the same frequency for 120 days.

The remedial effects of strychnine on the various symptoms of phthisis often show themselves in many particulars. The nervousness, sleeplessness and pain in the chest are ameliorated and perhaps entirely suppressed, the cough, expectoration and dyspnœa diminish, vomiting improves and perhaps abates, the appetite becomes better, the patient frequently gains in flesh and in color, the heart-beats become stronger, and the whole outlook of the patient becomes more promising and hopeful.

Quinine is a valuable nerve tonic and is especially applicable in the treatment of phthisis. It aids in relieving the fatigue and exhaustion so common in this disease, and often improves the cough, expectoration and night-sweats. Besides it antagonizes the malarial element which is often present in these cases. As a tonic or stimulant it is to be given in one or two grain doses, and as an antiperiodic in six to ten grains, four times a day.

Cayenne pepper is one of our best diffusible stimulants, and is of special value in arousing and invigorating the debilitated phthisical constitution. It is most useful in quieting cough, and particularly in that of alcoholic phthisis when given in large doses. In ordinary cases the tincture is to be given in from five to fifteen drops, four times a day, while in alcoholic cases it must be given in doses from half to a teaspoonful every three hours. The powder, in doses from $^1/_8$ to 1 grain is also very serviceable.

The *hypophosphites* of lime, soda, and potash, introduced by Churchill, are favorite and well-deserved tonics in the treatment of phthisis, and are usually administered in the form of syrups. They are sometimes combined with the syrup of hydriodic acid and with advantage, according to the author's observation.

Digitalis and Strophanthus. There are very few cases of

phthisis in which the heart is not weak and does not need strengthening, and for which both these drugs are useful. The former is frequently given with good effect in the form of Heim's pill,* but is often combined with other drugs. It is preferably given in powder, in half-grain doses, three or four times a day. The tincture should be fresh and obtained from a reliable source, and be given in doses from five to ten drops, three times a day. Strophanthus is nearly always given in the form of tincture, and in some respects is superior to digitalis. It does not nauseate and, so far as the author knows, it controls the phthisical heart as well as digitalis. It should be given in doses from five to ten drops, four times a day.

Nitroglycerin has a very favorable influence on the fibroid form of phthisis, particularly when it is associated with a great deal of dyspnœa, as is nearly always the case. It seems to invigorate the respiratory capacity of the lungs, probably by stimulating the involuntary muscular fibres of these organs. It should be given for effect, *i. e.*, it should be pushed to the point where it produces flushing of the face. One or two minims of a one-per-cent. solution, given every three or four hours will usually reduce this effect.

Quebracho is also recommended as a remedy for dyspnœa. The author has used quebrachine hydrochlorate (Hesse—Merck) in doses of from a half to a grain and a half, every four hours, in difficult respiration and with good results.

Antipyretics.

Among the principal antipyretics that are used in the treatment of phthisis are *phenacetin, thermol, acetanilid, antipyrin* and *cold external applications. Phenacetin, thermol* and

* ℞ Quininæ Sulph., gr. 20; Pulv. Digital, gr. 10; Pulv. Ipecac, Pulv. Opii, aa, gr. 5. M. Ft. mas. div. in pil., No. 20. Sig. One pill three times a day.

acetanilid are closely allied in action and the author is accustomed to give two and sometimes all of them in combination, in from one and a half to two and three-grain doses of each, believing that thereby he gets a more concentrated febrifuge action and avoids the disagreeable effects which an equal dose of one of the three would produce. Besides their antipyretic properties these drugs appear to have a tonic effect on the whole constitution, for the author has often observed that in small doses they have a beneficial influence on patients who have no rise in temperature. *Antipyrin* is one of the most valuable and at the same time one of the most objectionable of these four coal-tar products in the management of phthisis. Besides reducing fever it occasionally exerts a most favorable influence on those cases of phthisis which are in the last stage and which are not relieved by ordinary therapeutic measures. The author has seen cough, rigor, fever and night-sweats improve very markedly, appetite return and breathing become more comfortable under the influence of seven and a half grains, given every four hours. Unfortunately, however, in the course of ten days or two weeks it produces an erythematous rash, which itches intensely and sometimes a cutaneous eruption of the most painful character and which becomes a greater torture than phthisis itself if the drug is not discontinued.

Cold is usually employed in the form of ice placed in large, flat, rubber bags and applied over the chest. As a rule it is not necessary to call this mode of treatment into requisition, except in cases of acute tuberculosis where the temperature is very high. Under these conditions as many bags as are needful to cover the affected part of the lungs should be appplied, and one or two should be used on the head at the same time. An ice-bag applied over an irritable heart, in ordinary cases of phthisis without much fever, has the power of greatly reducing the pulse-rate and of quieting cardiac excitability.

Antirheumatics.

From what has already been said it appears that rheumatism is liable to be associated with phthisis, and it is for this reason that antirheumatic remedies often have a favorable influence on the progress of the latter disease. For this purpose sodium and cinchonidia salicylate, lithium citrate, potassium carbonate, ammonium acetate and guaiacum may be employed. Experience teaches that better results are obtained in treating chronic rheumatic symptoms if these agents are combined in small doses than if they are given singly and in large doses. The following formula is one which is frequently prescribed by the author in this connection: ℞. Sodii salicyl., Potassæ carbonat., Lithiæ citrat., aa ℥i. Liq. Ammon acetat. Inf. Gent. Comp. aa. fl℥i. Elix. Lactopeptini. fl℥ii. M. Sig. One teaspoonful four times a day, in water. The following prescription has also been found useful, but on account of containing potassium iodide it cannot be given for as long a time as the former: ℞. Calcii chloridi (granular), Potassæ Iodidi, aa ℥ii. Sodæ Salicyl., ℥iiss. Liq. Potassæ arsen. ℥ss. Tinct. Gent. Comp., Elix. Lactopeptini, aa ℥iss. Syr. Sarsaparillæ, q.s. fl℥iv. M. Sig. One teaspoonful after meals, in water.

Iron is an important drug and should not be overlooked. The tincture is probably the preferable form.

Outline of Drug Treatment. The following outline may serve as a useful guide in the drug treatment of phthisis. For a patient suffering from the ordinary symptoms of this disease the following may be prescribed:

℞	Strychninæ Sulph	gr. i
	Quininæ Sulph	℥ iss
	Phenacetini, Thermolis aa	℥ i
	Pulv. Capsici, Pulv. Digitalis aa	gr. vi
	Atropinæ Sulph	gr. 1/24

M. Ft. Capsulas No. XXXII.
Sig.—One capsule four times a day.

In all cases the strychnine is increased as indicated above until the limit of its toleration is reached. Sometimes the strychnine, instead of being given in the capsule, is administered in the same manner with the syrups of the hypophosphites and hydriodic acid, half a teaspoonful of each, four times a day. If more than an ordinary degree of fever exists the phenacetin and thermol may be increased. If there is a tendency to diarrhœa, add five grains of powdered opium. In the presence of a syphilitic history combine half a grain of the corrosive chloride of mercury with the other ingredients. In case of a rheumatic complication administer the antirheumatic agents, as prescribed above, at the same time. In addition give the following with each meal, unless acids are actually contraindicated:

℞ Acid Phosphoric (dil.).....
Acid Nitro-Muriatic (dil.).....
Acid Sulphuric Aromat.....
Tinct. Ferri Chloridi..... .....aa ℥ ss.
M. Sig.—Thirty drops in half a glass of sweetened water with meals.

External Applications.

Various forms of external applications have been employed in the treatment of phthisis for the purpose of producing stimulation and counter-irritation. Among these are static electricity, iodine, mustard, blisters, etc.

Static Electricity. As a stimulant static electricity is certainly a most useful application. It has been used on a large scale by the author with a great deal of satisfaction. It gives tone to the whole body, improves the circulation, quiets the breathing and nervous irritability, secures sleep and in many cases it improves the appetite and increases the body-weight. A large static machine* is the most useful, although smaller appliances are also serviceable. One method of applying this

* The author employs a six-plate machine manufactured by the Galvano-Faradic Manufacturing Co., 300 Fourth Avenue, New York.

form of electricity is to place the patient on the insulated stool and to simply charge him with it for a period ranging from half an hour to an hour or even longer, once or twice a day. The positive breeze may be applied at the same time. This is done by the operator holding an electrode near enough to the patient's body without drawing a spark. A part of the electricity with which the patient's body is charged is drawn off through the operator's electrode, and this produces a peculiar breezy noise, which is both grateful and soothing. It may be applied to the head, spine, chest or any part of the body; or the breeze may be applied in the following manner: The patient's trunk is surrounded by, but nowhere in contact with, a galvanized iron wire-netting, suspended from the ceiling, while he is sittnng on the insulated stool. On the inside of the netting, and at intervals of about every six square inches are fastened small bundles of gold-foil, with very fine wire. As soon as the machine is put in motion a breezy noise is perceived, which is caused by the gold-foil and wire netting attracting the electricity from the patient's body. On the other hand, the patient sitting on the insulated stool, may be given the interrupted current by separating the two poles of the machine for half an inch or farther, if it is found that the jarring interruptions do not have any unpleasant or prejudicial effects.

A powerful current is not necessary to produce the greatest amount of good. According to the author's observation it is the mild stimulation of the static current of low power, continued for two or three hours every day, that is followed by the greatest benefit.

Mustard plasters and *tincture of iodine* are often usefully applied over painful areas of the chest. Equal parts of *menthol, chloral* and *camphor* have a similar effect. The application of *fly blisters* relieves pleuritic pain.

Respirators.

In cases of excessive cough and expectoration, especially if the latter is offensive, the wearing of an ori-nasal respirator, bearing on its cotton ten or fifteen drops of a medium-strong solution of ichthyol, ichthalbin, carbolic acid, thymol or eucalyptol, is a serviceable measure. Besides the good effects on the above-mentioned symptoms the cotton filters the air and prevents the inhalation of dust. The instrument should be worn more or less constantly throughout the day. The respirator of Genois is very effective and also reasonable in price.

Inhalation of Compressed and Rarefied Air.

The breathing of compressed or of rarefied air is the most powerful means at our command for increasing the chest-capacity. One of the best measures for the accomplishment of this purpose is the pneumatic cabinet, in which the air may be compressed or rarefied and also medicated with various substances, according to indications. Another very simple apparatus by which the air may be compressed and rarefied is found in a machine with two double cylinders, made according to the plan of Weil, of Berlin. It is actually a double Waldenburg apparatus, in which the cylinders are so adjusted to each other that one contains compressed and the other rarefied air. With this the patient alternately inhales compressed and exhales into rarefied air at the same sitting. When air is breathed in this manner there is felt during inspiration a gentle distention of the chest cavity, while during expiration a feeling of emptiness is experienced. The same sensations are felt when breathing in the pneumatic cabinet. Waldenburg states that in this way from fifty to two hundred and fifty cubic inches more air can be inspired than during ordinary respiration.

Pulmonary Gymnastics. Very few if any persons ever completely inflate their lungs except at a time when they make a special respiratory effort. This is due to the fact, as has been experimentally demonstrated by Mosso, that the breathing capacity of man is about one-fourth larger than is necessary for the ordinary requirements of life; hence there is always a certain portion of the lungs practically idle, more or less, all the time. This peculiar physiologic relation has an important bearing on the question of air-supply to the body. It has given rise to the idea that the human race would be less subject to disease, and particularly to pulmonary consumption, if the air contained a greater abundance of oxygen. There does not seem to be a particle of proof to sustain this proposition. For, aside of the fact that three-fourths of the respiratory surface is capable of furnishing the requisite amount of oxygen for the ordinary pursuits of life, it is well known that a large portion of the inhaled oxygen is not abstracted by the blood, but returns with the expiratory air. Indeed, when this whole subject is viewed strictly from the point of developing the chest capacity, the conclusion appears inevitable that phthisis would be much less common if the air were deprived of a portion of its oxygen. People living in the rarefied air of high altitudes have larger chests, and are less liable to phthisis than those living in lowlands, and it is conceded that the beneficial influence of high mountain air on this disease is largely attributable to the deeper and fuller respiratory movements which are necessary to obtain the requisite amount of oxygen for respiration from the attenuated air. So far as pulmonary consumption is concerned it is, therefore, not so much a question of abundance of oxygen in the air as it is one of complete lung-expansion; for, owing to the downward distribution of the large bronchial tubes, the lower parts of the lungs are filled first and most completely, and the apices last. Hence, if the lungs are not fully distended, it falls to the lot

of the apices to remain idle, and it is in a great measure due to their inactivity and consequent weakness that the upper parts of the lungs mark the beginning of consumption in the great majority of cases.

It is very essential, therefore, that the whole lung-surface should be developed and be made to take part in the function of respiration. To achieve this end deep, voluntary breathing should be encouraged at intervals of an hour or two throughout the day, and for ten or fifteen breaths in succession at a time. This can be done everywhere, care being, of course, taken that the air is inhaled only through the nose, especially in the open, so that the latter is fairly warmed before it reaches the lungs. Among the many breathing exercises the following movements may be practised with good effect: The arms being used as levers, are swung backward as far as possible on a level with the shoulders during each inspiration, and brought together in front on the same level during each expiration; or, the hands are brought together above the head while inspiring and gradually brought down alongside the body while expiring. Another very effective method to expand the chest is to take a deep inspiration and, during expiration only, the person will count as long as possible in a loud voice. A male person, with a good chest, can count up to sixty or seventy, while in a female, even with good lungs, this power is somewhat reduced. Practice of this sort greatly enhances the breathing space and the ability to count longer is a means whereby the improvement going on within the chest can be measured. The effects of many of these movements may be facilitated by the use of dumb-bells and of chest-weights made specially for this purpose.

Bodily Exercise. While it is well understood that bodily exercise must be entirely avoided during the activity of the disease, it is very desirable, in fact, incumbent on the medical attendant to advise it under certain limitations. One of

the most efficient modes of bodily exercise is walking. Dr. Brehmer, of Görbersdorf, was probably the first to advocate walking up a gradual ascent for consumptives. He prescribes a walk of half an hour or an hour, twice daily, either on the level or on a slight upward grade, with great benefit. Other modes of exercise may be undertaken, but whatever their nature may be great care must be taken that they neither tire nor become wearisome.

CHAPTER XVI.

THERAPEUTICS CONTINUED: MEASURES TO INCREASE THE VITAL RESISTANCE OF THE PULMONARY NERVE SUPPLY. INJECTIONS OF SILVER-NITRATE, ETC.

If the neurotic theory correctly explains the pathogenesis of pulmonary consumption, it has for some time appeared to the author that direct or immediate stimulation of the trunk of the vagus should exert a beneficial influence on the condition of the lungs in this disease. For is it not true that painful affections of the peripheral nerves, like sciatica, intercostal neuralgia, herpes zoster, etc., are relieved by applying a stimulating fly-blister to or near the roots of the nerves which are involved in these disorders, and why should it be impossible for a similar impression made on the vagus, although it is chiefly a motor nerve, to have a like influence on the organs to which its peripheral fibers are distributed?

Moved by an impulse of this kind it was determined nearly two years ago to give special attention to the vagi in the neck by massaging, kneading and compressing them through the overlying textures. While these measures seemed to benefit the cough, expectoration, and oppression in the chest to some extent it soon became evident that their influence was not far-reaching enough to be of decided value, yet they gave sufficient encouragement to continue work in the same direction. About this time the author learned of the good effects which were obtained in severe cough, associated with ophthalmic goiter, and also in epilepsy, by stretching* the

* "Stretching of the Pneumogastric Nerve." By M. Jaboulay, *New York Medical Journal*, May 7, 1898, abstracted from *Lyon Médical*, April 7, 1898. M. Jaboulay also recommends this operation in pneumonia.

vagus in the neck, and it was concluded to test the value of this operation in phthisis. Before it was carried into practical effect, however, it appeared to the author that probably the subcutaneous introduction of some active although conservative irritant, like nitrate of silver, immediately over the course of the pneumogastric nerves in the neck might furnish the stimulus necessary to arouse them out of their abnormal condition. An effort was made to carry out this idea practically, and the first injection was given in the latter part of August, 1898. After many trials of various dilutions of silver-nitrate it was found that from four to seven minims of a two-and-a-half-per-cent. solution of pure silver-nitrate answered the purpose of bringing about the desired counter-irritant reaction; although more recently five minims of a five-per-cent. solution have been used with better effects in some cases. In fact it has been found that some cases require larger doses than others, and that a stronger solution is borne by the same patient as the treatment advances. The place which has been selected for its administration is immediately over or slightly behind the pulsating carotid artery, in the region of the neck, in a line between near the angle of the jaw and the clavicle, and nearer the latter than the former point. Points higher up along the course of the nerve may also be chosen.

The side of the neck in which the injections are given is also of importance. As a rule it will be found that pressure over the course of one vagus, in the region of the neck, produces more pain on one than on the other side in most phthisical persons, and that this supersensitiveness corresponds with the side on which the affected lung is situated. If the physical signs and the supersensitive spot in the course of the vagus are found to occupy the same side it is advisable to give most of the injections on that side, and occasionally one on the other. If it is found, however, that the sensitive spot is located on the side opposite to that of the diseased

lung it is good practice to give an injection on each side at the same time. If it is found that both lungs are involved simultaneously it is best to administer an injection on each side at once.

In order to avoid puncturing the carotid artery or its neighboring jugular vein it is important to lift the skin between the thumb and the forefinger of the left hand and introduce the needle just under the elevated skin. The silver solution alone produces considerable local pain immediately after its introduction, and in order to avoid this it is necessary to precede it with an injection of five minims of a two-and-a-half-per-cent. solution of cocaine* hydrochlorate.

The following plans have been found the most practical for the introduction of these agents: Inject the cocaine solution; detach the syringe from the needle, and let the latter remain in the puncture. Wash out the syringe with water, draw the silver solution into the syringe, attach the latter to the needle, and throw in the required amount. Or a double-barreled syringe, as has been suggested by Dr. W. O. Hermance, of this city, containing a stop-cock and a single nozzle, to which is attached the needle, may be employed. One barrel is filled with the silver and the other with the cocaine solution, and the required amount of each may be injected without detaching the syringe from the needle. The author has used this syringe with a great deal of satisfaction. The reason that the silver and the hydrochlorate of cocaine must be injected separately lies in the fact that when mixed they form a flocculent precipitate, which clogs the needle very often. To obviate this the author has recently used the nitrate in place of the hydrochlorate of cocaine mixed with the nitrate of silver in the same solution, and injects ten minims of the same in place of injecting successively five minims of each of the two separate solutions. The last method is to be preferred.

* Renew the cocaine solution every two weeks.

The local effects of the injections show themselves in nodular, sometimes in diffuse swelling, and in redness and pain, but according to the author's experience none of them become markedly pronounced or intolerable. In order to alleviate the pain it is a good plan to apply an ointment of cocaine* for the first twenty-four hours after each injection. Abscesses have occurred perhaps fifty times, and small sloughs four times, in about two thousand injections, but neither the abscesses nor the sloughs were followed by the least detrimental influence on the patients.

The number of injections which are necessary depends on circumstances. The highest number that has been administered by the author to one patient were twenty-one in the course of eleven months. The injections should be repeated every week or ten days, unless the previous injection is followed by too much irritation and local disturbance, and should be continued so long as they seem to confer benefit on the patient. In urgent cases due to excessive coughing the injections must be repeated in three or four days.

During the last two years the author has administered the nitrate of silver injections to two hundred and fifty consumptive patients, from which some deductions in regard to their action may be drawn, and the following analysis of their effects on the symptoms and conditions gives at least an approximate idea of its remedial value in this disease. For the purposes of convenience and illustration these cases are divided into the three following classes: Incipient, those with beginning physical signs, some emaciation, cough, expectoration, etc.; advanced, those without excavation, but with emaciation, much cough, expectoration, night-sweats, loss of appetite, etc.; and far advanced, those with excavation, marked emaciation, night-sweats, cough, expectoration, etc.

* ℞ Unguenti Ichthyolis, 25 p.c. ..... ℥ ss.
Cocainæ Hydrochlor..... ..... gr. vi.
M. Sig. Use as an external application.

Influence on Cough and Expectoration. When these injections were first undertaken it was entirely with the thought of benefiting the cough and expectoration in this disease and it was, therefore, gratifying to find that with but few exceptions these symptoms improved in a very marked degree, not only in the incipient and advanced cases but in the great majority of those which were far advanced. This was so striking that it could not be mistaken. A number of cases testify, however, that both cough and expectoration become aggravated for from twenty-four to forty-eight hours after the injections are given and improved from that time on. One of the very far-advanced patients, who has since died, had a harassing cough day and night and also complete aphonia, volunteered the statement in a letter to the author that "my cough is almost entirely relieved for three or four days after each injection, but as soon as the effects of the same die out it returns as severe as before. During this time my voice is stronger, too." Another patient in the advanced stage of the disease had such violent coughing spells that he was unable to sleep. He received the first injection in the afternoon, two years ago, and slept well the following night until three o'clock next morning. His cough and expectoration disappeared almost entirely in a few days, and he made a permanent recovery. Besides relieving cough and expectoration the injections also have a beneficial influence on dyspnœa and oppression in the chest. This property is well brought out in the alleviation which they give to the symptoms of asthma.

Influence on Vomiting. One of the most distressing features of phthisis is the vomiting which ensues in its later stages. It usually occurs after meals and is nearly always preceded by cough. The injections frequently have a good influence on this symptom, even in far advanced cases.

Influence on Sleep. This function is usually improved, but it may be an indirect effect of the betterment of the night-cough brought about by the injection.

Influence on the Appetite. The injections seem to possess a marked influence on the digestive function, for in nearly every case that came under observation the power of eating was increased, and sometimes to a remarkable degree. This was, of course, most noticeable in the incipient and advanced, although it also held true in some of the far advanced cases.

Influence on General Strength. This was greatly benefited in most of the incipient and advanced cases and also in a number of the far-advanced cases. How to account for this improvement, which frequently follows immediately after an injection, on the score of any theory is a difficult matter, but the fact exists and forces itself too often on one's attention to escape notice.

Influence on the Physical Signs. In many of the incipient and in some of the advanced cases there was noticed improvement in the physical signs. This was so frequently the case that it could not have been a mere accident.

Influence on Fever. A decline in fever has often been noticed to follow the injections, both by others and by the author; but whether this is due to their direct influence on the pyrexial process or whether it comes directly through an improvement of the general system it is difficult to say at present. It is necessary to first study their effects on acute pulmonary fever before coming to any definite conclusion on this point.

Influence on Night-Sweats. Diminution of the night-sweats has been noticed by the author, but not to the same extent as that which was observed by Dr. Gosman* in the twenty cases which he treated. In his summary of results (*see below*) he states that this symptom "stopped absolutely in eighteen and improved in the other two cases."

* "A Report of Twenty Cases of Pulmonary Tuberculosis treated by Hypodermic injections of Silver Nitrate over the Pneumogastric Nerves," by G. Henry Rankin Gosman, of Brooklyn, N. Y.; Member Medical Staff Kings County Hospital. *Philadelphia Monthly Medical Journal*, July, 1899, p. 406.

Influence on Weight. As has been stated already these injections were begun solely for the purpose of alleviating the phthisical cough and expectoration, and it was surprising, therefore, to find that they also exerted a most beneficial influence on the body-weight. The gain is chiefly confined to incipient and advanced cases, but it has also been observed in the far advanced condition to some extent. Indeed this phenomenon, as it has shown itself in some cases, is really surprising, since it seems barely possible that an almost insignificant quantity of nitrate of silver, thrown in the neck once a week, should produce such striking effects on the weight of the body. That this power exists is graphically illustrated in the following weight-charts:

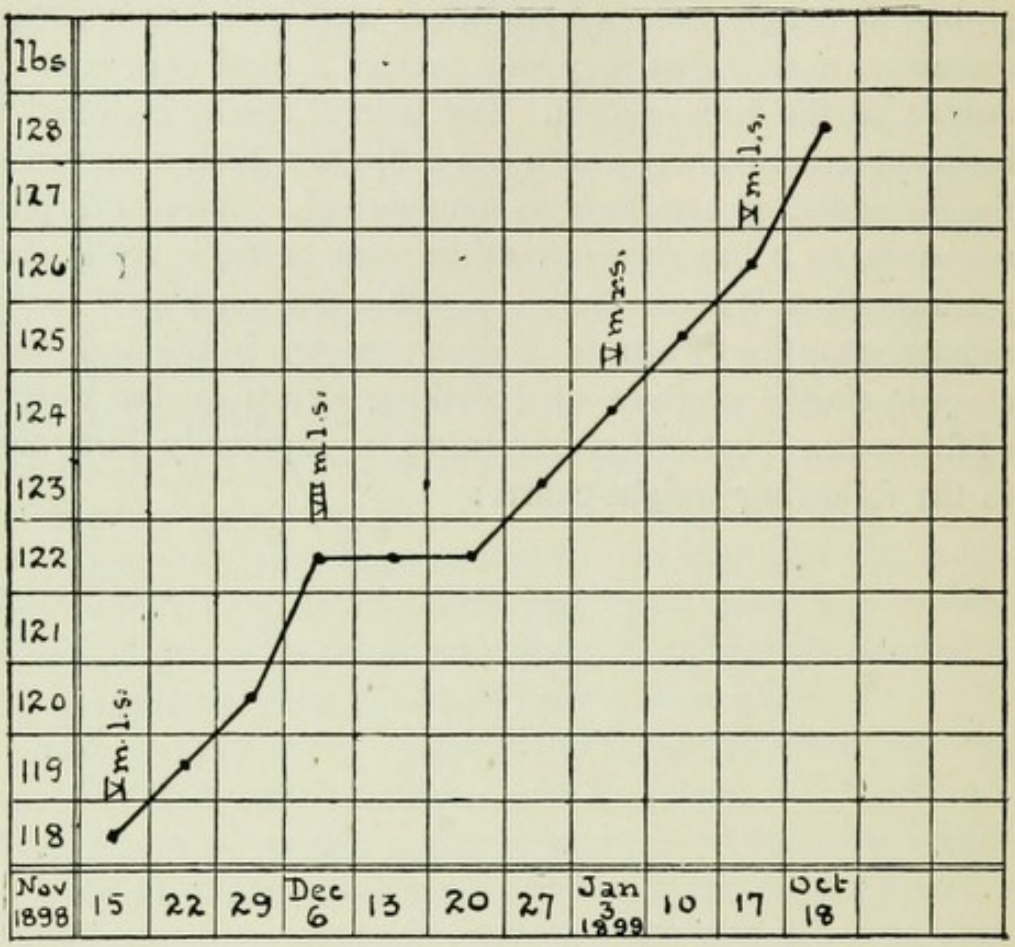

Fig. 28.—Represents the case of a young man aged 18, who was first seen November 15, 1898, and whose father, maternal grandfather, aunt, and uncle died of phthisis. Hemoptysis two years before. Coughed and expectorated and lost in flesh. Weight 118 pounds. Dulness in left supra clavicular and superscapular regions, and extending to first intercostal space and to middle of interscapular region. Sibilant and sonorous and subcreptant râles distributed over upper half of left lung. November 20, 1899, weighs 128 pounds; dulness is materially less, and a few sibilant râles are heard in upper part of left lung.

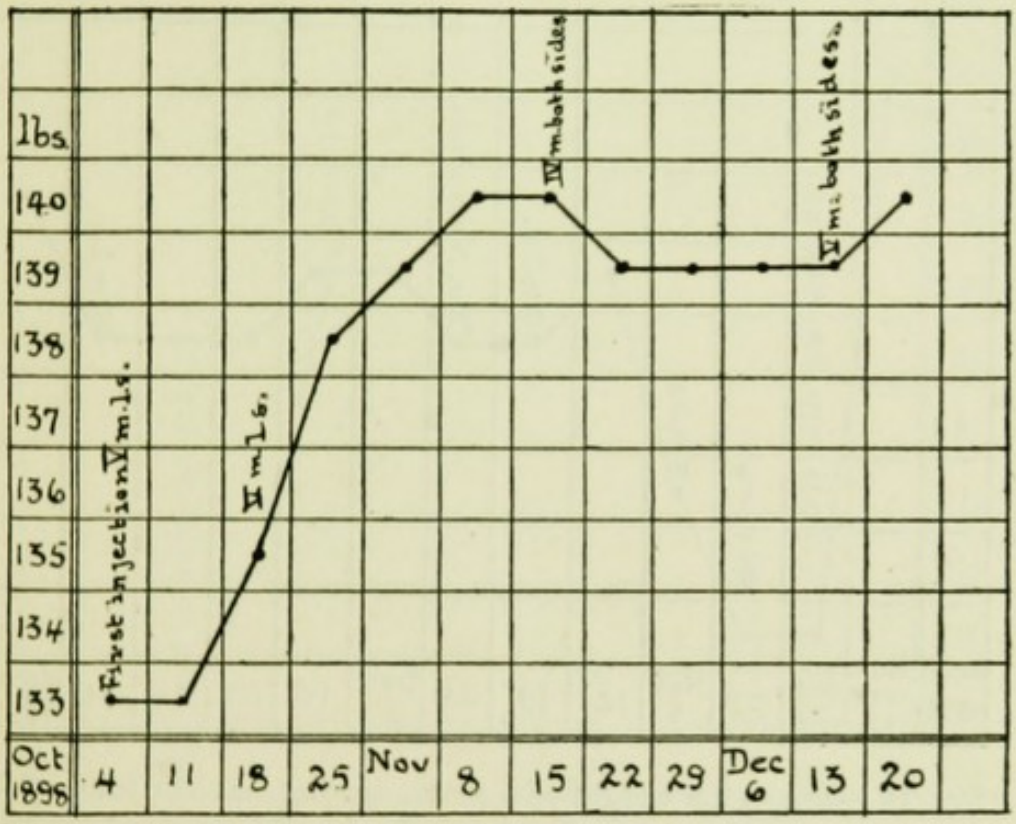

Fig. 29.—Gives the weight-history of a male, aged 42, a tailor by occupation, who received the first injection October 4, 1898. For two years he had been having cough, blood-spitting off and on, and a mucopurulent expectoration. He had dulness and subcrepitation in the upper part of left lung. He has been working at his trade since January, 1899.

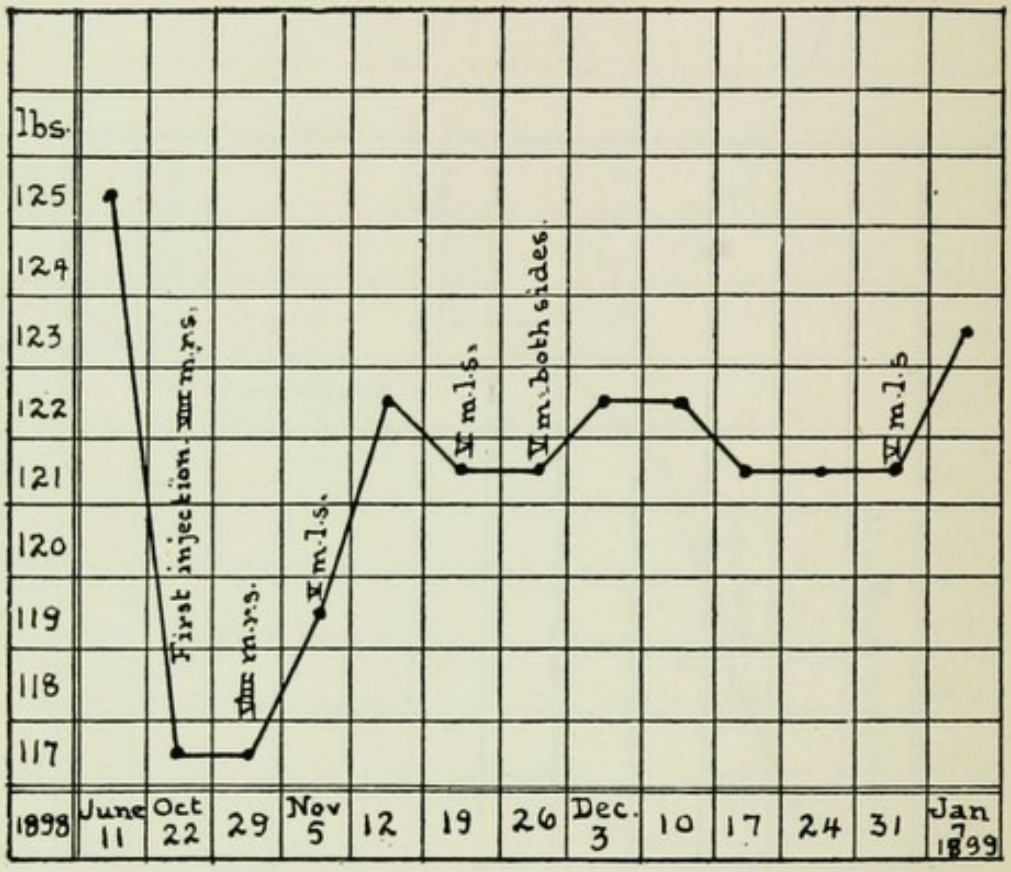

Fig. 30 —Shows the weight of a male, aged 47, who in June, 1898, weighed 125 pounds, and on October 22d, when he received the first injection, weighed 117 pounds. Cough and expectoration rather profuse, latter yellow and sometimes blood-streaked. Dulness and excavation in left apex, and sibilant râles extending to base on same side This patient has not been seen for some time, and from his physical condition one would not be led to expect any permanent relief, yet the above tracing shows that the injection had an influence on his weight. (Has since died.—Author.)

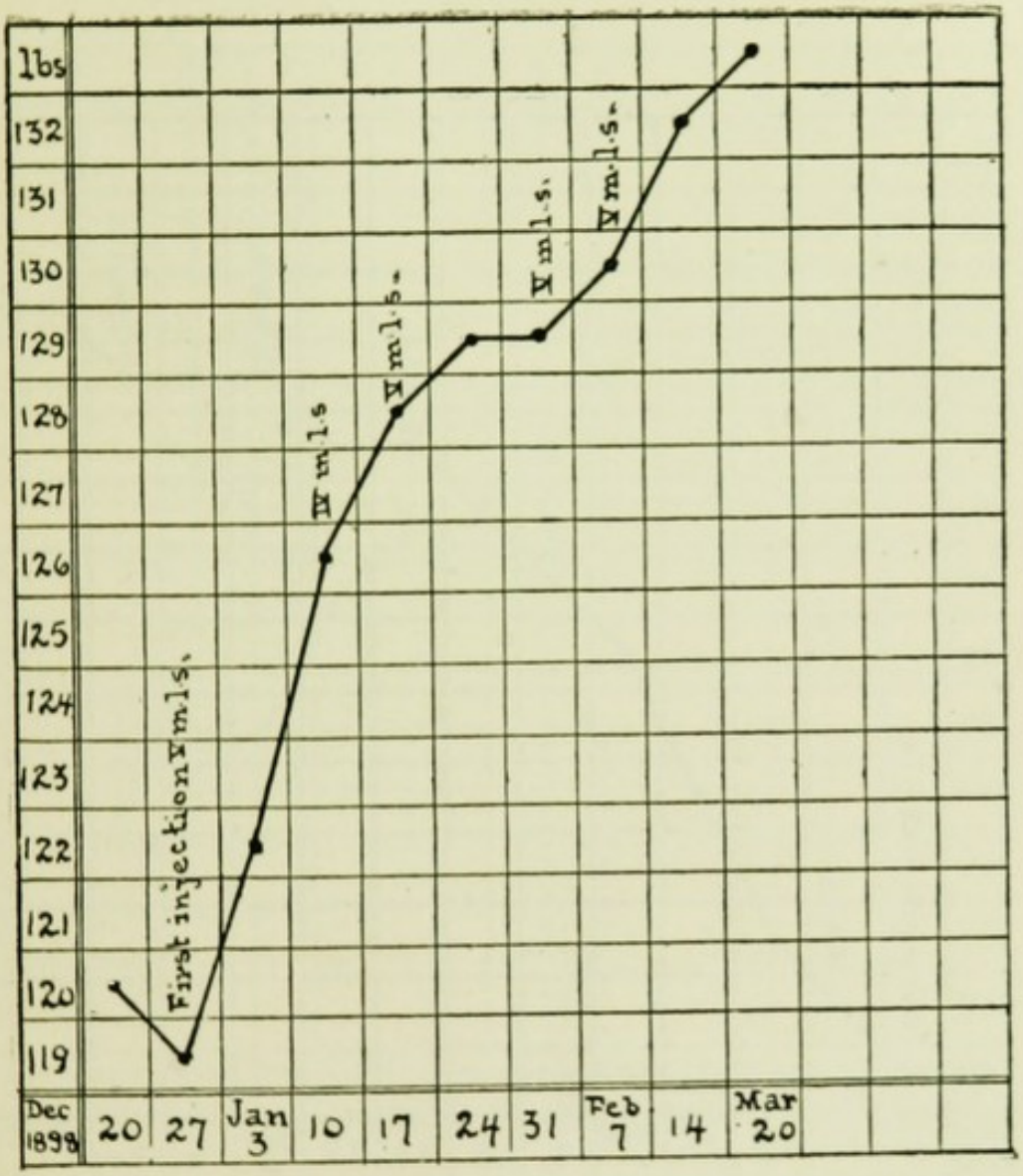

Fig. 31.—Represents the weight of a colored man, aged 27, who was admitted December 20, 1898, for anal fistula, associated with dulness and moist râles in left apex. At this time his weight was 120 pounds. Had been coughing a great deal for several years. One week after admission, and on the day when he received his first injection, he weighed 119 pounds, a loss of a pound in a week. After this he gained and is doing very well at present, and worked steadily for the last year and a half.

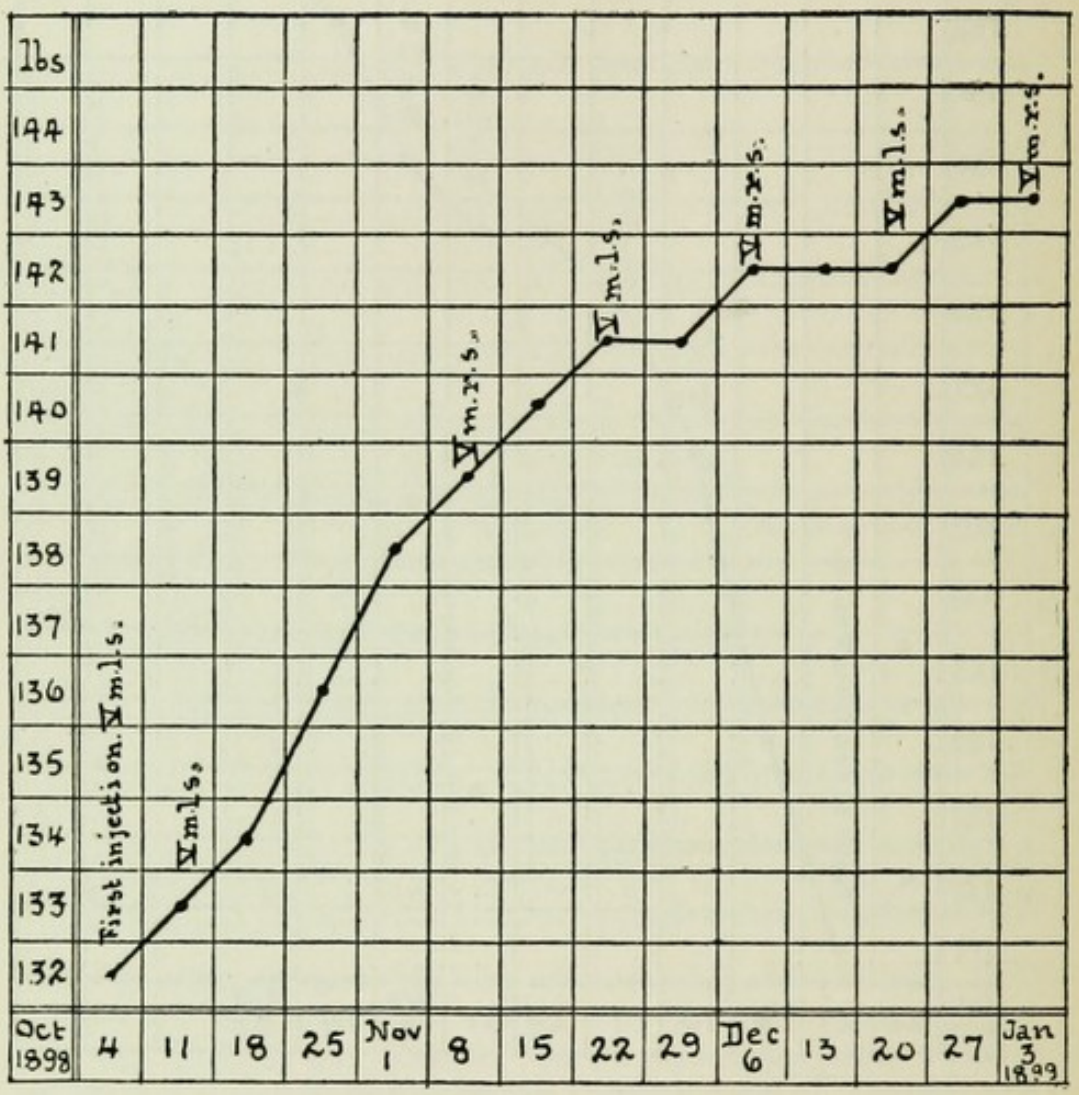

Fig. 32 —Shows the weight of a male, aged 69 who had severe cough, profuse expectoration, and great dyspnœa. There were dulness in the left supra- and infraclavicular regions, and subcrepitant and sibilant râles over his whole chest when the first injection was given. He began to improve at once, and at the present time his chest is free from all abnormal signs, and he is doing very well.

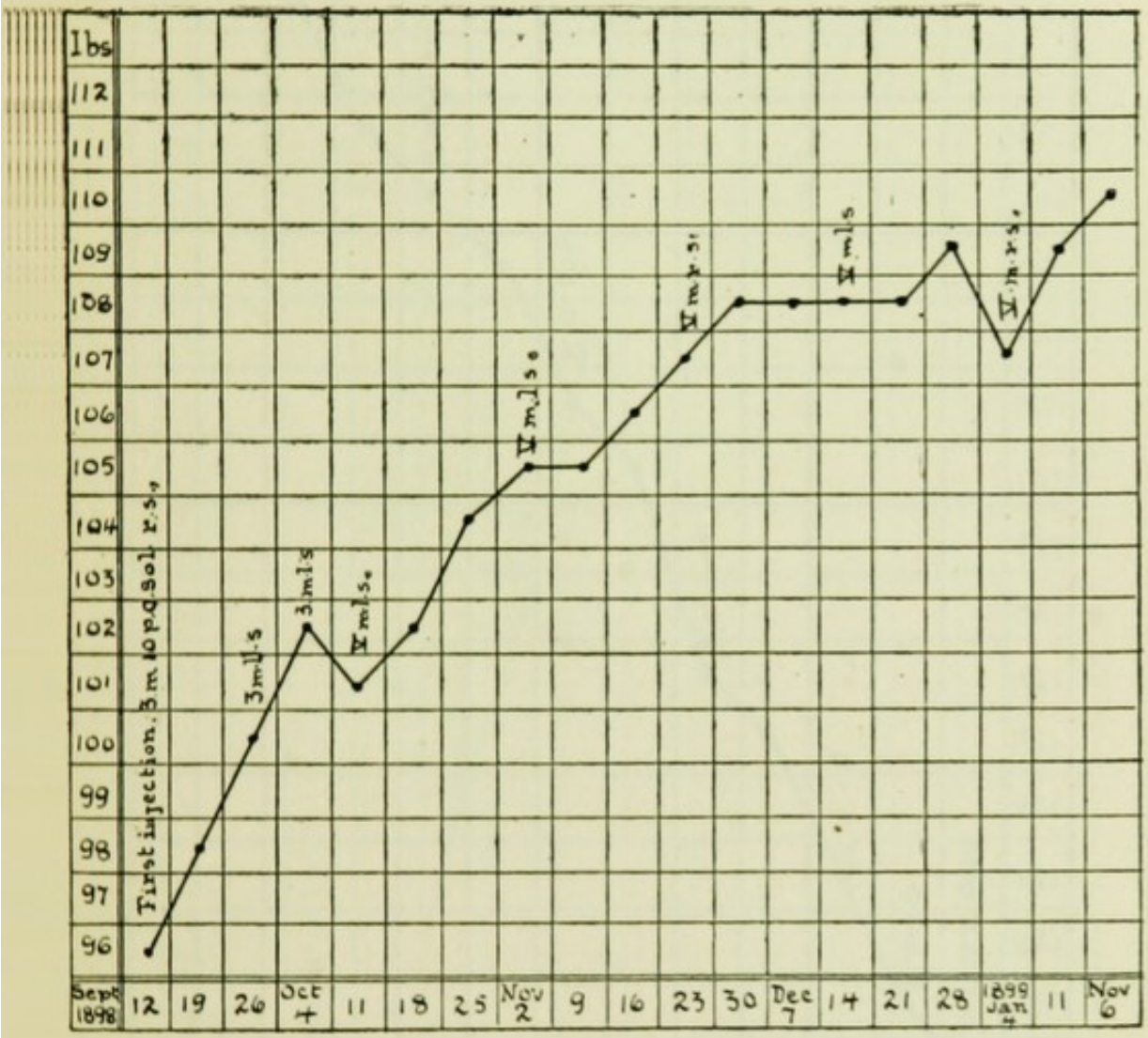

Fig. 33.—Represents the weight of a consumptive female, aged 35, who had been coughing for more than two years before she received the injections. Her cough was troublesome, the expectoration profuse, and the appetite poor. There were dulness, and a few moist râles in right supra- and infraclavicular regions. After the injections she improved in symptoms and physical signs. Her strength returned, and she went to work the last of January, 1899, and with the exception of a few weeks' vacation during the summer she has been engaged up to the present time. Her staying power is better now than it has been for several years.

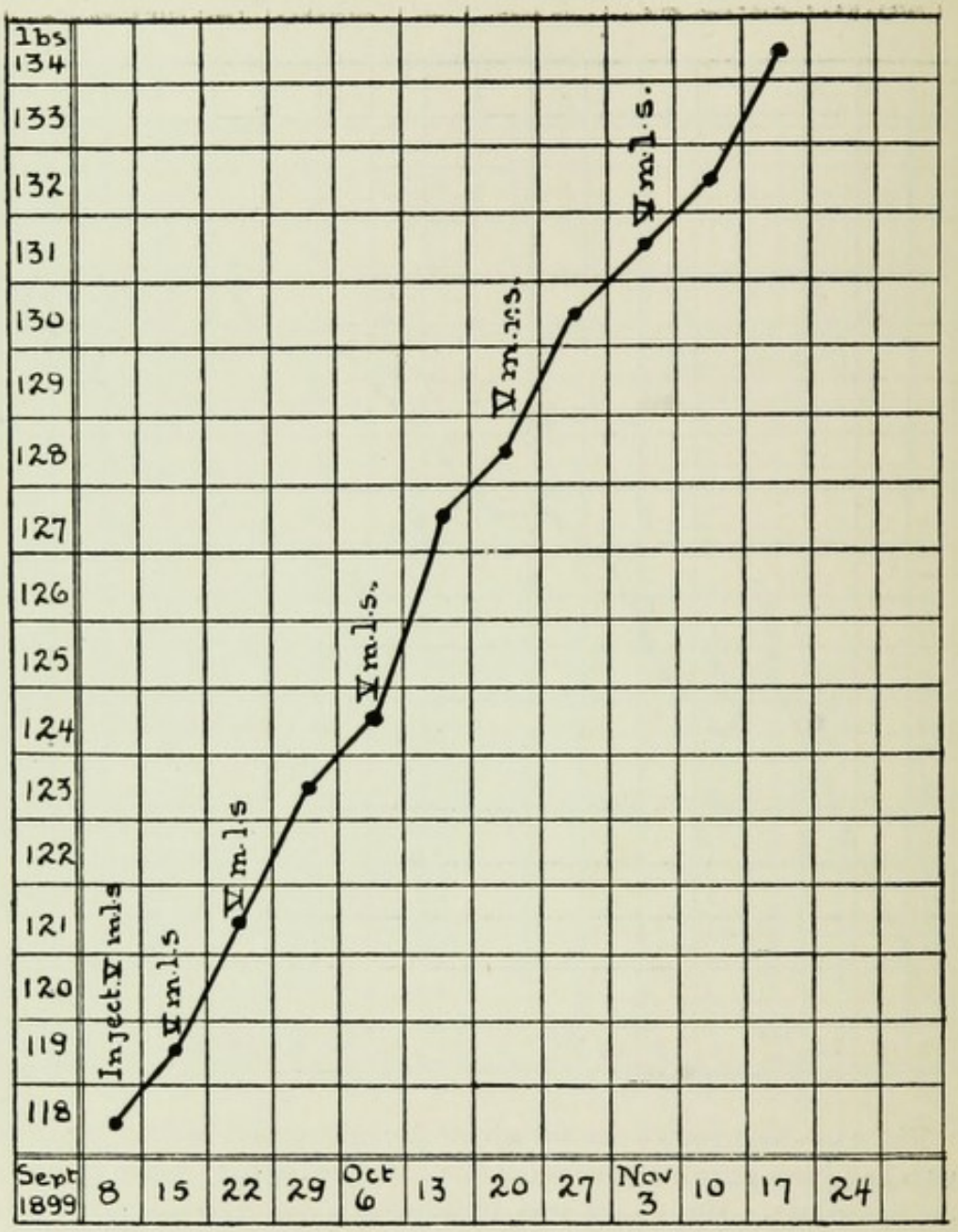

Fig. 34 —Male, aged 25, with cough and expectoration for several years, had recently much dyspnœa, night-sweats, poor appetite, and vomiting. There were dulness, moist râles, amphoric breathing, and pectoriloquy in left supra- and infraclavicular regions. Beside the increase in weight his other symptoms markedly improved.

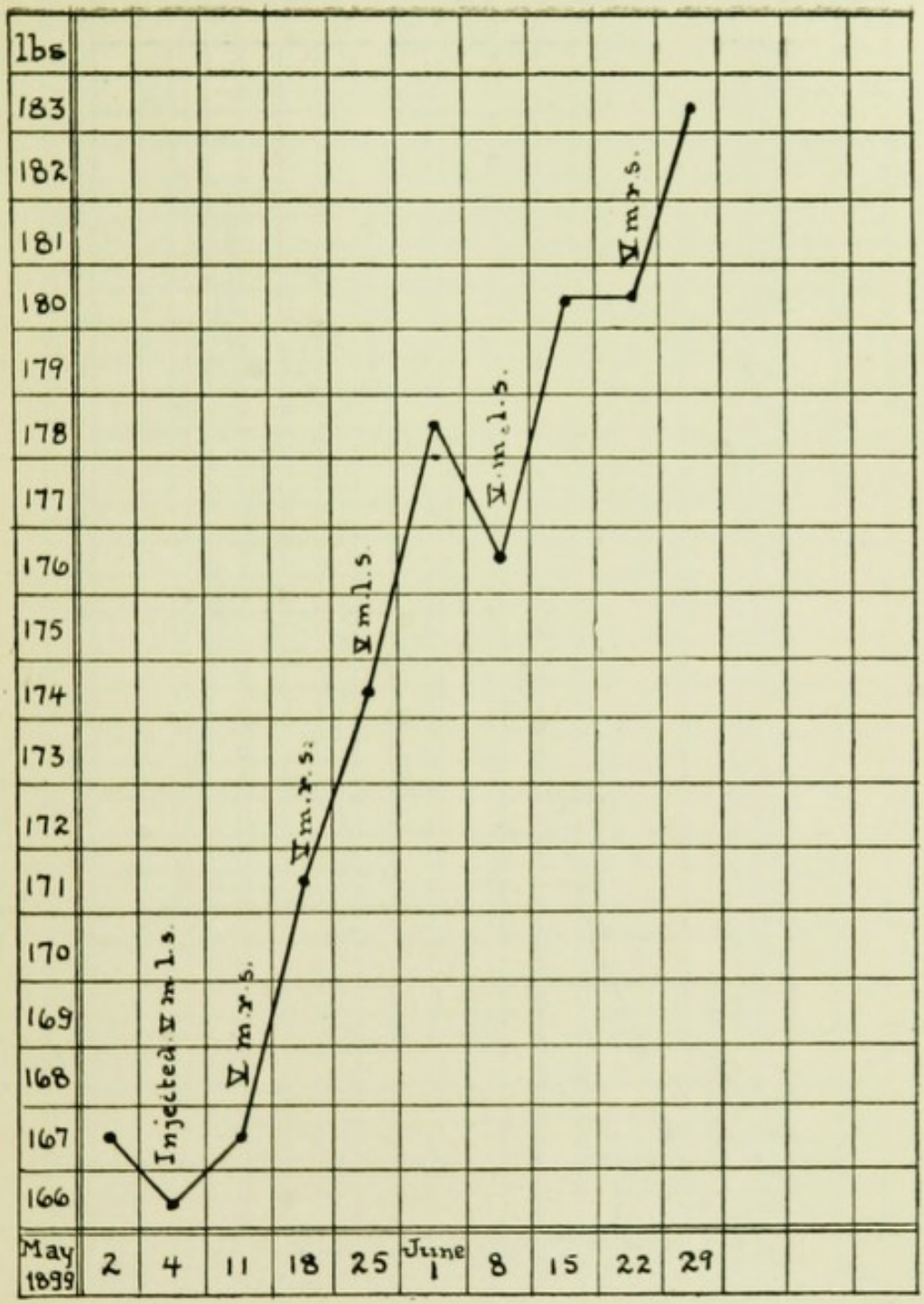

Fig. 35.—It will be seen from this chart that from the second to the fourth of May this patient, who was a colored male, aged 27, lost one pound in the two preceding days of his first injection, and he improved in symptoms and physical signs from that time on.

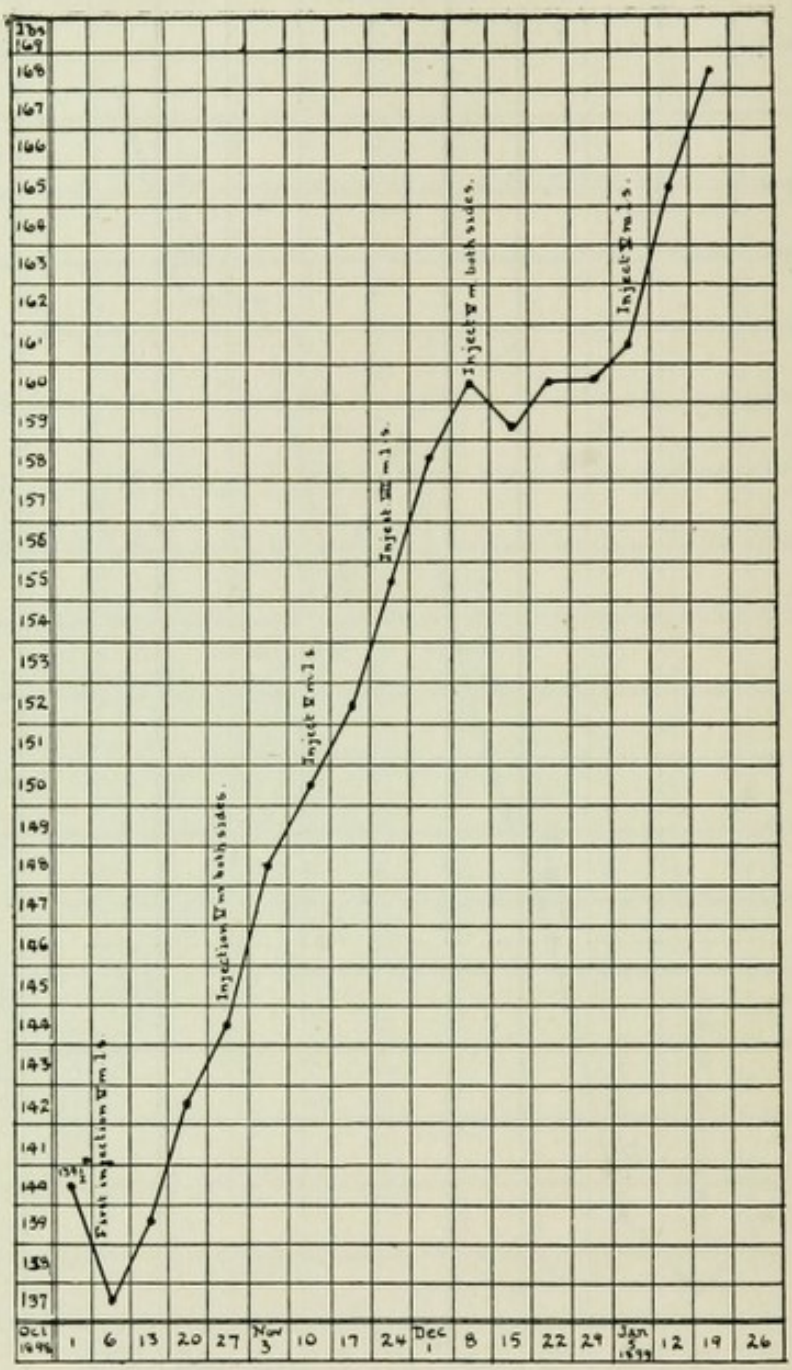

Fig. 36.—Illustrates the weight of a male, aged 60, who had chronic infiltration of the whole of the left lung with a great deal of cough and expectoration. It will be observed that he lost two and a half pounds in the five days immediately preceding the first injection. After this he made a steady and an almost uninterrupted gain.

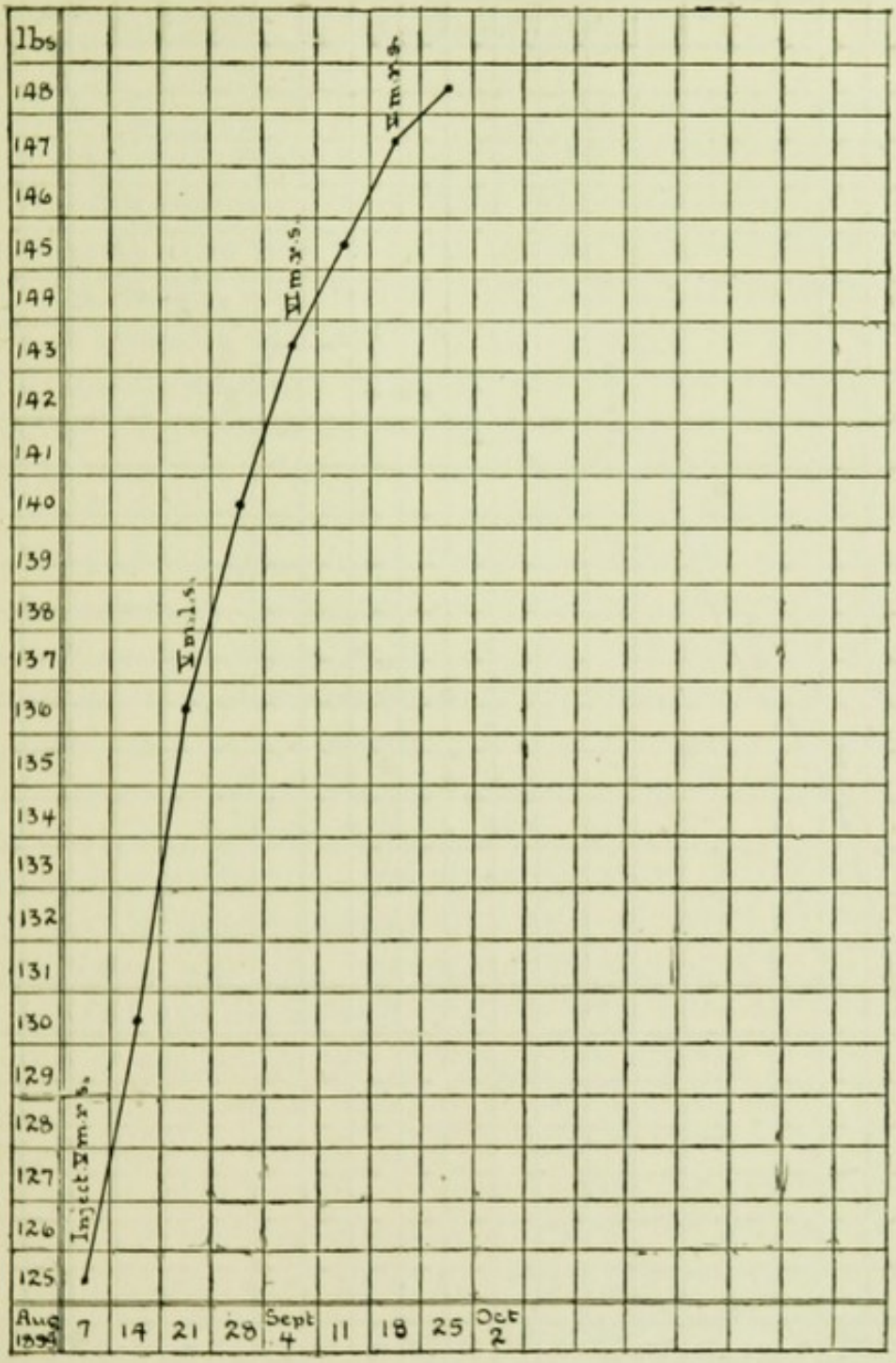

Fig. 37.—Shows the weight of a male, aged 47, mildly insane, with cough, expectoration, dyspnœa, and loss of flesh and appetite for a year. There were impaired percussion resonance and subcrepitation in right apex. He increased from 125 to 148 pounds in seven weeks, and went to work, and continues in good health up to the present time (November, 1900).

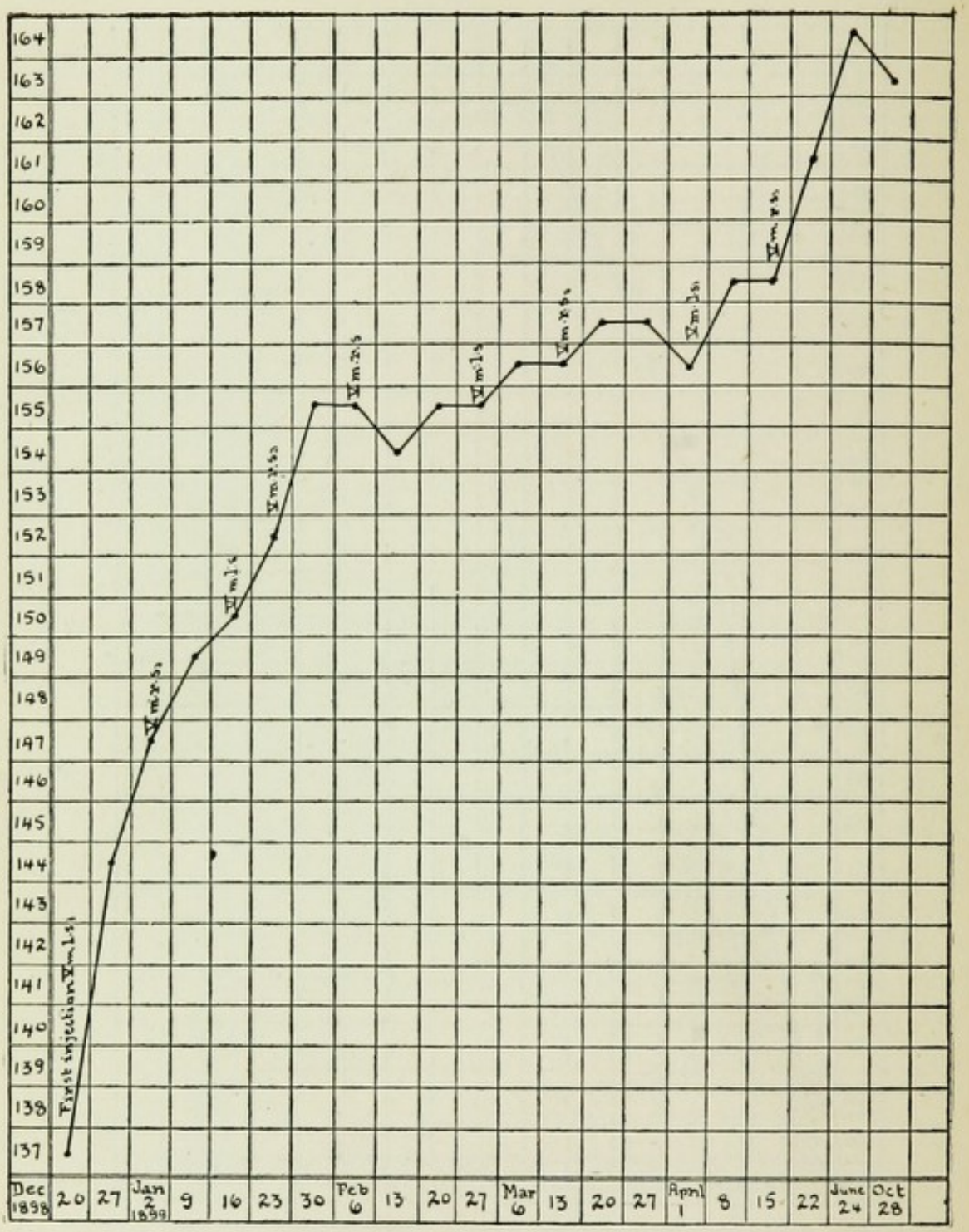

Fig. 38 gives the weight of a male, aged 42, who, when he came under observation, had complete infiltration of his right lung, accompanied by severe cough, profuse expectoration, vomiting, anorexia, chills, fever (101 3/5° F.), night-sweats, and decided loss in flesh.

So far as the author is aware there are no published reports of any further experience in regard to the action of the silver injections in phthisis except those which are passed in review below. The first of these is that of Dr. Gosman, of Brooklyn, N. Y., who treated twenty cases,* an analysis of which is given in the table on page 368.

The conditions under which these cases were treated are stated by Dr. Gosman to have been as follows: "(1) They were all cases in which the diagnosis was unquestionable, the bacilli being found in the sputum. (2) They all had been treated for some time without any benefit. (3) In all, the surroundings, habits, general treatment, etc., were identical before and after the injections. (4) No case is reported that was under observation less than five weeks, and most of them were observed for two months or more. (5) Fifty per cent. of them had complicating diseases, which were anything but favorable to good results."

Now in analyzing the contents of the above table it will be seen that the aggregate gain in weight of seven of the incipient cases was 39¼ pounds, or an average gain of five pounds and a half—a result which is not an unfavorable exhibition of the action of a remedy when applied to cases in which other treatment had proved futile, especially when it is considered that at least three of the seven cases had complications. It must be stated, however, that the greatest gain took place in the non-complicated cases. In the advanced and far-advanced cases there was practically no gain in flesh. Cough and expectoration ceased entirely in two, improved in ten, and failed to improve in eight cases (Gosman). In those cases where a record is given of the physical signs it is shown that these disappeared in one, improved in three, and became more pronounced in two. The temperature improved markedly in five, slightly improved in eight, and did not improve in seven cases (Gosman). Its action on the night-sweats

* See foot-note on page 354.

Tabular View of the Condition of Dr. Gosman's Twenty Cases of Pulmonary Tuberculosis Treated by Hypodermic Injections of Silver-Nitrate.

No.	Duration of Phthisis.	Stage.	Complications.	Influence of Injections on				
				Weight. Gain. Loss.	Cough and expectorat'n	Physical signs.	Temperature.	Night-sweats.
1	4 yrs.	Incipient.	Mitral regurg.	G. 1¼ lbs.	Greatly improved.			Ceased.
2	3 mos.	"	None.	G. 17 lbs.	Not stated.	Same.	Normal.	"
3	1 yr.	"	Tubercular abscess.	G. 12 lbs.	Much improved.	Same.		"
4	1 yr.	"	Drinker.	Same.	Cough ceased.		Normal.	"
5	6 mos.	"	Syphilis.	G. 3 lbs.	Improved.	Improved.	Same.	"
6	9 mos.	"	Mitral regurg.	G. 1½ lbs.	Improved.	Disappear'd		"
7	2 yrs.	"	None.	Lost.	Cough better.	Improved.		"
8	5 mos.	"	Laryngeal tuberc'losis	Same.	Cough improved.		Same.	"
9	6 mos.	"	Drinker.	G. 1½ lbs.	Not stated.	Not stated.	Improved.	"
10	2 yrs.	"	None.	L. 2 lbs.	Cough improved.		Same.	"
11	5 mos.	"	None.	G. 3 lbs.	Improved.		Improved.	"
12	7 wks.	Advanced.	None.	Not stated.	Cough improved.	More pronounced.	Improved at first.	"
13	8 mos.	"	Laryngeal tuberculosis	Same.	Same.		Same.	"
14	8 mos.	"	Gastritis.	Lost.	Slightly improved.	More marked.	Slightly improved.	Improved.
15	6 mos.	"	Mitral stenosis.	Lost.	Not stated.	Markedly improved.	Markedly improved.	"
16	7 mos.	"	None.	G. 2 lbs.	Not stated.	Same.	Worse.	Ceased.
17	1 yr.	Far advnc'd Died.	Nephritis. Double hernia.					Had stopp'd
18	1 yr.	Far advnc'd	None.					Ceased.
19	1 yr.	"	Aortic regurgitation.					"
20	4 or 5 mos.	"	Aortic and mitral disease.	Lost.			Improved.	"

• *Philadelphia Monthly Medical Journal*, July, 1899, p. 406.

seems to have been the most uniform and constant. This symptom abated entirely in eighteen and improved in two of the cases. The general strength, appetite and sleep improved in about half the cases (Gosman).

The other report* above referred to is that of a case treated by Dr. G. H. Franklin, of Hightstown, N. J., the interesting history of which is partially given in the following abstract: Male, aged 50, without a family history of phthisis, had hemoptysis February 24, 1897, which was believed to have been of traumatic origin. With the exception of general malaise, of which he complained in the spring of 1898, he was comparatively well until the following October, when he began to lose flesh. In December of the same year he began to have slight hectic, morning chills, afternoon fever, and night-sweats. January 1, 1899, he had an attack of grip, from which he rallied imperfectly. On the 16th he had right-sided pleurisy, with infiltration of upper part of lung on same side, and his evening temperature now began to range from 101° to 103° F., his pulse from 100 to 110, and his respiration was about 24. Night-sweats, cough, expectoration and anorexia continued and he lost flesh rapidly. The treatment consisted of alcohol baths, rest in bed, cod-liver oil, hypophosphites, strychnine, creosote, forced feeding, and codeine to control the cough. On February 1st, when the case presented no visible improvement, a hypodermic injection of silver-nitrate was given over the course of the right vagus—all the other conditions concerning treatment, etc., being maintained exactly the same as they were previous to the injection. His expectoration gradually lessened, and his cough ceased on the fourth day after the injection, so that the codeine was discontinued. After the fifth day his temperature became normal, pulse fell to

* "History of a Case of Incipient Phthisis Treated by Hypodermic Injection of Silver-Nitrate over the Vagi," *Philadelphia Medical Journal*, April 15, 1899.

80, respiration to 18 and 20, night-sweats ceased, appetite improved, and he began to gain in flesh. The whole aspect of the case was changed, and he rapidly improved. A second injection was given on the left side on the eighth day, and another, a week later, on the right side, although they did not seem necessary.

When in health the patient usually weighed 140 pounds. On December 12th he weighed 130 pounds. Estimating that from that date to February 1st, the time of the first injection, he lost at least 10 pounds, his weight on the latter date would have been 120 pounds. February 25th, twenty-five days after the first injection, he weighed 148 pounds—having gained approximately 28 pounds. March 6, 1899, his weight reached 152 pounds, and now weighs ten pounds heavier than he weighed for ten years.

What, now, are some of the practical deductions that may be drawn from the action of the silver injections in the treatment of phthisis? What is their value and what is their limitation? From the evidence which has been brought forward in the preceding pages it is qute clear that they have a marked beneficial influence on some of the most important symptoms of this disease. They ameliorate the cough and expectoration, increase sleep, appetite and general strength, check night-sweats, reduce fever, improve the physical signs and sometimes increase the body weight to a remarkable extent. The best results are obtained, of course, most often and most readily in incipient cases, although, according to the author's experience, which is also confirmed by the improvement in Dr. Franklin's case, they are equally good in many of the advanced cases. In the author's collection of 40 cases* there were 18 in the advanced stage, and 8 of

* "The Hypodermic Injection of Silver-Nitrate over the Course of the Vagi in the Treatment of Pulmonary Consumption." By Thomas J. Mays, A M., M.D., *New York Medical Journal*, February 11, 1899 *Philadelphia Medical Journal*, February 11, 1899 ; *Boston Medical and Surgical Journal*, February 9, 1899.

these made an average gain of almost eight pounds. The five advanced cases in Dr. Gosman's series gained practically neither in weight nor in any other respect, excepting in improvement of cough and cessation of night-sweats. In the great majority of far advanced cases the cough, expectoration, night-sweats, vomiting and some other prominent symptoms are frequently alleviated, but the local condition of the lungs remains unchanged.

Another important question is in regard to the duration of the effects of the injections. Are they merely of a temporary character, or do they give promise of something better? Time alone will be able to determine this point. Of the 40 cases* reported by the author, not quite a year ago, there were 7 incipient, 18 advanced, and 15 far advanced cases. The incipient cases are all well, and are at work, or able to work. Of the advanced cases 5 died, 2 continue invalids, and 11 are well and at work. Of the far advanced cases, 11 are dead and 4 are alive, 2 of whom are able to work. In regard to the high death-rate among the last class it may be stated that seven of these were certain to die when the injections were begun—the latter having been given merely for the purpose of ascertaining what influence they would exert on this stage of the disease. Taking it all in all, therefore, and deducting the 2 cases that are still invalided and the 4 far-advanced living ones (although one of these is doing housework) there remain 20 or 50 per cent. who are well and capable of doing work at the end of almost a year and a half after being treated.

Measures Which Meet Special Indications.

Indigestion. Persistent dyspepsia is often relieved by suitable special medication. If this depends on too much gastric acidity, as is frequently evidenced by sour eructa-

* *Op. cit.*

tions it will be alleviated by administering an alkali like liquor ammonia acetatis, in combination with tinctures of quassia, gentian, or serpentaria, an ounce of the former to three ounces of one of the latter. The addition of one drop of the fluid extract of xanthoxylum to each dose will enhance its efficacy. Sometimes equal parts of the tinctures of gentian, colombo, cinchona comp., and hydrastis canadensis, in teaspoonful doses, before meals will have a good effect. A grain of hydrastine hydrochlorate is sometimes followed by good results, especially if there exists torpidity of the liver. The combination of acids with the tincture of the chloride of iron, as given in the formula on page 342, is an excellent tonic when acids are indicated, and when marked anemia is present. If pain exists in the gastric region the application of a mustard plaster, once or twice a day, over that area is beneficial.

Insomnia. If sleep cannot be procured in any other way narcotics must be administered, and of all these morphine and codeine are preferable. A ten-grain suppository of asafœtida, at bedtime, is very frequently employed by the author, with good hypnotic effects.

Diarrhea. So soon as the bowels become rebellious no solid food is to be allowed, except thoroughly dried toast, but liberal quantities of liquid diet, like beef juice, milk, boiled, as well as peptonized, are to be given. The bowels should be flushed out with large quantities of warm water every day or two. If necessary one grain doses of powdered opium must be administered, two or three times a day, by the mouth. Or, what is better, the application of a mustard plaster over the whole abdomen, morning and evening.

Excessive Cough. If the cough depends on laryngeal embarrassment, the inhalation of watery vapor, impregnated with the compound tincture of benzoin, or the inhalation of the fumes of gum-camphor, or of five or ten drops of chloroform from a handkerchief, or the spraying of the

larynx with a solution of cocaine hydrochlorate, fifteen to twenty grains to an ounce of water, are very useful methods for allaying laryngeal irritability. Lozenges containing cocaine hydrochlorate, sugar, licorice powder and powdered acacia, and allowed to dissolve slowly in the mouth, also have a soothing effect.

Cayenne pepper ranks very high as a stimulant in the cough of phthisis. When phthisis follows the excessive use of alcohol it is of special value, but under these circumstances it must be given in very large doses, diluted with water. In ordinary cases of cough the tincture is to be given from two to fifteen drops, four times a day, while in that of alcoholic phthisis the author has seen doses of from half to a teaspoonful, given at the same intervals, do a great deal of good. The powder may be used in doses of from one-eighth to one grain, four times a day, in ordinary cases.

The liquid extract of euphorbia pilulifera and the compound tincture of benzoin may be given in the following combination:

R Tinct. Benzoin comp. } aa ..... fl. ℥ ss.
Ext. Euphorb. pil Fld. }
Tinct. Nucis Vom. } aa ..... fl. ʒ iii.
Tinct. Capsici }
Chloroformi ..... fl. ʒ ss.
Syr. Senegæ ..... fl. ℥ i.
Syr. Tolu, q.s ..... fl. ℥ iv.

M. Sig. One teaspoonful four times a day.

The inhalation of a few drops of chloroform, as advised above, or of the fumes of camphor, tend to quiet cough. In cavities, bronchiectasis, or bronchorrhœa, carbolic acid, or creosote is to be inhaled through the respirator.

Cough may be excited by congestion or inflammation of the pharyngeal mucous membrane, and by the elongation of the uvula to such an extent that it touches the base of the tongue. Astringent gargles of tannic acid, sugar of lead, or the spraying of the pharynx with Dobell's solution, or with a weak solution of cocaine, or excision of the uvula are the local measures which must be resorted to.

Excessive Night-sweats. Night-sweats should always be checked, if possible. A very effective measure for the suppression of this drain is sponging of the body with dilute aromatic sulphuric acid, quinine and water, in accordance with the following formula:

℞ Quininæ Sulph..... ℥ ss.
Acid Sulphuric Aromat..... fl. ʒ iv.
Aquæ Cologniensis, q.s..... fl. ℥ vi.
M. Sig. Sponge the whole body morning and evening.

Another useful antihydriotic is oxide of zinc, given in a capsule in five-grain doses at night. Atropine in $^1/_{150}$ grain, and picrotoxine, in $^1/_{40}$ grain doses, are sometimes found of benefit. Another method, which is based on the principle that the sweats are due to exhaustion, is to feed the patient after midnight with fresh beef juice, milk, malted milk, egg-nog, or soup. This should always be tried. Sulphonal, in three-grain doses, in the mornin gand evening, is very effective in a large number of cases.

Irritable Heart. This annoying symptom is not always readily subdued. Digitalis, strophanthus and caffeine may be given, but the best measure which has been found by the author is the application of an ice-bag directly over the heart, and which is kept there from morning until evening.

Dyspnœa. This frequently becomes a distressing symptom of phthisis, especially in the fibroid variety. Nitroglycerine, in the following combination has been found efficacious in this condition:

℞ Sol. Trinitrin (1 p.c.)..... ..... gtt. L.
Tinct. Strophanth.
Tinct. Nucis Vomicæ
Ext. Euphorb. pil. Fld. } aa..... fl. ℥ ss.
Syr. Senegæ
Liq. Ammon. Acetatis..... .... fl. ℥ i.
Syr. Acidi Hydriodici, q.s..... fl. ℥ iv.
M. Sig. One teaspoonful every three hours.

When the dyspnœa is very persistent and stubborn, and interferes with sleep, a hypodermic injection of a quarter of a grain of morphine is very serviceable in relieving it.

Laryngeal Tuberculosis. This is always an unwelcome factor in the development of phthisis, and it should be watched with more than ordinary care and attention. The most important indication in its treatment is to give the affected organ as much rest as possible. Both phonic and whispering speech should be curbed as far as this is practicable, and the patient should limit his communication chiefly to the language of signs and of writing. This should be continued for months. Cough should be alleviated, and if this cannot be done the question of the advisability of tracheotomy comes up for serious consideration. Irritability of the larynx produced by the inhalation of damp or cold air is frequently allayed by wearing a respirator charged with a weak solution of carbolic acid. Smoking, or sitting in an atmosphere laden with tobacco smoke, or dust should be avoided. Local applications to the larynx of a twenty-per-cent. solution of lactic acid is very useful. Dr. Bennett* speaks very favorably of the application to the affected parts of a solution of nitrate of silver, varying in strength from one scruple to one drachm to the ounce of distilled water.

In cases of great difficulty in swallowing the following will prove of some service: ℞. Cocainæ Hydrochlorat., gr. vi.; Morphinæ Sulphat., gr. i.; Codeinæ Sulphat., gr. ij.; Pulv. Glycyrrhiz, q. s. ft., Tab. No. XVI. Sig. Dissolve one tablet slowly in the mouth, every two or three hours. The inhalation of watery vapor impregnated with the compound tincture of benzoin is of great value in relieving cough and laryngeal excitability. When the difficulty of swallowing fluids is marked the following method†, which is recommended by Wolfenden, may be adopted: "The patient lies on a couch, stomach down, and with the legs elevated, and

* "Pulmonary Tuberculosis," John Hughes Bennett, M.D., F.R.S.E., 1854, p. 127.

† "Year-Book of Treatment for 1888," p. 307.

sucks, by means of an India-rubber tube, fluid from a tumbler held in his hand."

Meningeal Tuberculosis. The treatment of meningeal tuberculosis is at best often a hopeless task. Application of leeches to the nape of the neck is of some service, but by far the best measure to reduce local irritability is to surround the head constantly with two or three large, flat ice-bags.

Leucorrhœa. This is a frequent symptom of phthisis, and should receive prompt medical attention. Sometimes vaginal irrigation with simple hot water, by means of a Davidson or a fountain syringe, morning and evening, will correct the discharge. The following combination is also useful:

℞ Zinci Sulphat..... ℥ ii
Plumbi Acetat..... gr. v.
M. Ft. pulv. No. XX. Sig. Dissolve one powder in a pint of warm water and use as a vaginal injection twice a day.

When the discharge refuses to yield to the treatment here outlined, other and more special measures must be resorted to.

Fistula in Ano. A patient with this lesion should always and at once be referred to the surgeon for appropriate interference with the same. It is poor policy to allow it to persist, in the vain hope that it may have some derivative influence on the pulmonary disorder.

Bed-sores. There are very few cases of phthisis that do not, toward the end of the disease, suffer from bed-sores, or ulceration of the soft parts of the back, especially in the regions of the hips and sacrum. These parts should, therefore, be carefully watched, and means be taken to counteract this tendency. Placing the patient on a spring, or water-bed, is a useful measure of prevention. Another is to frequently bathe the back with brandy alone, or with alcohol and water, one part of the former to two of the latter, and in either of which is dissolved some tannic acid. A circular air-cushion, or a cushion made of down, or of cotton, is a

useful appliance for resting the affected part. A thick piece of felt, such as is used to cap the heads of piano keys, with a hole the size of the sore cut out of the center, and maintained in position by adhesive strips, is a serviceable application.

CHAPTER XVII.

CLIMATIC TREATMENT.

THE subject of the treatment of phthisis by change of climate is in rather an empiric state, although, on the whole, there are some pretty well-defined principles which may be followed in the selection of a suitable climate for the various stages of this disease.

In subjecting a phthisical patient to climatic treatment it must be remembered that under all circumstances an antagonism exists between man and his environment, although through a long series of actions and reactions a gradual adjustment has taken place btween the two, and this means that he attains the best health and the longest life in the climate in which he and his ancestors have been accustomed to live. (See p. 212.)

Now, when we come to look for the special physical elements in man's surroundings to which he has become adapted, we shall find that these consist principally of temperature, dryness, humidity and altitude, and these will be passed briefly in review.

Temperature. It is well known that the human body can, with very little detriment to itself, be gradually exposed to a remarkable degree of either cold or heat. Explorers of the far north have frequently been exposed to a temperature varying from thirty to seventy degrees below zero, while, on the other hand, individuals have been subjected to a temperature higher than that of boiling water. In exposures of this kind a large amount of energy has to be expended by the body in order to preserve its uniform range of tempera-

ture. In the Arctic explorer this energy is chiefly devoted to the purpose of upholding the process of oxidation and of supplying the excessive amount of heat which is drained and subtracted from the body by its cold environment. In the case of exposure to a high temperature the layer of heat which is in immediate contact with the body is converted into work, *i.e.*, it dilates the blood-vessels in the skin, quickens the circulation, calls forth and changes into vapor a large quantity of perspiration. It is a fundamental principle that heat cannot do two things at the same time, and hence a special layer of heat in contact with the body cannot raise the temperature of the latter while it is engaged in vaporizing the sweat, and in doing other work. For this reason the body is preserved from immediate mischief, but it is also obvious that exposure of the body to a degree of heat or cold other than that to which it has been habituated entails a great waste. This waste is counterbalanced, however, by constitutions which are sufficiently strong to react on it. This is particularly true of cold, which, at a low degree, has a profoundly depressant action. On the other hand, this agent is a powerful stimulus in a moderate quantity. As such it contracts the blood-vessels, augments the flow of blood and increases muscular, nervous and mental activity. It is like the spur that is applied to the lazy horse, or the cold douche, or the plunge-bath to the body. Its first effects are painful and unpleasant, but it calls forth the latent or reserve energy, and is exhilarating and healthful in the end.

Dampness. The effects of dampness have been discussed on page 216.

Altitude. It is probably true that no other element in our environment has greater power to modify bodily structure more palpably than mountain elevation. This influence pertains especially to the respiratory organs, and is principally, if not entirely, exerted by the thinness or attenuation of the atmosphere and by a diminution of air pressure on the out-

side of the body. It is estimated that at an elevation of six thousand feet the air contains about twenty-five per cent. less oxygen that it does at the seashore, and that the body is relieved of nearly seven thousand pounds of outside pressure. Hence ascension into a rarefied environment accelerates the pulse from fifteen to twenty beats per minute, quickens the respiration in order to obtain the required amount of oxygen, increases the evaporation from the skin and lungs, and diminishes the amount of urine. Protracted residence in such a region enlarges the chest-capacity to a marked extent. The Quichua Indians, who dwell on the elevated table-lands of Peru, have enormous-sized chests, which are out of all proportion to the sizes of the individuals. Dr. Denison says that children born in the Rocky Mountains have chests of unusually large capacity, and M. Jaccoud observes that at St. Moritz the respirations are not only more frequent but more full.

Having briefly considered the physiologic effects of the various climatic changes it is clear that the whole question of submitting a consumptive to a change of climate depends largely on the degree of vigor which he retains. For, as has already been said, acclimatization is an effort on the part of nature to readjust the relation between the body and its surroundings, and this implies the expenditure of an extra amount of bodily energy. The tangible effects of this interior work frequently show themselves at first in nervousness, irritability, sleeplessness, rise of temperature, etc. It is very evident, therefore, that a consumptive should not begin life in a region of this kind unless he has sufficient reserve power to ward off these evil tendencies.

Now, from what has been said, it is obvious that all consumptives with much destruction of lung tissue, combined with serious constitutional disturbance and defective energy, should remain or be placed in a climate which requires as little readjustment as possible. They cannot endure the

climatic demands of a high altitude, and hence their proper place is either at home or in the tropics, where the unfriendly action of the climate is reduced to a minimum.

Two important factors enter into the problem of sending very sick consumptives to a distant climate. The first is money and the second is the wear and tear incidental to a long journey. Want of the first, of course, debars the indigent from any advantage of such a change; but, on the other hand, it is a grave question whether any consumptive of this class should undergo the fatigue and enervation of travel which may overbalance all the possible good that can come from such a transition. Inconsiderate changes of this character are chiefly responsible for the high death-rate which prevails in many valuable resorts for victims of this disease.

In order to facilitate the selection of climate for any particular class of consumptives that may receive benefit, climates, as a whole, may be divided according to the therapeutic properties which its several parts possess. Thus, first, we have the altitude climate; second, the marine and inland climate of warm countries; third, the warm climate of the dry, inland plains, and, fourth, the climate of the ocean.

Altitude Climate. The therapeutic action of this climate is that of a strong stimulant to the whole constitution, and especially to the nervous system. The air is cold, dry and attenuated, and is markedly invigorating. Consumptives in the incipient, or in a tolerably advanced stage of the disease, after having extracted all the possible benefit from their domestic environment, are often decidedly amenable to its reconstructive influence.

Such a climate is found in Colorado, Arizona and New Mexico; as in Denver, Colorado Springs, Manitou Springs, Poncha Springs, Phœnix, and Santa Fé; in the Alps of Switzerland, as in Davoz, St. Moritz and Maloja; in the Andes of South America, as in Bogota, Quito, Arequipa,

Huancago and La Paz; in the Himalayan Mountains, as in Simla, Landour and Subathoo; and in the South African Mountains, as in the Orange Free State and Transvaal.

Marine and Inland Climate. Such a climate is sedative in its influence, and is suited for patients whose recuperative power is more limited than that of the last class, *i.e.*, patients who are persistently weak, with poor appetite, rapid pulse, weak heart, and perhaps quite a rise in the evening temperature. A climate of this kind is found along the western coast of the Gulf of Mexico, as in Ocean Springs, Biloxi and Pass Christian, in Mississippi; Santa Barbara, Los Angeles, San Diego and San José, in California; Madeira, Egypt, etc.

Warm Climate of the Dry, Inland Plains. This climate is tonic and simulating, to a less degree, however, than that of high mountains, and is also adapted to the wants of the last class. It is found in the plains of Texas, as in San Antonio; and in those of Australia.

Ocean Climate. This is sedative in its action, and is most suitable for those consumptives who are rather advanced in the disease. Besides its soothing and quieting influence its greatest advantage lies in the fact, perhaps, that it forces perfect physical rest on the patient. He can recline in his easy-chair all day and be bathed in plenty of sunlight and in warm, balmy air. An ocean voyage for a consumptive should be confined to the waters of warm latitudes as much as possible, and the depressing effects of tropical areas should be avoided. The most effectual way to accomplish this end is to sail from one part of a warm country to another. When this cannot be done, a voyage in a sailing vessel from San Francisco to Australia or to the Philippines and back, will be of equal utility. Any trip on the sea, to be beneficial, must be prolonged, but the confinement, the monotony and the lengthy absence from home, which are incidental to this requirement, may become serious drawbacks to its ideal fulfilment.

THE DEPORTMENT OF CONSUMPTIVES WHILE UNDERGOING CLIMATIC TREATMENT. A very important point to be determined is as to the conduct of the consumptive while he is undergoing climatic treatment. The solution of this question might be wisely left to the physicians living at the various resorts that are visited, were it not for the fact that the majority of those who go there do not put themselves under close medical supervision, and prefer to act as their own advisers. Now what are such persons to do? Shall they go about indiscriminately? Shall they take abundant exercise, or shall they keep quiet? Shall they go horseback-riding, skating or hunting; or shall they undertake tedious or perilous journeys among the mountain peaks?

Dr. Hermann Brehmer, the founder of the famous institution for the treatment of consumption at Görbersdorf, who devoted the greater part of his long and useful life to climatic treatment of the consumptive class, and whose opinion has been previously quoted, says* on this topic that even in health each individual is not the same to-day as he was yesterday; that which is easy for him to accomplish to-day may be a burden for him to-morrow. That is true in the highest sense when applied to the invalid. Here exercise must be constantly and guardedly adapted to the strength and condition of the patient. That which is to me of fundamental importance is to strengthen the heart gradually through exercise without fatigue, and to protect the lungs from undue activity. The amount of exercise which is necessary for him can, however, only be determined by the patient after he is taught to guard himself continually against every physical strain and fatigue. Therefore, on the day the patient arrives he receives the following instructions: "Remain in the open air of the surrounding park. Commence to walk on the level, and regulate your exercise

* "Mittheilungen aus Dr. Brehmer's Heilanstalt für Lungenkranke," 1889, p. 45.

so well that on retiring in the evening you are free from any conscious reminder that you walked at all. To accomplish this he must only walk a short distance at a time, and must always anticipate fatigue by resting. His line of conduct, therefore, must be opposite to that which is pursued by the healthy individual. The latter rests when he is tired, while the consumptive rests before he is tired—rests in obedience to orders, so he may not become tired. At the same time I insist that he must not walk aimlessly, *i.e.*, he must not endeavor to reach a certain point in a certain time, because in doing so he may tire himself before he gets there. I also urge the necessity of making abundant use of the benches which are distributed throughout the park. Furthermore, he must understand that he is to walk slowly, because quick walking produces shortness of breath, causes violent action of the heart, and throws extra work on the lungs."

Dr. Volland, of Davos Dörfli, makes the following statement* concerning the deportment of consumptives while undergoing treatment in mountain resorts: "In the early stages of treatment rest in the open air is of paramount importance."

In 1892 the late Dr. John M. Keating, of Philadelphia, read a paper† on rest in the mountain treatment of phthisis before El Paso County Medical Society in Colorado, and in the discussion which followed there was scarcely a member, most of whom had a large experience in dealing with patients of this class in that altitude, and had, therefore, an unquestionable right in expressing an opinion on this subject, who failed to approve of the rest cure in this disease. The following quotations are typical of the general trend of the discussion: Dr. B. F. D. Adams said: "I have seen enough to have a

* "Behandlung der Lungenschwindsucht im Hochgebirge," p. 18.

† "The Rest Cure in the Treatment of Incipient Phthisis." Published in the *Climatologist*, January, 1892.

few facts deeply impressed on my mind. One of these is the importance of rest in all cases of advancing phthisis and another the natural converse of this, that there is nothing so injurious to the consumptive as over-fatigue. I have had to unlearn much that I thought I knew about phthisis since living in this altitude. The natural history of the disease is, in some respects, different from that in the East. I strongly suspect that over-fatigue is more injurious than at the sea-level, and I am sure that the danger-point is more quickly reached at this altitude. When there is fever there should be rest, but unfortunately we constantly see patients here impressed with the one idea of exercise in the open air, doing themselves irreparable injury by horseback-riding and other violent exercise, when the clinical thermometer should send them to bed."

Dr. Tucker: "When exercise is ordered or permitted, it should be impressed on the mind of the patient that any exercise, however slight, which is followed by rise of temperature, loss of appetite, or a condition of fatigue from which recovery is not rapid, is too great."

Dr. N. A. Campbell: "I feel it a physican's duty in incipient-stage cases, especially in those attended with pyrexia, to insist upon the patient being quiet, and take but little exercise."

Dr. James A. Hart: "I believe that we should take advantage of every circumstance to encourage rest in the majority of cases."

Dr. Keating in closing said he had noticed that nearly all the patients who came to Colorado Springs and neighboring resorts have received the advice to buy a horse and ride it, that camping out seems to improve every one of them, and that they are advised before they come here to take active measures from the start. The object of his paper was to determine whether it was or was not the sentiment of the meeting that people coming here with phthisis should take active exercise at once.

CHAPTER XVIII.

NURSING OF CONSUMPTIVES.

THE treatment of consumption without competent nursing is futile. Because it is a chronic disease with few abrupt changes in its general downward course, it is the general belief that all it requires is air, food, exercise and cod-liver oil. The truth, however, is that drugs and attendance to the general wants of the patient play but a very small part in the work which is to bring about his restoration. The one essential part in his treatment consists, as has been stated already, in placing him under constant and watchful supervision. A supervision which is a school of discipline in which he is educated to a new life—a life which is different from that which led him into his present difficulty. Nursing is, therefore, the work of a conscientious attendance to details, a task in which little things are of greater importance than great ones.

The following rules are laid down for the guidance of those who have consumptive patients in their care:

On account of dampness of the lower floor of a house, a consumptive patient should not occupy a room lower than the second floor. If possible this should have a southern exposure during the winter.

Heating and ventilation should be well regulated. A uniform temperature of 70° F. should be maintained. Attention should be paid to the proper moisture of the air. If it becomes too dry it causes cough. This is more frequently the cause of cough than is suspected. A bowl of water placed near the patient, or a few towels saturated with

water, suspended around the room, will obviate this difficulty. The room should be furnished neatly and plainly. No superfluous furniture should be allowed.

The sputum should be expectorated into a cup containing a disinfectant. For this purpose Platt's chlorides as a disinfecting agent are very useful.

The temperature, pulse and respiration should be taken at least at 8 A. M. and 6 P. M., and sometimes at 2 P. M. additionaly, and in exceptional instances in which the temperature fluctuates irregularly it should be taken every three hours during the day.

Unless the patient perspires too freely the underclothing should be worn day and night, without being changed, for three or four days. This is to be done for the purpose of maintaining an equable temperature on the surface of the body. The underclothing should be of woolen texture, except in the summer, when it may be changed to silk, or to a mixture of cotton and wool.

During the time the patient is confined to bed his body is to be sponged thoroughly with warm water and soap or ammonia. When he is able to be about he is to take a bath in lukewarm water every week. No cold plunge or shower-baths are to be indulged in, except under strict orders of the physician as to the degree of temperature, and the length of time that it is to be applied.

Cough is a necessity, but if left to itself it is frequently futile in its effects, and may become a very annoying habit. Patients should, therefore, be taught how to cough so as to expel the greatest amount of expectoration with the least expenditure of force. In order to do this, coughing should, in a measure, be controlled by the will. The patient should be taught not to cough for every slight tickling or irritation that may happen to occur in the throat. After the cough is brought somewhat under the power of the will, the chest is to be filled completely with air and emptied with one ex-

pulsive effort. This should be repeated until the offending catarrhal material is expelled.

When coughing is painful the painful spot should be supported by the hand, or the chest should be tightly bandaged with a towel, or be made immobile with adhesive strips.

Cough is provoked by cold bed clothing or by the inhalation of cold or damp air. In the case of the former the clothing should be warmed before retiring, and in that of the latter a respirator should be worn in the open air.

Patients are very much less liable to cough when they lie on the well than on the affected side. In troublesome cough it is always a good rule to make the patient recline on that side of the body on which the normal lung is located.

When a cavity exists, cough often persists in the morning until its contents are cleared out. This operation may be very much facilitated by teaching the patient to bend his head as low as possible, just as if he were in the act of tying his shoes; or, if he is confined to bed, he should lie on his stomach, depress the head as low as possible, and at the same time elevate the lower portion of the trunk. Coughing in this position gives the law of gravity a chance to assist in expelling the fluid accumulation in the cavity.

A simple irritable cough, such as often occurs when the patient assumes a recumbent posture in bed at night, or from any other transient cause, may be quieted by a drink of wine, or whiskey or brandy, or by the inhalation of the fumes of gum-camphor, or of from five to ten drops of chloroform from a handkerchief, or by the spraying of the throat with a weak solution of cocaine.

The bowel movements of a consumptive should, if possible, be under control, and not occur oftener than once a day. Any tendency to looseness should be restrained either by washing out the bowel with a large quantity of warm water every day or every other day, and, if this fails, by the administration of camphorated tincture of opium in tea-

spoonful doses every hour or two, or by the application of a mustard plaster to the abdomen, twice daily. Sometimes the bowels may be checked through the influence of the patient's will-power. He should always be encouraged in exercising this restraining power.

In case of high fever the patient's body is to be sponged with cold water, with the addition of cologne, alcohol or vinegar. Application of the ice-bag over the affected lung is also a useful measure to reduce fever and may be used with good effect as a febrifuge.

In case of hemorrhage from the lungs the patient is placed at once on his back, a quarter of a grain of morphine is given to him hypodermically and one or two ice-bags are applied over the affected lung area. The ice-bags should be retained in position for a number of days, and until all danger of subsequent hemorrhage is passed.

Pain in the chest is often relieved by applying mustard, capsicum or horseradish plasters over the painful spot. A coat of iodine sometimes accomplishes the same purpose. When the pain is of a stitchy character, and comes with each deep inspiration, a towel should be pinned tightly around the chest.

The patient should be weighed every week at regular intervals, with the same amount of clothing, and at the same hour each time.

After a patient has been confined to bed constantly for weeks and, perhaps, for months, it is very important to guard him against making the transition to the sitting up position, or to walking, too rashly. On the first day when the change is made he ought to be allowed to sit up no longer than from fifteen minutes to half an hour. This time is to be gradually extended as he gets stronger. Whether he sits, stands or walks he should lie down before he feels very tired.

CHAPTER XIX.

HEMOPTYSIS.

DEFINITION. Hemoptysis simply means spitting of blood. It must be remembered, however, that the blood which may be spat can come from the lips, gums, pharynx, nose, larynx, lungs, etc., and as this chapter will be devoted to a consideration of bleeding from the lungs it is quite evident that the terms pulmonary hemorrhage or pneumohemorrhagia would be more accurate and appropriate in defining this affection, inasmuch as they at once locate the source of the blood. But custom has so far sanctioned the use of the word hemoptysis in connection with this subject that the author has seen fit to retain it on this account.

SYNONYMS. Pulmonary hemorrhage, Pneumohemorrhagia, Blood spitting, Blutspucken (German), Hemoptysie (French).

SOURCE OF THE BLOOD IN HEMOPTYSIS. . .

From the consideration of the anatomy of the pulmonary circulation given in former pages it is quite clear that the blood in blood-spitting is more liable to originate in the pulmonary than in the bronchial vessels. In spite of this apparent greater natural liability there is a division of opinion on this subject. Laennec held that the bronchial surfaces, and hence the bronchial blood-vessels, were chiefly involved in hemoptysis. In his work "On the Chest,"* (p. 132) he says: "It can no longer admit of question, in the present

* Edition 1830. Translated by Dr. John Forbes.

state of medical knowledge, that the greater number of cases of slight or moderate hemoptysis consist in the simple exhalation of blood from the bronchial membrane, while the severe cases originate chiefly in the vesicular structure of the lungs and constitute the affection which will be noticed hereafter, under the name of "pulmonary apoplexy." However, after referring to the fact that he had already expressed the view that slight blood-spitting comes from the bronchial mucous membrane, he states (p. 188) that those cases "of violent and extreme hemorrhage, which often resist all medical treatment, arise from a very different and more dangerous cause. In these some part of the pulmonary substance has undergone great changes, being indurated to a degree equal to the completest hepatization. The induration, however, is very different from the inflammatory affection of the lungs distinguished by this term."

From Laennec's further consideration of the morbid condition, which he believes underlies the source of the blood, it seems doubtful whether it is one of pulmonary apoplexy or of phthisis. Still greater doubt arises on this score when he describes the symptoms of the hemoptysis which accompany the latter condition, for he says (p. 191) "the spitting of blood is the most constant and most severe. This is commonly very copious, returning by fits, with cough, oppression, anxiety, intense redness or extreme paleness of the face, and coldness of the extremities. When the hemorrhage is very great it comes on sometimes with a very moderate degree of cough, and is accompanied by a convulsive elevation of the diaphragm, like that which takes place in vomiting. . . . The quantity of blood expectorated is sometimes enormous. I have known ten pounds lost in forty-eight hours by a young man, who died under the hemorrhage. In cases of less acute character I have seen about thirty pounds lost in a period of fifteen days."

This is indeed an accurately drawn picture of the hemop-

tysis which so frequently happens in pulmonary phthisis, even in its incipiency, as well as in acute pneumonia, especially in that form of the latter disease which is known as grippe-pneumonia. Surely the blood in these instances does not come from the bronchial tubes, although as Laennec says these structures may be a little softened and impregnated or tinged with blood throughout their entire depth (p. 132). While, therefore, this distinguished authority may not be exactly clear regarding the anatomy of hemoptysis it is certain that he is very accurate in the delineation of its clinical manifestations.

The fact that blood is found in the bronchial tubes is not very good evidence that the blood originates here. For Dr. Reginald E. Thompson* says that he "examined many cases of fatal hemorrhage in which the blood has been traced to a pulmonary vessel eroded, or in a state of aneurism, to the bursting of an aortic aneurism into the trachea; in all these cases blood was found in the bronchi; moreover, it is difficult to determine by inspection of the condition of the bronchial tubes from which lung the blood has started in cases of pulmonary hemorrhage, although the actual source may be subsequently determined: it can then be positively asserted that the presence of blood in the bronchial tubes does not establish the existence of bronchial hemorrhage; it shows, in a remarkable manner, the tendency of the inspiratory force to translate matter from one lung to the other, or from one part to another." On the same subject Walsh says† that "setting aside those instances—mere curiosities from their singularity—in which ulcers in the bronchial tubes, or plastic bronchitis, furnish the blood of hemoptysis, I have never yet seen a case where blood, discharged in any quantity during life, either seemed, from the nature of the case clinically or was proved on inspection of the bronchial tubes,

* "Pulmonary Hemorrhage," p. 10.

† "Diseases of the Lungs," p. 393.

to have come from their substance by molecular ruptures or by exhalation."

It may be concluded, therefore, that whenever blood is raised in any quantity from the lungs it almost invariably comes from the pulmonary capillaries, although there is a possibility of its coming from the bronchial vessels. With this view the author's experience coincides entirely. This deduction also accords with that which would be made from a reasoning based on the anatomical differences of the two systems of blood-vessels.

Pathology.

Since the pulmonary blood-vessels are chiefly involved in most cases of hemoptysis it becomes of great interest to inquire whether the latter depends primarily on diseases of the lungs or on disease of the blood-vessels themselves. In other words, can disease of the pulmonary capillaries exist independent of and lead to disease of the surrounding pulmonary structure, or can it not? Of course in this connection it must be recognized that, according to some authors, hemoptysis may always become the indirect cause of pulmonary disease, *i.e.*, some of the blood may remain behind or be aspirated into fresh parts of the lungs and, by decomposing, establish new foci of disease; yet at the same time the important fact must not be lost sight of that in the great majority of instances blood-spitting is not only a symptom but a part of the disease itself.

Disorder of the pulmonary blood-vessels being, therefore, the fundamental lesion in hemoptysis, it is in order to inquire into the nature of the cause or causes of the defection in the blood-vessels. Is it due to an increase of blood pressure within, or is it caused by a weakness or disintegration of the wall of the blood-vessel itself? That a simple fulness of or increased pressure within the pulmonary capilla-

ries does not of itself readily lead to hemoptysis is evidenced by the fact that in acute congestion or inflammation of the lungs, in which the blood pressure is certainly much increased, hemorrhage is rather an infrequent occurrence.

Laying aside all other anatomical influences there is no structure that is more closely interwoven with the life of a blood-vessel than its supplying nerve. Its caliber, its nutrition, and its power to retain its contents depend in a great measure on the influence which comes from its nerve element, and hence we must not, in seeking for possible causes of hemoptysis, ignore the part which these, the vasomotor nerves of the lungs, play in this pathological process. In a very lucid paper* on hemoptysis, in which he discusses this particular phase of this subject, Dr. G. M. Garland, of Boston, Mass., uses the following language: "There is one suggestion regarding pulmonary hemorrhage which has been advanced by Virchow and others, but which has received less attention from clinicians than it deserves. I refer to the influences of the nerve-centers. Virchow says: 'If we cannot deny, therefore, the greater frequency of hemorrhage by certain weather changes, yet it appears that here also a contributing cause (Wallungszustände†) must be sought, which possibly lies in the nervous system.'" After referring to certain experiments of Cohnheim, which show that intravascular pressure does not of itself produce hemorrhage, he continues: "Admitting these facts, we ask if similar changes in the condition and resistance of the vascular walls can be wrought by any perversion of nerve influence alone, and we receive an immediate answer in the affirmative. I need only refer to a few illustrations. We are all familiar with the ecchymosis, which forms on the under lid of suicides, when the bullet has pierced a certain part of the brain; Fasians

* "The Pathology of Hemoptysis from Chronic Pulmonary Disease," *Medical News*, August 17, 1889, p. 178.

† Orgasm, excitement, or congestion.

and Kellar describe subcutaneous hemorrhages resulting from great moral commotion in neuropathic persons. . . . . In conclusion, therefore, I would say that I believe that a more careful discrimination of our cases of pulmonary hemorrhage would reveal some of purely neurotic origin."

The line of thought advanced here by Dr. Garland on the neurotic origin of bleeding is corroborated by the following evidence, to some of which he refers in his paper: Von Recklinghausen states* experiments on animals clearly demonstrate that injury to the nervous system brings about hemorrhage into the viscera of the body, and especially is this true of the lungs and the stomach. This opinion is based on the results of the following experiments: Schiff and Brown-Séquard, by injuring the pons and other parts of the base of the brain; Nothnagel, by scratching the convexity of the brain; Vulpian, by damaging the trigeminal nerve; and Bernard by irritating the vagi and the sympathetic ganglion of the neck called forth hemorrhage in the substance of the lungs and in the pleura.

Nor is clinical evidence wanting to show the intimate relationship between diseases of the brain and nervous system, on the one hand, and hemorrhagic lesions in the lungs and their appendages. Dr. C. Handfield Jones, in considering the respiratory neuroses, states† that "if the principles I have laid down are correct, hemoptysis may be included among the neuroses of the respiratory organs. I would not be understood to affirm this in an exclusive manner, as if there were no other causes of this hemorrhage; the only point I wish to maintain is that in certain cases, and these not very infrequent, hemoptysis may be reasonably regarded as a paralytic neurosis of the vasomotor pulmonary nerves.

* "Handbuch der Allgemeine Pathologie des Kreislaufes u. der Ernährung," S. 94.

† "Functional Nervous Disorders," p. 649.

In a fatal case of tuberculous hemoptysis I could discover no special source of the profuse gush which destroyed life; it seemed as if the blood had escaped from the vessels everywhere. This is the general experience of others. When we look at a good specimen of injected lung and add a thin slice of pulmonary tissue we cannot but be struck, on the one hand, with the extreme vascularity of the air-cells and, on the other, with the absence of support to the capillaries compared with those of other organs. In fact, one can hardly help wondering, as a friend once remarked to me, that we don't all die of hemoptysis. Trousseau relates the case of a lady who was subject, when a child, to some somnambulism, and since then to the most *bizarre* nerve disorders. When about thirty years old she had such profuse attacks of hemoptysis and so much dyspnœa that phthisis was suspected, though no physical sign could be detected. Till the menopause occurred she often had alarming menorrhagia. At present her skin flushes very readily, but the above-mentioned symptoms have ceased, and she presents no sign of the presence of tubercles. Andral relates a fatal case of hemoptysis, the lungs of which, at the autopsy, were found free from tubercles. The girl, aged 21, had suffered for a year from violent palpitation, progressive loss of strength, and was put out of breath on the least exertion. These are common signs of a neurolytic condition." Dr. O. Kohts, in describing the effect of fright during the bombardment of Strassburg, reference to which is made on a previous page, says* that diseases of the respiratory apparatus were markedly aggravated, and that hemoptysis often occurred for the first time in the phthisical. In a paper† on unilateral pulmonary apoplexy in hemiplegia, Dr. A. Olivier clinically confirms the well known experiment of Brown-Séquard by contributing three cases of cer-

* *Centralblatt f. d. med. Wis.*, 1873, S. 286.

† *Centralblatt f. d. med. Wis.*, 1873, S. 779.

ebral apoplexy, in which existed apoplexy and congestion of the lungs, as well as sub-pleural ecchymosis on the side opposite to that where the apoplexy was located in the brain. Olivier connects the brain lesion with the pulmonary hemorrhage on the score of irritation of the vasomotor nerves. In a very interesting article* on this subject Dr. L. Fleischman contributes the post-mortem results of two cases of pulmonary and pleural hemorrhage, which co-existed with degeneration of the brain. In an original paper on extensive capillary extravasation of bright-red blood into the pulmonary tissue of the insane, Dr. Jehn, after referring to Nothnagel's experiment, which shows that pulmonary hemorrhage follows irritation of the brain of rabbits, says† that this investigation leads one to anticipate the occurrence of widespread infiltration of bright-red blood into the alveolar spaces of the insane. As a matter of fact, this expectation was verified on the post-mortem table, for he found such extravasation in the alveoli of the bodies of five persons who had been insane. The hemorrhagic foci were scattered over both lungs; some were large and others were small, but the blood was of a bright-red color. The bronchi were empty. Pieces of lung floated under the surface of water. The red discoloration of the lungs was uniform. The areas which were free from extravasation were œdematous and contained pneumonic deposits. Microscopically the alveoli were, without exception, filled with red blood-corpuscles, and in the blood-vessels and lung tissue no morbid change was discernible. The pathologic condition of the central nervous system was partly negative, and in part showed itself in old and new meningeal neoplasms, pachymeningitis hemorrhagica, diffuse redness of a few gyri, and in two cases there was capillary apoplexy of the cortex. Of the five there was one each of melancholia and mania, and three were

* *Jahrbuch f. Kinder Heilkunde*, 1871, 4, S. 283.

† *Centralblatt f. d. med. Wis.*, 1874, S. 340.

paralytics. There was no evidence of lung degeneration during life.

From the foregoing experimental and clinical data it may be concluded then that any disease of or injury to the nervous system, if it implicates the vasomotor nerve supply of the lungs to a sufficient degree, becomes a cause of hemoptysis. So, likewise, any agent or influence which has the power of devitalizing the nervous system in general and the pulmonary nerve supply in particular, becomes a possible cause of hemoptysis.

Causes.

The influences which produce hemoptysis by impairing or by disintegrating the pulmonary nerve supply are, first, a strong neurotic tendency; and, second, the poisons of alcohol, syphilis, rheumatism and influenza. It is also brought about mechanically, as by forced breathing, cardiac disease, aneurism of the aorta, and pregnancy; or by the various diathetic conditions known as hemophilia, general hemorrhagica and vicarious hemorrhage, as well as by parasitic invasion of the lungs. Each of these influences will be considered in the order just given.

Neurotic Hemoptysis. There are certain individuals belonging to the neurotic or hysterical type who are excessively prone to spit blood from their lungs. They are usually burdened with a neurotic ancestry, belong to the female sex, are anemic, dyspnœic, and perhaps weaklings from childhood, and for no very accountable reason raise copious quantities of blood from their lungs without sustaining any marked permanent deleterious effects. Some authors have conceived the idea that a certain degree of antagonism exists between hysteria and phthisis, and hence these patients are regarded as being immune from pulmonary disease. This is a serious error, for by viewing these sufferers in

such a light they are often allowed to drift into confirmed phthisis before their true condition is discovered.

Alcoholic Hemoptysis. Among the toxic agents which have the power of engendering hemoptysis and pulmonary consumption alcohol stands pre-eminent. One of its principal tendencies seems to be to destroy the elasticity of the walls of the capillary blood-vessels. This is found in the mucous membrane of the stomach, where its habitual and continuous use produces a permanent congestion. That it has a similar influence on the blood-vessels of other parts of the body is manifested by the ruby nasal organs and the chronic dilatation of the superficial capillaries on the cheeks of many alcoholics. That it produces the same effect on the mucous surface of the lungs there can be but little doubt. Laennec says* that the abuse of spirituous liquor is an occasional cause of hemoptysis. Examples of such cases occur in the practice of every practitioner whose work lies among the beer-drinking class. Thompson relates† the interesting case of a cellar-man, aged 23, who had been accustomed to drink beer in large quantities. His face was very congested and in a general state of purpura, and his gums were very spongy and bleeding. He had a pimple on his nose, which, when scratched, would bleed for an hour. There was no albuminuria, but two months before admission he had hematuria, and three months before he spat a pint of blood. Both lungs showed abnormal physical signs. Under iron and cod-liver oil he improved and no bleeding recurred. Dr. Austin Flint gives‡ an account of a similar case. The patient was corpulent and an habitual beer drinker. After complaining for several days of some stitch-like pains in the chest, which symptoms were also associated with tremor of the tongue and limbs, he was suddenly seized

* *Op. cit.*, p. 33.
† *Op. cit.*, p. 39.
‡ "Phthisis," p. 85.

with hemoptysis and died within a few moments. He raised nearly two quarts of blood, and a large quantity was found after death in the stomach. The lungs presented no apoplectic extravasation, but they were intensely congested and the bronchial mucous membrane was exceedingly so.

Now, how does alcohol bring about these results? There is good reason for believing, as has been shown already, that it accomplishes this by disintegrating the nervous system, and especially the vasomotor respiratory nerves. The marked effects of alcohol on the nervous system are known by the name of alcoholic paralysis, or alcoholic neuritis—a disease which is characterized by numbness, tingling, hyperæsthesia, anæsthesia, paralysis of motion, quickened pulse, shortness of breath and frequently by pulmonary embarrassment. The late Dr. Anstie, in his article on "Alcoholism,"* states that "the changes which have been observed in the brain, oblongata, etc., of confirmed drinkers consisted essentially of a peculiar atrophic modification, by which the true elements of nervous tissue are partially removed and the total mass of nervous matter wastes. Essentially similar changes are observed in the lungs, the liver, the heart, etc. There is much in these changes which reminds us forcibly of the effects on nutrition of tissues produced experimentally by Schiff and Mantegazza by the section of compound nerves, and suggests the idea that in alcoholic poisoning the starting point of degenerative tissue-changes may consist in paralysis of those nervous branches which preside specially over nutrition, the distinct character of which has been so well pointed out by Brown-Séquard." Sharkey relates† the history of a female, aged 32, who was addicted to the excessive use of alcohol. She suffered from hemoptysis, dyspnœa, diarrhœa and finally died from failing respiration. Both lungs were found to be phthisical, and degeneration of the dorsal and

* "Reynolds' System of Medicine," Eng. ed., vol. ii, p. 165.

† *Trans. Lond. Path. Society*, 1888, p. 27.

cervical regions of the cord, the vagi, the phrenici and other nerves were present.

Syphilitic Hemoptysis. That syphilis has the power of producing pulmonary consumption is substantiated by the fact that many writers on chest diseases recognize a distinct affection of the lungs under the name of syphilitic phthisis. Hemoptysis, abundant and profuse, is frequently one of its constant factors. Dyspnœa is also a prominent element which may become asthmatic or paroxysmal. As a rule, the temperature in syphilitic phthisis is near the normal line and there is not much fever present until there is considerable destruction of lung-tissue. Sometimes the physical signs, as, for instance, the crepitant and mucous râles develop very decidedly in a certain area, and then disappear and reappear in a remarkably short space of time in the same spot. This has been observed by the author in a number of cases.

Syphilis being a nerve-destroyer, there are many reasons for believing that this poison, like alcohol, produces hemoptysis and pulmonary consumption through its action on the brain and nervous system, and especially on the cranial nerves, for which it appears to have a special selective affinity.

Rheumatic Hemoptysis. Although there is said to be a natural antagonism between rheumatism and pulmonary consumption the author is convinced that not only no conflict exists between these two conditions, but that the former frequently paves the way for the development of the latter affection. And he furthermore believes that the phthisical manifestation which comes with rheumatism, or follows in its wake, is more predisposed to hemoptysis than when not so associated. A patient suffering with hemoptysis of this kind may be briefly described as follows: Seemingly he is in comparatively good health and without much difficulty, is able to pursue his daily vocation. He probably is, and for

some time has been, tired out and overworked, but this is not considered to be of much importance. While at work, perhaps, a sudden hemorrhage startles and overwhelms him. He is taken to his home, and his physician is called in. A careful examination, so far as this is allowable under the circumstances, probably discloses no physical evidence as to the source of the blood in the lungs. There are present no alcoholism, no venereal taint, and no striking or definite family history of pulmonary consumption. Absolute rest is ordered and morphine is given in connection with gallic acid, lead acetate, ergot, turpentine, geranium, etc. The bleeding may cease and apparently is checked, but by the following night, or a few days afterwards, it may recur with increased severity. Hemorrhage of this sort seems to have a partiality for coming on in the night, although this is not always true. In spite of the treatment which he receives the patient becomes progressively weaker and is in danger of dying from exhaustion, or from an acute or subacute pulmonary inflammation. On close inquiry in the early stage of the affection it would, in all probability, have been found that this patient had been subject to what is known as the rheumatic or gouty diathesis, or that rheumatism or gout existed in his family. He may have had no outspoken pain in his joints at the time of or during the attack of bleeding, but his urine is scanty and probably of a high color, and he may have had some stiffness in his shoulder-joints, or some rheumatic pain, or what is called neuralgia, in various parts of his body during recent years, or such pain may show itself after the hemorrhage has subsided; or, as has been witnessed by the author, a patient may be perfectly free from a personal history of rheumatism and still have a concurrent attack of articular pain and hemoptysis. All cases of this kind are strongly dominated by the rheumatic spirit, belong to the rheumatic class of diseases, and must be promptly treated with anti-rheumatic remedies.

This affiliation between hemoptysis and articular disease had been recognized by Sir Andrew Clark as early as 1875, and in an article* on the non-tubercular and non-cardiac hemoptysis of elderly persons he says: "Since 1875 he has seen some twenty cases of hemoptysis in elderly persons, which were free from either tuberculosis or heart disease, and in which the ordinary methods of treatment failed. Some seven years ago he, with Sir William Jenner and Dr. Wilson Fox, was summoned to consult about a lady suffering from an incoercible hemoptysis. She was very stout, very rheumatic, had nodular finger-joints, frequently recurring bronchial asthma and occasional outbreaks of either eczema or of urticaria. Ten days before our visit, when suffering from an ordinary catarrh without accompanying fever, the patient began to cough up blood, and had continued to do so, in small quantities, at intervals of three or four hours since. She had a heart somewhat large, but there was no murmur and no evidence of systemic arterial disease. Her temperature was nearly 100° F., her pulse quick and frequent, and there were signs of a generalized bronchial catarrh, of emphysema and of basic congestion. She complained of frequent cough, of great oppression of the chest, and of growing difficulty in expectorating. She had, furthermore, a loaded tongue, thirst, loss of appetite, a swollen liver, and all the signs of a gastro-enteric catarrh. She had been carefully treated by absolute rest, fluid food, ice to the chest, and in succession by lead, gallic acid and hypodermic injection of ergotin. After consultation it was ordered that she should have a light and rather dry diet, to be sparing in the use of liquids, to discontinue the ice, to have a calomel pill at night, followed by a saline cathartic on the following morning, and to take an alkaline mixture, with ammonia,

* See *Lancet*, Oct. 26 1889; *British Med. Journal*, Oct. 26, 1889; *Phil. Med. and Surg. Reporter*, Nov. 30 1889; *Boston Med. and Surg. Journal*, April 10, 1890.

between meals, twice a day. In thirty-six hours the bleeding had ceased and she made a speedy recovery.

He describes another case in full, which is equally interesting in its history and results of treatment, and after referring to a number of similar instances, of which the two he details are typical, he gives the following post-mortem appearances of the first case in his experience: The patient, a man, lived to an age of 50 or 60, had moderate progressive osteo-arthritis for many years, and for four or five winters before death suffered from severe bronchial catarrh. The hemoptysis from which he suffered failed to yield to absolute rest, combined with ice to the chest and a liberal use of astringents. The autopsy revealed no tubercular disease, malignant growth, or any sort of coarse structural change which could account for the fatal hemorrhage. The bronchial mucous membrane almost everywhere was swollen and congested, violet in color, and coated with muco-purulent secretion. The anterior parts of both lungs were pale, dry and emphysematous, and curious, isolated patches of emphysema surrounded by hemorrhagic extravasations were noticed in the back and lower parts of both lungs, which were loaded with blood. The microscope showed the seat of hemorrhage to have been in the immediate neighborhood of the emphysematous patches, and that the minute vessels, chiefly the terminal arteries, were, in these localities, always diseased, the changes being similar to those found in the diseased articulations of the arthritic diathesis.

Now in what especial manner does rheumatism bring about hemoptysis? This is probably accomplished through the agency of uric acid which, according to the researches* of Dr. Haig, is active in promoting many prominent disorders of the nervous system. He shows that uric acid is a nerve poison, and hence there is good reason for believing

* "Uric Acid in Causation of Disease," London: J. & A. Churchill, 1896.

that this agent, in common with alcohol and syphilis, produces disintegration of the pulmonary capillaries, and as a sequence hemoptysis, by impairing the integrity of the pulmonary nerve supply.

Symptoms. Among the symptoms which distinguish rheumatic hemoptysis from other forms of pulmonary hemoptysis are (1) articular pain or stiffness in some of the joints; if these are absent there exists, in all probability, a family history of rheumatism or of phthisis, most likely the former, in connection with the latter. (2) Pain in one side of the chest, which may or may not be of pleuritic origin. (3) Aching or numbness in one or both forearms. This may be confined to the arm of the same side on which the chest pain is located. (4) The bleeding is either copious or slight, and in the latter event the expectoration is protractedly discolored or streaked. (5) The cough and expectoration are comparatively light. (6) Dyspnœa, as a rule, does not exist in an aggravated form, although in some cases it is very pronounced. (7) Fever, as a rule, is low, or absent, except at the beginning of the attack, when it may be very high. (8) In some cases there is pain in the region of one kidney which radiates towards the bladder and which may be accompanied by vesical and urethral irritation. (9) There is often a diminished amount of urinary secretion. (10) In some instances the author has seen this condition associated with appendicitis. (11) The disintegration in the pulmonary organs, as shown by the physical signs, is out of all proportion to the degree of constitutional exhaustion and depression.

Mechanical Hemoptysis. Hemoptysis may ensue from causes which lie exterior to the lungs and which have the power of increasing the blood pressure within the pulmonary capillaries. This occurs in cardiac lesions, especially in disease of the mitral valves. Under these circumstances the blood circulates imperfectly through this valve, dams up in

the left auricle and pulmonary veins, and its increased pressure is thrown back on the pulmonary capillaries, which break under the strain. Aortic regurgitation may lead to the same end.

Another source of hemoptysis is the strain which comes on the pulmonary capillaries from forced breathing. The forcible dilatation of the thorax, the sudden inflation of the lungs, the marked distention of the capillaries and the pressure which is exerted during expiration, exposes these delicate vessels to an excessive strain which may readily lead to a rupture of their walls. Such accidents are witnessed sometimes in the consultation-room when a patient, who is undergoing a physical examination, is asked to take a number of deep breaths in succession. It may also be experienced when one ascends into the rarefied atmosphere of a high mountain, or when a patient undergoes the pulmonary gymnastics which is necessary in using a compressed and rarefied air machine. It may also be brought on by severe or excessive athletic exercise.

Pregnancy. Hemoptysis occurs during pregnancy. It is an open question, however, whether the blood-spitting in this condition is caused exclusively by the mechanical interference of the blood flow through the lungs in virtue of the pressure of the enlarged uterus on the important blood-vessels, or whether there are not at work other influences of a diathetic, miasmatic or nervous nature in bringing about this result.

Aneurism. Hemoptysis may be produced by the weeping, oozing or rupture of an aortic aneurism into a bronchial tube.

Vicarious Hemoptysis. Bleeding from the lungs occurs as a result of irregular menstruation, and often without any obvious harm. Sir Thomas Watson gives the interesting case of a woman, in his lectures, who menstruated regularly through her lungs for forty-two years. In females who are

predisposed to pulmonary troubles, or in whom the disease is already active, hemoptysis is more liable to occur at the menstrual period than at any other time.

DIFFERENTIAL DIAGNOSIS. There are very few things which demoralize a patient more than the spitting of blood, and hence the first pressing question that comes from him is, "Does the blood come from my lungs?" In unravelling this question the physician tries to ascertain whether it emanates from some other source, like the nose, gums, throat, stomach, aneurism, etc. If it comes from the posterior nares the color of the blood may not be bright red, and it may be seen trickling into the throat. If it comes from the gums they will be found swollen and spongy, and can be readily made to bleed. It may come from varicose capillaries in the pharynx, larynx and trachea, and if so this can readily be determined by an examination with reflected light, but, as a rule, blood comes very rarely from these structures unless it is brought forth by traumatism. If it comes from the stomach it is of a dark color and grumous, and often mixed with particles of food. Then, too, it may also appear in the stools. The blood from an aneurism gushes out of the mouth and nose, and is of a scarlet color, and in many instances proves speedily fatal. This is not always the case, for the blood of an aneurism may slowly and irregularly ooze into the lungs for months before the final rupture occurs.

Now blood which comes from the lungs is, in the great majority of cases, at the onset of a bright red color and generally accompanies the act of coughing. Sometimes when very copious it may be of a darker color, indicating that it may come from a branch of the pulmonary artery. It may also be dark when it comes from a cavity where it has had the chance of accumulating before being ejected. Then, too, hemoptysis is sometimes preceded by a tickling in the throat, by dyspnœa and by pain and oppression in the chest. The

physical signs are frequently disappointing, for every objective trace of a copious pulmonary bleeding may be wanting entirely, or, if present, may vanish from the lungs in an incredibly short time. It must always be remembered, too, that alcoholism, syphilis or rheumatism specially predispose to hemoptysis.

PROGNOSIS. The prognosis of hemoptysis is usually favorable if the blood does not proceed from a large pulmonar cavity or from an aneurism. Indeed it is often the case tha blood-spitting removes the fulness and oppression in the chest and is, therefore, a benefit rather than an injury. This is surely many a time the case when the hemoptysis depends on a valvular lesion of the heart, and it is by no means infrequent in other forms of pulmonary hemorrhage. The author does not believe, therefore, that hemoptysis is the grave affection which it is generally regarded to be, and holds that, unless the hemorrhage threatens life immediately, it is the positive duty of the physician to allay and to calm the patient's fear concerning the almost universal fatality which is commonly believed to attend the disorder, and to assure him that it frequently acts as vent to the overloaded and distended blood-vessels, whence the blood proceeds. Success in this direction will restore the patient's confidence, and will go far towards warding off evil consequences and enable him to resist the further encroachment of the affection.

TREATMENT. In the management of these cases we must not only bear in mind the bleeding from the lungs, which is the most prominent element in view at the time, but we must also give our earnest attention to the underlying neurotic condition, which is really responsible for the bleeding.

The hemoptysis must be treated by constant and systematic rest in bed, in a room kept dark and as near absolutely quiet as possible. Everything offensive to the patient's senses must be removed. Cold, in the form of ice-bags, or

compresses, the former preferable, must be applied to the chest if cold is at all permissible. To secure full repose the patient should not receive less than a quarter of a grain of morphine sulphate, twice a day, and a suppository of ten grains of asafœtida at night. Nourishing liquid food, in the form of freshly-expressed beef juice, two ounces three or four times a day, and milk, a glassful every three hours, must be given, cold, to the patient. After the worst of the bleeding is over a course of treatment is begun, which has in view the special improvement of the nutrition of the body. To achieve this best it is of the utmost importance that rest should be continued. The patient's energy must be conserved. The waste of strength must be curbed by reducing muscular activity to a minimum. This can only be accomplished by confining the patient to bed. Next in importance comes nutritious food, and the first of this is cold, freshly-expressed beef juice, two ounces four times a day. Good fresh milk, not less than five glasses daily, besides beef, mutton, lamb, eggs, oysters, soups of all kinds, and vegetables. Ergot, lead acetate, gallic acid or turpentine may be given. Strychnine should be begun in a not smaller dose than $^1/_{32}$ of a grain, four times a day, and gradually increased until the point of physiologic toleration of the drug is attained. This is not reached in most cases until it is pushed to $^1/_{12}$ or $^1/_{10}$ of a grain, four times a day, and perhaps not then. After this, the largest dose is continued. The suppositories of asafœtida will be continued every evening if the patient has a tendency to wakefulness at night. Quinine, acetanilid and phenacetin, in one, or two, or three grain doses, four times a day, are useful tonics, quinine being especially indicated if malaria is present. So is cod-liver oil, half an hour after each meal, if it is tolerated by the stomach. The hypophosphites, phosphoric acid and iron are all very highly indicated in the treatment. The patient must not be allowed

to practice forced breathing, or throw any strain on the respiratory organs. If the cough is troublesome, morphine or codeine will have to be given for the time being to relieve it. After the patient is well enough to be up, the getting out of bed must be undertaken gradually. At first he must not sit up longer than twenty minutes or half an hour each day, but in the course of a day or two this time may be gradually extended. The best index of the patient's inability to sit up is the feeling of tiredness, or exhaustion. As soon as this begins to manifest itself the bed must be sought at once. If this fails to show itself any longer, he can sit up and walk about during the whole day.

In addition to the general treatment of hemoptysis just outlined, these forms of the affection which depend on specific causes require special medication. Thus patients suffering from alcoholic hemoptysis are peculiar in several respects. They bear larger doses of strychnine than other patients. As a rule it is good practice to begin with $^1/_{20}$ of a grain, four times a day, by the mouth, and a similar amount, twice a day, hypodermically, and if this is borne well it should be gradually increased until it shows some evidence of its physiologic action. They also tolerate larger doses of capsicum. Red pepper is one of our most diffusible stimulants, and when its tincture is given in from half a drachm to drachm doses, every hour or two in water, a very beneficial impression is made, not only on the general well-being of the patient but also on the pulmonary bleeding.

Syphilitic hemoptysis, being in all probability produced by the action of a specific poison on the nervous system, it does not require much evidence to show that the remedy which is indicated in the treatment of other syphilitic affections is also applicable here. Hence, in connection with what has already been presented, good results are obtained from $^1/_{32}$ or $^1/_{20}$ of a grain of the corrosive chloride of mer-

cury, given four times a day, in half a drachm of syrup of sarsaparilla, or from the following combination:

℞ Strychninæ Sulphatis } aa..... gr. i
Hydrarg. iodidi vir }
Pepsini..... gr. xxx.
Pulv. glycyrrhiz, q.s.....
Ft. Capsulas No. xxxii.
Sig. One capsule four times a day.

The strychnine must, of course, be gradually increased and after the mercury, or the iodide of mercury, has been used for three or four weeks the syrup of the hydriodic acid may be substituted for a short time. When, as in the case of very severe and copious hemoptysis, it is desired to make a speedy impression the ointment of the nitrate of mercury must be rubbed into the axillary fossæ, groins and inside of the thighs.

The special therapeutic indications in the rheumatic form of hemoptysis are fulfilled by giving sodium salicylate, cinchonidia salicylate, potassium acetate, ammonium carbonate, lithium citrate, potassium iodide, calomel, colchicum, guaiacum and alkaline, bitter waters, like Carlsbad, Marienbad, etc. The following is a convenient and useful formula: ℞. Sodæ salicylat, ʒiiss; Cinchonidiæ salicylat, ʒii; Liq. ammoniæ acetati, ℥i; Vini colchici fl ʒi; Aquæ gaultheriæ, q. s., fl ℥iv. M. Sig. One teaspoonful every four hours. Lithium, in the form of effervescent tablets, dissolved in a glassful of water, is to be given with each meal.

In *cardiac hemoptysis* the disturbing cause must be sought and, if possible, removed. If this is rheumatic or gouty the salicylates and other anti-arthritic remedies are indicated. As in the treatment of other forms of hemoptysis, rest must be strictly enforced, and it is perhaps always in place to administer a heart sedative like aconite. In the *hemoptysis* which *accompanies pregnancy* or *follows parturition* the author has seen good effects from quinine, in four-grain doses, every four hours, when all other ordinary hemostatics had signally failed.

CHAPTER XX.

ACUTE PNEUMONIA.

DEFINITION. An acute inflammatory disease of the air-cells of the lungs, which is characterized by turgescence of the pulmonary capillaries, transudation of serum and of white and red blood-corpuscles, proliferation of the epithelial elements, and an accumulation of these products in the alveolar cavities. It is accompanied by fever, dyspnœa, cough, expectoration and by the physical signs of consolidation.

SYNONYMS. Croupous pneumonia, Lobar pneumonia, Catarrhal pneumonia, Lobular pneumonia, Bronchopneumonia, Secondary pneumonia, Pneumonie (German), Fièvre pneumonique (French).

PREDISPOSING CAUSES.

Age. Although acute pneumonia has no special affinity for any age-period, and may hence develop at any time in life, it must be admitted that in its catarrhal form it is very liable to appear as a sequel to the exanthematous and contagious diseases of childhood, and in this respect may be said to prevail most frequently during infancy. It must be borne in mind, too, that acute pneumonia is prone to follow the contagious diseases of adult life.

Race. Unacclimated races, or people of a lower civil status coming in contact with a highly civilized race, are strongly predisposed to pneumonia.

Climate. It is most prevalent in moist and raw climates,

or in seasons when there are sudden and extreme variations in the atmospheric temperature.

Occupation. Employments which are subject to sudden changes of temperature, as from hot to cold and wet to dry, like brewing, dyeing, baking, glass-blowing, etc., or occupations like stone-cutting, milling, working in iron dust, in which air laden with irritating particles is constantly breathed, or the inhalation of poisonous vapors, as is the case with the manufacture of mercury, phosphorus, etc., are excessively liable to bring on catarrhal pneumonia.

Constitutional Vigor. It is certain that alcoholics, or diabetics, or persons who follow certain injurious occupations, or those who are constitutionally weak, are specially predisposed to acute pneumonia ; yet, on the other hand, it seems as if this disease attacked those who are apparently in the best of health. Medical opinion is, therefore, somewhat divided on the question of liability in this respect. Hippocrates taught that pneumonia was specially prevalent among the robust, a view which was strongly shared by Grisolle, but which has been disputed by Bennett, Juergensen, von Ziemssen and others. With a view of throwing some light on this problem I was given access to the experience* of a large life-insurance company in regard to the fatality of acute pneumonia among its policy-holders. Such an organization contains a class of selected lives who were in good health when insurance was effected, and if the normal, healthy state protects against pneumonia then this disease should not occur as frequently among such lives during the first few years of insurance as some time later when the state of health may have become impaired.

Now the experience of this company on which these observations are based, extended from 1850 to 1897—a period of forty-seven years—and during this time

* Furnished through the kindness of Dr. A. B. Bisbee, Medical Director of the National Life Insurance Company, of Montpelier, Vt.

acute pneumonia caused 206 deaths; 15.04 per cent. of these deaths occurred before the expiration of the first year of insurance; 6.79 per cent. during the second year; 11.65 per cent. during the third; 2.91 per cent. in the fourth; 5.34 in the fifth, and so on, the last figure named being not exceeded in a single year afterwards. From these statistics it must be inferred, therefore, that a superior degree of personal health does not afford any immunity from an attack of acute pneumonia.

These statistics also show that a family history of pulmonary disease does not exert any special influence in the production of acute pneumonia. For, while the largest number of deaths occurred during the first three years of life-insurance, a careful examination of the records develops the fact that a family history of pulmonary disease was less marked during this than during subsequent periods.

From these data it appears, therefore, that health and vigor do not only not give absolute security against acute pneumonia, but, in accordance with the teaching of Hippocrates and Grisolle, entail a liability to this disease.

Pathology and Exciting Causes.

Pathology. In all that will be said on acute pneumonia the author fully recognizes the ordinary difference between the croupous and catarrhal forms of this disease, that one is lobar and the other lobular, that the deposit in one is chiefly fibrinous and in the other principally catarrhal, that in one the expectoration is rusty and in the other it is generally not, that in one pleurisy is nearly always present and in the other it is nearly always absent, and that they often exist independent of each other; but it will be maintained that this difference is neither vital nor essential fundamentally, that both are, in a great measure, the products of the operation of the same pathologic principle, that clinically

one cannot always be separated from the other, and that their likenesses and differences can be better studied and discussed in a single article than if they would be considered apart.

Among the earliest manifestations of acute pneumonia are distention and engorgement of the pulmonary capillaries, and a consequent sluggishness and stasis of the circulation in the affected parts. In the croupous form this seems to occur suddenly while in the catarrhal it is more gradual. Let us briefly glance at the effects of this change in the pulmonary capillaries in relation to the pneumonic process. This is, perhaps, nowhere better illustrated than it is in embolic pneumonia, in which the pulmonary capillaries are blocked with fine particles of foreign material. On this subject Dr. Hamilton* states: "It frequently happens that otherwise healthy subjects, who receive a simple fracture of a medullated bone, die from the effects of fat embolism. The oil from the medullary cavity is absorbed and carried to the right side of the heart, and thence into the pulmonary artery; its globules cannot pass the terminal branches of the artery, and become impacted in them. The emboli are minute, and are present in great numbers, while they are also bland and irritating. The state of occlusion caused by them will, therefore, be exactly that which, *a priori,* I would say should give rise to a croupous pneumonia, on the principles just discussed, namely, that certain of the blood-channels are obliterated, and that the blood forced into those which are still open is consequently at a higher pressure than under natural circumstances. . . As an actual fact, croupous pneumonia, either of a general or of a localized character, is frequently associated with this embolic condition. I have in my possession several preparations of this kind taken from persons who, previous to the time of injury, were in perfect health, but who died in from forty-

* *Op. cit.*, p. 118.

eight to sixty hours, with intense pneumonic effusion into one or both lungs, accompanied by widespread fat embolism. In all respects these lungs, with the exception of the emboli in the vessels, presented the appearances of a red hepatization, and I think the conclusion inevitable that the obliteration of the vessels was primary, the croupous exudation secondary."

Such, then, are the effects of sudden dilatation of the pulmonary capillaries. When this is brought about more gradually, as is the case in disease of the mitral valve, the results are quite different. According to Hamilton (p. 131) a lung of this kind is œdematous and the pleural vessels are deeply congested, and pneumonic patches of a brownish-red color are scattered throughout the substance of the organ. He says, when these brown patches are examined microscopically, they are seen to have the appearance shown in Fig. 39, which represents the whole of an alveolus.

In this figure the capillaries (*a*) around the alveolus are markedly distended and engorged with blood. The alveolus, besides dark-brown blood pigment, contains the epithelial cells (*b*) of its surface, which have desquamated. At the lower part of the figure an epithelial cell is partially cast off. Sometimes fibrin is deposited in the alveolar cavity.

The difference in the morbid products between these two cases depends largely on the varying degrees of pressure applied in each. In the first the pressure comes quickly and the serum of the blood is suddenly forced through the walls of the blood-vessel into the alveoli, together with some white and red blood-corpuscles, while in the latter the pressure is probably less intense and certainly more gradual in most instances, and is diffused over a longer period of time, and the blood-vessels have a better opportunity to adjust and to accommodate themselves to the new order of things and in this way prevent a copious outgush of blood-serum. In other words, here the intensity of the morbid

process is directed more towards undermining the vitality of the alveolar epithelium than towards the forcing of serum into the alveoli. That these two morbid manifestations are but different steps in the same pathologic process is, according to Hamilton, shown by the fact that when a lesion of the mitral valve is suddenly developed, as is the case in

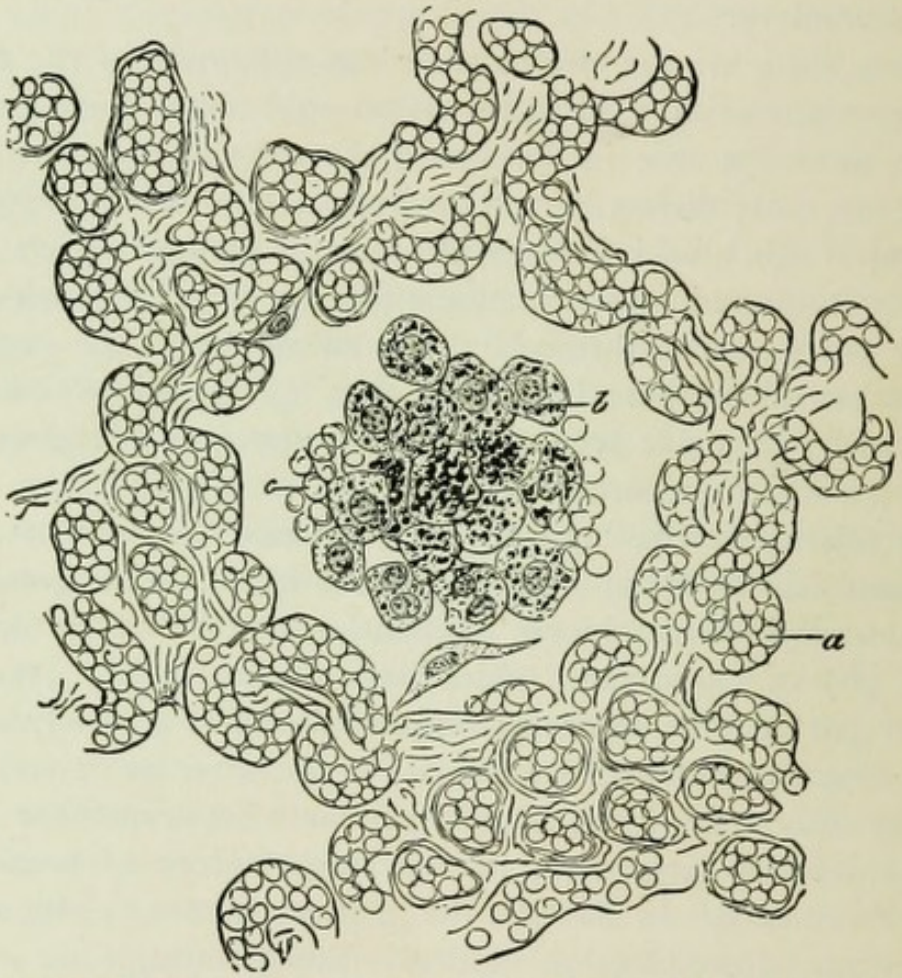

Fig. 39.—Section of an alveolus from lung of a person who died from mitral disease. *a*, distended and projecting alveolar capillaries; *b*, desquamated epithelium; *c*, blood-corpuscles extravasated into alveolar cavity.—*Hamilton.*

rheumatism sometimes, croupous pneumonia and pleurisy, instead of pulmonary œdema, are engendered.

Now there is good reason for believing that catarrhal pneumonia is dependent on the operation of the same morbid principle, although in a milder form, than that which ob-

tains in the case of croupous pneumonia and pulmonary œdema. With the exception of the whole condition being less active than it is in the two latter affections, the distension of the pulmonary capillaries and the sluggish circulation within them are the same as is seen in Fig. 40, and it is

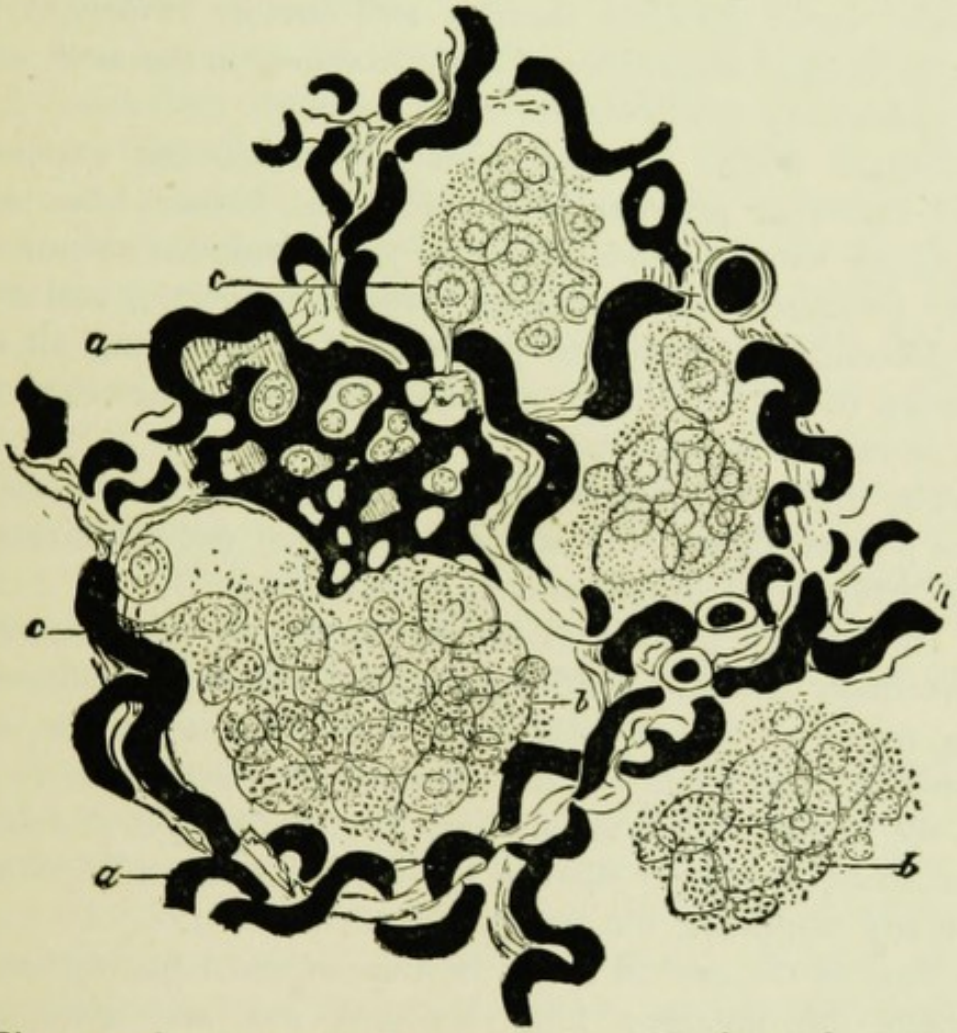

Fig. 40.—Acute catarrhal pneumonia. Blood-vessels injected. x 450 diams. *a*, injected capillaries of alveolar wall; *b*, catarrhal cells lying in the alveolar cavities; *c*, the same, sprouting from the wall.—*Hamilton.*

probable that on account of this difference there is less or no fibrinous exudation and œdema and a greater predominance of the catarrhal element. In other words, it appears as if the morbid process is not powerful enough to overflow and overwhelm the alveolar cavities with serum, but

just sufficiently strong to promote an exuberant epithelial overgrowth on their surfaces and to fill them with the accumulated products of the same. The relationship between croupous and catarrhal pneumonia is, therefore, shown to be a very intimate one; and that no sharp and impenetrable barrier exists between them is still further evidenced by the occasional occurrence of both processes in the same area of pulmonary consolidation.

Neither is the common division-line between croupous and catarrhal pneumonia—making the former lobar and the latter lobular—an absolute one; for croupous pneumonia may be disseminated in spots throughout a lung and thus be lobular; and catarrhal pneumonia, by involving all the lobules of a lobe or two, assumes a lobar character, so far as extension is concerned.

The different changes which the lungs undergo in croupous pneumonia are (1) engorgement, (2) red hepatization, (3) gray hepatization, and (4) resolution.

The morbid process in the first stage of this disease is principally confined to the pulmonary capillaries, although the nuclei of the alveolar epithelium begin to divide, and some red and white blood-corpuscles may already be found in the alveoli. The affected portion of the lung is of a deep-red color, is heavier than in the normal state, still contains air and crepitates, but does not sink in water.

Next is the second stage, or that of red hepatization, in which the inflamed lung becomes red and solid and heavy, and sinks in water. The alveoli are now filled with coagulated serum, which exudes from the blood-vessels, and with red and white blood-corpuscles and epithelial cells. An incision will draw very little serum or blood, but the cut surface looks rough and granular—each granule representing a group of alveoli filled chiefly with exuded material.

Then comes the third and really the critical stage of acute

pneumonia. It marks the transition period between solidification and softening; for now the inflammatory products are beginning to undergo chemical metamorphosis, which terminates either in fatty degeneration, or, rarely, in caseation. The lung is changed from its former red to a yellow-grayish color on account of the discoloration of the exudate and also by reason of the anæmia, which is now present in the pulmonary tissues. A cloudy, milky fluid may be pressed from its cut surface and small plugs of exudate may be extricated from the alveoli and small bronchi.

In the last stage, which is the fourth, the change of the alveolar contents into a soft and fatty detritus, which began in the preceding one, is consummated. It appears that the amount of fat which is produced in a pneumonic lung at this period of the disease, and which is largely governed by the degree of infiltration and by the consequent defect in the supply of oxygen and of nutritive material, rises to fifty per cent., according to Guillot, while the normal proportion of fat to tissue is only ten per cent. The products of this metamorphosis are finally removed by absorption and expectoration, chiefly by the former.

Catarrhal pneumonia does not divide itself into well-defined stages, like the croupous form, but it also has a well outlined and characteristic pathology. It is essentially an inflammation of the air cells and of the bronchial tubes, the latter being generally primarily affected. A lung in this conditions feels vesicular throughout, and is capable of being considerably inflated, but on its surface are small areas of a leaden or bluish color, which are almost totally non-vesicular. These areas mark lobules, or groups of lobules, which are involved in the inflammatory process and which cause the lung to feel nodular or like a mass of frogs' spawn. Bordering on these areas the lung tissue is more vesicular and may be emphysematous. Acute pleurisy and pleural adhesions are rarely seen. An incision into the lung

reveals irregularly shaped, inflammatory patches of a greenish-yellow color, out of which may be squeezed a thick, yellowish fluid. The mucous surface of the bronchial tubes is always in a state of deep congestion and out of their cut ends may be pressed a yellow, catarrhal secretion.

A microscopic section of such an inflammatory patch is well represented in Fig. 17, page 262.

In the center of this figure, as is usually the case, there is a bronchiole (*b*), distended with catarrhal products, and surrounding these are the air cells (*a*), also filled with catarrhal material, while in the periphery of the figure are seen the distended and emphysematous air vesicles (*c*). The catarrhal contents of the air cells and tubes furnish the yellow fluid, which, on pressure, exudes from the cut surface.

What then, is the nature, source and destiny of the alveolar contents? In answer it may be stated that besides leucocytes, and sometimes fibrin, they chiefly consist of large and small epithelial cells, some with more than one nucleus, and others without any, which are cast off from the epithelial cells of the alveolar lining. These cells proliferate and multiply rapidly under the influence of some irritation and accumulate in the air cells and tend to undergo the liquefaction of fatty degeneration. This degeneration gives rise to the yellow fluid-material which is found in the bronchioles, and which pressure can force out of the pneumonic area.

The capillaries surrounding the alveoli are, like in croupous pneumonia, largely involved. They are always in a state of dilatation and congestion, and the small arteries and veins contain a superabundance of blood, which is in partial or total stasis.

Exciting Causes. While it has been shown so far that in the main both croupous and catarrhal pneumonia are the result of a variation in the same pathologic mechanism, *viz.*, engorgement of the pulmonary capillaries, and that genet-

ically they are, therefore, closely related, it is quite obvious that the source of the inflammatory process is not accounted for and that the latter must be the outward manifestation of some deeper-lying cause. Now it is of the highest scientific interest that an analogous, if not an identical, condition may be produced in the lungs of animals by dividing the vagi, and it will be seen from what follows that perverted pulmonary innervation is frequently though not always the cause of acute pneumonia.

In a series of experiments performed by Dreschfeld* it was found that from four to ten hours after division of the vagi the pulmonary capillaries around the alveoli are distended and tortuous and that besides a few red-blood corpuscles, lymph cells and some pigment, the alveoli were completely filled with large, granular, nucleated cells. The alveolar surfaces were denuded of their epithelium, and this experimenter believes that the large cells found in the alveoli are metamorphosed epithelial cells. Gärtner, in a similar investigation† on the influence of vagus-section on the lungs, found the following microscopic changes: The alveoli were filled with reddish-gray masses, which gave the section-surfaces a kernel-like appearance, these masses consisting of young cells and blood-corpuscles. The interalveolar septa were infiltrated. The mucous surface of the bronchi was intensely reddened and covered with mucus, and its epithelium was frequently wanting. The infiltrated spots mark the areas in which the bronchi are most seriously involved and this leads Gärtner to believe that the inflammation produced by vagotomy extended from the bronchi into the alveoli.

* "Experimental Researches on the Pathology of Pneumonia." *Virchow's Jahrbericht.* 1876. Bd. II., 2.

† "Ueber die sogenannte Vaguspneumonie," *Allgemeine Wiener med. Zeitung*, 30, 1885, 5, 38 u. 50.

According to Friedländer,* six hours after vagotomy a series of dark-brown infiltrated spots appear in the lungs, which on microscopic section show that they consist of stringy or fibrinous, coagulated masses of large, granular cells and red-blood corpuscles, which are packed into the alveoli. These large cells cling closely to the alveolar walls, but also protrude into the lumen of the alveoli. Friedländer says they arise directly out of the alveolar epithelium; that they are like the cells which have been described and characterized by Colberg in the catarrhal pneumonia of man as "swollen epithelium," and that they finally undego fatty degeneration.

From these investigations it appears that the microscopic anatomy of vagus pneumonia agrees with that which is found in acute pneumonia of man, in the following particulars:

1. The pulmonary capillaries are engorged and tortuous.
2. An inflamed bronchiole frequently occupies a central relation to the infiltrated area.
3. The infiltrated areas consist of large and small nucleated and non-nucleated cells, of a stringy, fibrinous exudation and of white and red-blood corpuscles, which pack and distend the alveoli.
4. The large and small cells are derived from the epithelial cells of the alveoli; and,
5. The alveolar contents undergo fatty degeneration.

These experiments demonstrate, therefore, that there is a certain definable relationship between lesions of the pneumogastric nerves and acute pneumonia, and it will now be shown how amply this demonstration is confirmed and strengthened by evidence of a clinical character, which has been collected from the literature of the subject. Thus:

* "Untersuchungen ueber Lungenentzündung nebst Bemerkungen ueber das normales Lungenepithelium," *Centralblatt f. d. med Wis.*, 1873, S. 536.

Case 220. Sommer contributes (*Charité Annalen.*, 1888, p. 647) the case of a boy, aged 10, who suffered from nephritis and cardiac hypertrophy. Death in twelve days. On section there were found pulmonary infiltration and dilatation of the heart. The left vagus was imbedded and compressed by a fibrous growth, and had undergone degeneration.

Case *221.* Hewson* reports the case of a male, aged 55, who had an aneurism involving the innominate and the arch of the aorta. Ten days after ligation death resulted from asphyxia. The aneurism had eroded the sternum and clavicle, and compressed the right vagus and recurrent laryngeal. The lungs were solidified in spots and filled with frothy serum.

Case 222. Johnson† cites the history of a male, aged 45, whose vocal cords were almost motionless during phonation and who had a feeble voice, with stridulous breathing. There were dulness and a distinct systolic bruit over the manubrium of the sternum. Tracheotomy afforded temporary relief. He died from apnœa. Gray hepatization existed in the base of each lung, and there was found an aneurism of the aortic arch, in which the left vagus was imbedded and which caused atrophy and compression of the left recurrent.

Case 223. Wallis reports the history of a male (*Hygiea*, xlvi, 9, p. 545, 1884), aged 23, who had for a number of years been suffering from cough, hoarseness, and dyspnœa. Death caused by pulmonary embarrassment. There was consolidation of right lung, and fibroid degeneration of left. Both vagi were hypertrophied and neuromatous.

Case 224. Schmidt‖ reports the history of a man, 30 years old, and colored, who was admitted with right-sided

* *Amer. Jour. of Med. Sciences*, vol. 3, 1842, p. 221.
† *Trans. London Path. Soc.*, vol. XXIV., 1873, p. 42.
‖ *Centralblatt f. Nervenheilkunde*, vol. vi, p. 39.

pneumonia and died in two weeks. Autopsy showed hepatization of right and œdema of left lung, and purulent pericarditis. The largest of two gliomatous tumors, in the fourth ventricle, pressed on the vagus center.

In a very interesting article* on the subject of vagus pneumonia, Bianchi discusses a pneumonia which frequently occurs in paralytics,and which differs clinically and anatomically from croupous pneumonia. The temperature is usually low, while the pulse-frequency varies from 50 to 140, cough and expectoration are absent sometimes, and the respiratory movements are slow and superficial. Neither percussion nor auscultation always gives positive results. Anatomically there is seldom any fibrinous exudation. The affected lung usually remains in a hepatized condition. Frequently there exist larger or smaller gangrenous foci, and nearly always, if the case is of long standing, there is puriform infiltration of the bronchi and alveoli. All these manifestations simulate those of the pneumonia which is produced by section or compression of the vagi, and which Bianchi produced artificially in rabbits and dogs. In a number of paralytics who died of such a pneumonia he was able to trace a primary degenerative atrophy of the vagi, and hence he believes that this affection is dependent on vagus degeneration, and differs from Traube and Frey, who hold that it is engendered by the aspiration of particles of food.

In a very interesting research by Dr. J. Chrichton-Brown† the post-mortem examination of the bodies of one hundred persons who died of general paralysis showed that pneumonia and pulmonary congestion were present in sixty-two instances.

The series of cases and observations thus far presented il-

* "Pneumonia in Paralytics with Degeneration of the Vagi." *Neurologisches Centralblatt*, 1890, p. 249.

† "The Pulmonary Pathology of General Paralysis," *Brain*, vol. vi, 1883, p. 317.

lustrates very clearly that pneumonia in man may be caused by injury to or by disease of the pneumogastric nerves, and in this respect fully confirms the results which have been obtained in the lungs of animals whose vagi were divided. Now, if disease of the vagi from local causes leads to pneumonia in man, it seems altogether reasonable to assume that disease or injury of the brain, or of the important nerve centers, especially of the base of the brain—the seat of the origin of the respiratory nerves—should, if other things are equal, also be followed by pneumonia, or at least by some pulmonary disorder, in man. This assumption is not unsupported by facts and the proof which is here submitted to substantiate it consists of clinical observations, abstracted from the records of medicine, in which the higher nerve centers had either sustained injury or were involved in disease. Falk* cites this interesting case:

Case 225. Female, aged 27, in good health, was shot in the left temple. Convulsions followed in eighteen and death in twenty-four hours. Section: Right lung dark-red, sunk in water, and from its cut surfaces oozed a brown-red viscid fluid. The branches of the pulmonary artery were empty and the larger bronchi contained froth. The left lung was œdematous throughout. Dr. Falk says that the lung-condition here is similar to that which is described by Rokitansky as inflammatory engorgement, or by Laennec as engorgement, precisely the early stage of croupous pneumonia.

Case 226. Rocks† records the case of a male, aged 22, who fell from the third story of a house and produced a large scalp wound. He was unconscious, but in three days he became rational. In about a week after this he died of croupous pneumonia. *Post-mortem* examination showed an unfractured skull and an uninjured brain, except the base

* "Zur Frage der Pneumonien nach Kopfverletzungen." *Vierteljahrschrift f. gerichtliche Medicin*, N. F., 5, 47, 1887, S. 292.

† *Vierteljahrschrift f. gerichtliche Medicin*, N. F., 47, 1887, S. 2.

of which was infiltrated with blood from the pons to the commencement of the spinal cord. The same authority cites nine other cases of head injuries similar to the one just given, eight of which died of acute pneumonia and one of pulmonary œdema

Case 227. Goodhart* reports the case of a boy, 2 years old, who had a fall on the head. The third day he became drowsy, had a retracted neck, vomited, and had a fever of 101° F. He remained in this condition for a fortnight, when an apical pneumonia slowly developed, from which he ultimately recovered. In his comments on this case Goodhart says: "I really believe that injuries to the head are quite capable, by interfering with the control powers of the nervous centers, of leading to disordered function and to such pronounced organic departures even as acute pneumonia."

Case 228. Garber† relates the following interesting case: Male, aged 45, in good health, fell about eighteen feet, and landed on his feet, but losing his balance he fell on his right side, against a pile of lumber. He was unconscious for two hours. There were no marks of external injury, but he complained of pain along the spine from the cervical to the sacral region, with numbness and tingling in his feet and legs. Chest normal, but the left pupil was somewhat dilated, and he had pain in the right hypochondrium. The temperature was normal and in twenty-four hours he was comfortable. On the second day temperature 101° F., and left pupil still dilated; on the fourth day he felt quite well, and the limbs could be moved with comfort, but still felt numb. No cough nor expectoration and chest signs negative. On the seventh day there was dyspnœa and epigastric distress, and on the eighth day dulness, bronchial breathing and crepitant râles over left posterior base. No trouble on the right side. Left pupil dilated. On the tenth day left base cleared

* Harveian Lectures for 1891 on Common Neuroses, p. 41.

† *Jour. of the Amer. Med. Assoc.*, February 2, 1889, p. 176.

up and right posterior base became involved; no cough nor expectoration. Became progressively worse, and on the thirteenth day there was intense dyspnœa and cyanosis, and breathing was labored and shallow, and the right lung was completely consolidated and death occurred on the following night. No autopsy was allowed. Garber states that the nervous phenomena observed throughout this case indicate that the force of the injury was spent on the cerebro-spinal system and that the pulmonary consolidation was produced through a lesion of the pneumogastric nerves.

There is also a close affiliation between some idiopathic disorders of the brain, like hemiplegia, meningitis, etc., and pneumonia, as is shown by the following instances:

Case 229. Lepine* gives the case of a man who had sudden apoplexy after a few days of pneumonia. He was brought to the hospital unconscious the day after the apoplectic attack. There was complete hemiplegia of the right side, with flaccid muscles, the tongue pointing to the left. Death. Post-mortem examination showed complete hepatization of right lung and only congestion of the left. The left hemisphere of the brain was more watery and paler than that of the right. At the foot of the second left frontal convolution was a small spot of softening, extending through half the thickness of the gray matter.

Case 230. Dr. B. H. Stephens† mentions the case of a man who was suddenly attacked with apoplexy and in two days the whole of the right lung became involved in pneumonia. The pneumonia ran its regular course and the lung cleared up, but the paralysis of the arm, leg and facial muscles did not disappear for three months.

Case 231. In the case of a child, 2 years of age, which he also cites, the pneumonia was preceded a few hours by strong convulsions, after which symptoms of meningitis be-

* *Practitioner*, June, 1886, p. 446.

† *British Med. Journal*, Dec. 15, 1888, p. 1351.

came well marked. Death. Engel* states most apoplectics die of pneumonia, and that among 31 cases of brain disease only in 4 was there no affection of the lungs or intestines. In 14 there was pneumonia or pleurisy, and in 9 there was pulmonary œdema. Pneumonia occurred three times on the side opposite the apopleptic area.

E. Grawitz† found that in 30 cases of genuine cerebral hemiplegia respiratory disturbances were absent only in 7, and that in the 23 remaining cases—77 per cent.—the respiratory function was involved, in two of which Cheyne-Stokes breathing was present. In most of these cases there was a retardation of movement on the paralyzed side.

Pneumonia of Epilepsy: Furthermore, pneumonia is closely related to if not actually dependent on various disorders of the oblongata. One of these is epilepsy, which, from whatever source it may arise, essentially involves the integrity of the convulsive center or the center of muscular co-ordination. Anatomically and physiologically this center is intimately allied to the respiratory center, as has been said already, and hence it is reasonable to believe that disease of the one tends to lead to disorder of the other—a relationship which is substantiated by the well known fact that epilepsy is frequently complicated by pulmonary embarrassment, as is attested by the following quotations from the classical work of Echeverria‡:

Case 232. Boy, aged 3, died from exhaustion due to epilepsy. Autopsy showed red and gray hepatization in the posterior surfaces of the lungs, with extensive morbid changes in the oblongata and vagi (p. 101).

Case 233. Female, aged 40, epilepsy from childhood.

* *Prager Vierteljahrschrift*, vol. vii, Jahrgang Bd. 3, S. 19.

† *Zeitschrift f. klin. med.*, 1894, S. 26.

‡ "Epilepsy: Anatomic, Pathologic and Clinical Notes," M. Gonzalez Echeverria, M.D., New York, 1870.

Death in a fit. Post-mortem investigation showed hepatization of right lower lobe, and yellow softening of the cerebellum, oblongata and nuclei of both vagi (p. 138).

Case 234. Female, aged 31, epileptic and intemperate, died from pleuropneumonia. After death it was found that the vagi were very much altered and that the oblongata had undergone degeneration (p. 142).

Case 235. Male, aged 9, epileptic and idiotic. After death the lungs were found engorged and the oblongata and sympathetic degenerated (p. 142).

Case 236. Male, aged 43, epileptic and hemiplegic. Death. Lungs hepatized and oblongata degenerated (p. 144).

Case 237. Lemaine* reports the case of a male, addicted to alcoholic excess from youth, who was attacked with pneumonia at the age of 32 and again at 40, both attacks being ushered in with an epileptic convulsion. Neither before nor since had he had epilepsy.

Pneumonia in Cerebrospinal Meningitis: Another disease which attacks the brain, and especially its base, and is closely allied to pneumonia, is cerebrospinal meningitis. Indeed these diseases simulate each other so closely, particularly in epidemics of one or the other, that it becomes extremely difficult and sometimes impossible to differentiate between them. On this subject Surgeon J. J. Woodward† has already been quoted. On page 758 of same volume he furthermore states that exposure of the troops to sudden changes of the weather, as in the case of a snowstorm following a warm day, occasioned numerous cases of severe catarrh which rapidly passed into obstinate pneumonia. During one month (November) under such a condition the regiment had two hundred cases of pneumonia, of which fifty per cent. were catarrhal, forty per cent. croupous, and the remaining ten per cent. cerebral and

* *Centralblatt f. Nervenheilkunde*, Bd. II, 1880, p. 680.

† See pp. 103-104.

erysipelatous types, which were deadly. The cerebral type, which was at first mistaken for meningitis, as it was characterized by rigors, headache and but little pulmonary disturbance, proved fatal with convulsions and delirium in from twelve to twenty-four hours. In some of the erysipelatous cases the inflammation seized upon the pharynx, but in others the erysipelas was developed subsequent to the pulmonary symptoms and appeared more as an accidental complication. The post-mortem appearances in the lungs of these cases of cerebral pneumonia were comparatively slight, while the cortex of the brain was inflamed, the meninges congested, and the ventricles contained a serous exudation. Weber* and Kuhn,† in two epidemics of pneumonia, found cerebrospinal meningitis present eight times in fifteen post-mortem examinations. Dr. J. Netten Radcliffe‡ states that all the fatal cases of cerebrospinal meningitis which came under his notice the most prominent symptoms which preceded death were those which indicated impairment and perversion of the respiratory function; and that among its principal complications were pleurisy, pneumonia, bronchitis, or pericarditis.§

In an epidemic‖ of cerebrospinal meningitis which occurred in the city of Boston, in 1897, Councilman, Mallory and Wright made autopsies in 35 cases of deaths from this disease, and found pulmonary congestion and œdema in 13, bronchopneumonia in 7, croupous pneumonia in 2 and bronchopneumonic foci in 8 cases; or, in other words, the lungs were involved in 30 cases.

Pneumonia of Influenza: Whatever the precise etiology

* "Path. Anatom: Neugeborenen und Säuglinge," ii, S. 61.

† *Deutsches archiv f. klin. med.*, Bd. 21, xxi, S. 364.

‡ "Reynolds' System of Medicine," vol. i, p. 507.

§ For further reference, see pp. 103-104.

‖ *Amer. Jour. of Med. Science*, March, 1898.

of influenza may be, whether it is caused by a specific germ or some other toxic substance, it must be recognized that all the varied exterior phases of this disease are but expressions of a disturbance that exists in the nervous system beneath. The organs which are perhaps most frequently involved in this affection are the lungs, and the synonyms *peripneumonia notha, peripneumonia catarrhalis, etc.,* used by the older writers, designated the pulmonary manifestation of this disease more accurately than our modern terms of influenza and *la grippe.*

There is much reason for believing that the pulmonary troubles of influenza depend primarily on disorder of the central nervous system, and, secondarily, on perverted innervation of the vagi. This was the ground strongly taken by Graves* of Dublin; and Walshe† states that this poison (influenza) seems specially to exercise its influence on parts supplied by the pneumogastric nerve. Sansom‡ is of the opinion that all the consequences of influenza may be accounted for on the score of a "peripheral neuritis affecting the sympathetic ganglia and nerves, the vagus and the sensory motor nerve trunks."

M. Vovart§, of Bordeaux, looks upon influenza as a neurosis of the pneumogastric nerve. M. Huchard‖ states that the action of the influenza poison falls upon the peripheral nerves as well as on the central nervous system, attacking the several branches of the pneumogastric most violently, producing, through its cardiac branches, syncope, arrhythmia, intermittence, bradycardia, tachycardia, aginiform difficulty and sudden death; through its pulmonary branch, pneu-

* "On Influenza," *London Med. Gazette*, vol. xx, p. 10.

† "Diseases of the Lungs," p. 563.

‡ "On Some Painful Affections following Influenza," *Lancet*, Jan. 2, 1892.

§ "La grippe et sa pathogenie," Paris, 1881.

‖ *Revue générale de clinique et de therapeutique*, Jan. 16, 1890.

monia, hemoptoic pulmonary congestion, pulmonary œdema, bronchial paralysis and a pertussis-like cough.

In an epidemic of influenza in the Danish Asylum, of Aarhus, Helweg* made 11 post-mortem examinations and found a most intense hyperæmia of the pia mater and brain. This was especially marked in the arteries at the base of the brain, which were filled with blood to bursting and stood out as cylindrical cords, as if they had been injected with wax.

Cerebrospinal meningitis and influenza have, therefore, such a close clinical and pathological relationship that the former seems to be merely an intensified form of the latter. They are both the result of some toxicity of the nervous system, the evidence of which is largely confined to the brain, and especially to its base, and they both have a strong tendency to engender pulmonary complications. The existence of this alliance is still further confirmed by the simultaneous prevalence of the two diseases, a fact which has frequently been observed and one which is specially dwelt on by the committee† "on spotted fever, so-called," in the following report to the American Medical Association: "When the attention of the profession in Philadelphia was called to the existence of spotted fever there was prevailing in that city a severe and widespread epidemic of influenza, or epidemic catarrhal fever. . . . Several men in active practice were at once struck with the resemblance of many of the symptoms of the two diseases, and were led to inquire if influenza might not be but a mild manifestation of that epidemic influence which, in its intensity, produced spotted-fever. Nor, it will be seen, were their symptoms very dissimilar save in degree."

Pneumonia of Whooping-cough. Whooping-cough is

* "Influenzaens Virkninger," etc., *Hospitals Tidende*, Copenhagen, July, 1890.

† *Trans. American Medical Association*, 1866, p. 337.

essentially a nervous affection due to special intoxication of the respiratory nerves. Hufeland, Hoffman, Wendt, Walshe and Puldame ascribe it to irritation of the pneumogastric nerves, and Guibert to that of the general nervous system. In all severe cases of this disease there is congestion of the bronchi and lungs, and bronchopneumonia and even phthisis are frequent complications.

Pneumonia of Typhoid Fever: Typhoid fever is the product of a specific poison which, besides its deteriorating influence on the lymphatic glands of the small intestines, intoxicates the nervous system. Thus insanity, aphasia, temporary hemiplegia, paralysis and degeneration of the tibial and peroneal nerves, paraplegia, acute ascending paralysis, progressive muscular atrophy and ataxia may be established during or follow an attack of typhoid fever. Moreover, the vagi are also implicated, for in the examination of 26 cases whose deaths were caused by typhoid fever Dr. Lewin* found that degeneration of the vagus existed in every instance.

Pulmonary disease is also a frequent concomitant or sequel of typhoid fever. Louis made the observation that inflammation of the lungs is more frequent in typhoid fever than in any other acute disease. Murchison asserts that an attack of this disease is often followed by destructive disease in the lungs. Dr. Harley states† that in upwards of 30 typhoid cases which he examined he found the lungs free from more or less extensive inflammation only twice.

Pneumonia of Diphtheria: Diphtheria is well known as a disease that disintegrates the nervous system and also one that is prone to be followed by acute pneumonia. The nervous symptoms show themselves in loss of motion and sensation in the pharynx, larynx, soft palate and lower extremities. The heart and lungs become seriously embar-

* *Beiträge zur Vagus Pathologie*, 1888.

† "Reynolds' System of Medicine," vol. i, p. 378.

rassed through the action of the diphtheritic poison on the circulatory and pulmonary nerve supply. Retardation of the pulse and paralysis of respiration seem to be the result, as Ross says,* of a primary irritation followed by paralysis of the vagus. Squires† states that loss of power and of sensibility in the parts supplied by the par vagum occur at a somewhat earlier period in this disease than the paralytic affections of other parts of the body.

Case 238. Heath‡ relates the case of an adult male with a morbid growth on left side of neck which was excised, and in doing this the left vagus was exposed for five minutes and was also stretched. Death. Both lungs were œdematous and infiltrated. Left vagus-sheath inflamed.

Pneumonia of Rheumatism: Rheumatism and gout are at least concomitants if not causes of both acute and chronic pneumonia, and of many other pulmonary disorders. According to the researches of Dr. A. Haig§ there is much reason for believing that uric acid—the active toxin of these diseases—exerts an injurious influence on the nervous system and thus cause a number of nervous disorders like migraine, epilepsy, hysteria, convulsions, etc., and in view of such an action it is not very difficult to conceive that this agent may play an important rôle in the development of acute pneumonia.

Pneumonia of Malaria: Malaria is frequently associated with pneumonia in localities where intermittent fever prevails. This was especially noted among the soldiers of our late civil war. Dr. Woodward‖ gives

* "Diseases of the Nervous System," vol. ii, p. 977.

† "Reynolds' System of Medicine," vol. i, p. 125.

‡ *Trans. Lond. Path. Society*, vol. 35, p. 342, 1884.

§ 'Uric Acid in the Causation of Disease," third edition. London: J. and A. Churchill.

‖ "Medical and Surgical History of the War of the Rebellion," Part 3, Medical Volume, pp. 131–144.

the post-mortem results in 49 deaths which were caused by malarial fever. In 7 of these the lungs were not examined, in 13 they were found normal, but in 29 instances they were diseased. In a number of these cases, in which the brain was examined, there was found meningeal congestion and effusion of serum into the ventricles. Dr. Mason* describes a malarial form of pneumonia, which he considers the prevailing type of the disease in the South, as a severe remittent fever with pneumonic symptoms superadded. The lungs become permeated with a blood-tinged serum rather than consolidated by exuded plasma. Dr. Gaines† also writes on malarial pneumonia and says that cases ushered in with a severe chill are dangerous, as the lungs may be overwhelmed by the sudden congestion, but the fatality generally depends more upon the febrile disease than upon the pulmonary inflammation which accompanies it.

Contagious Pneumonia: Heretofore pneumonia has been considered as a disease of interior origin, *i.e.*, as being produced by causes which are directly generated within the body, but it is a question whether or not this disease may not emanate from external sources, *i.e.*, arise directly from other cases of pneumonia through infection or contagion? To show this, however, it must not only be demonstrated that a number of cases of pneumonia occur at the same time and in the same locality, for this may easily be explained on the basis that certain telluric, atmospheric or other epidemic conditions are influencing a given number of inhabitants in the same manner, but it must be made clear that the toxin of the disease has been

*"Malarial Pneumonia," O. F. Mason, New Orleans; *Medical News*, and *Hospital Gazette*, vol. iv, 1875–58, p. 400.

†"Malarial Pneumonia," E. P. Gaines, New Orleans; *Med. and Surg. Jour.*, vol. xx, p. 12.

Both citations taken from "Medical and Surgical History of the War of the Rebellion," Part 3, Medical Volume, p. 123.

transmitted to and produced the disease in persons living under different conditions and in different localities. While the author agrees with Dr. Wilson Fox* that "the evidence with respect to infection or contagion is much less strong in the large majority of these cases (pneumonia) than that of local epidemic influence," it cannot be gainsaid that occasionally and under certain exceptional circumstances cases of acute pneumonia have been observed which had an undoubted infectious or contagious origin. One of the few and one of the most marked instances of this kind that have come under the observation of the author is that contributed by Dr. D. J. Leech† of Manchester, England, and of which the following is an abstract:

(1) Mrs. S., aged 32, pregnant, living at 43 Acton street, Manchester, was attacked by pneumonia of left apex, January 9, 1890. On the 14th she appeared to be convalescent, and went out-of-doors on the 10th of February. On the evening of the same day she had a relapse of the disease, premature labor occurred on the 12th, and she died on the 13th of the same month.

(2) Mrs. B., aged 45, lived on the opposite side of the same street, and nursed Mrs. S. during her illness. On the 14th of February, after Mrs. S.'s death ,she cleaned the bedroom, removing two buckets of excreta covered with mould, which must have been there for a week or a fortnight. The rooms were extremely dirty, and the agent reported that when subsequently the place was cleansed, fæces were found all over the house. On the 14th she was seized with left-sided pneumonia, and died on the 22d.

(3) Mary F., aged 15, living 100 yards from Mrs. S.

* "Treatise on Diseases of the Lungs and Pleura," p. 262.

† "Clinical Lecture on a Case of Infectious Pneumonia followed by Peripheral Neuritis," *Manchester Medical Chronicle*, 1891, pp. 265, 337.

(43 Acton street), often visited the latter during her illness and went to see the corpse. She was attacked with basic pneumonia on the 16th of February, from which she recovered.

(4) Edward S., husband of Mrs. S., was suddenly seized with giddiness, nausea, vomiting and chilliness, but without distinct rigors, on the morning of the 20th of February, and on the 22d he had well-defined physical symptoms of pneumonia of the right base. He was admitted to the Infirmary and post-peripheral neuritis complicated the case, but he made a good recovery.

(5) Mrs. T., aged 65, grandmother of Mary F. (3), also lived 100 yards away from Mrs. S.'s house, but visited the latter, and took tea there on the day of Mrs. S.'s funeral (February 18th). On the 19th she was seized with pain in the chest and fever, and apparently passed through pneumonia, from which she recovered.

(6) James S., aged 65, father of Edward S., who lived half a mile from his son (43 Acton street), sat up with the latter one night and went with him into the Infirmary (22d). On the 23d he was attacked with pneumonia, from which he died on the 28th.

(7) Thomas F., aged 40, also lived 100 yards from Mrs. S.'s house (43 Acton street), visited Mrs. S. during her illness and saw her after she was dead; was taken with pneumonia, from which he died.

(8) William S., brother of Edward S., had not been at 43 Acton street for twelve months until the night of the 21st of February, when he sat up with his brother. He visited his father during his illness. On the 4th of March he was seized with pneumonia, which caused his death in three days.

Now, in view of the fact that no epidemic of pneumonia prevailed at that time in that district of Manchester in which Acton street is situated, and which comprises about 6,000

inhabitants of that city (only two cases of the disease occurring in the practice of the physician who attended this territory during that time), is is quite obvious that some special toxin infected the house, 43 Acton street, which not only infected the residents of the house but many outsiders who came in contact with it. The house, at all events, was extremely filthy, but that filth, *per se,* can generate pneumonia is incredible. Is it not probable that the first case of pneumonia (that of Mrs. S.) was caused by conditions which ordinarily give rise to pneumonia, but that the toxin or virus arising from this case, which would be harmless under better sanitary surroundings, attained such a degree of virulency in the rich culture medium which it found here, that, facilitated by the general depressing and devitalizing influence of filth on health, it effectively intoxicated all those who came under its local domination? This would, at any rate, be in conformity with the experimental work which has been done in this direction; for O. Weber,* in addition to other internal inflammations, produced diffuse pneumonia by injecting the blood of a febrile dog into another healthy one. Jens Schou† produced vagus pneumonia in animals and found an abundance of various bacteria in the alveolar and pleural exudation. From this he isolated three varieties of bacteria, which he cultivated in proper media. The first was an elliptic coccus of medium size, which, on being injected directly through the thoracic walls into the lungs, or introduced through the trachea, always generated a typical vagus-pneumonia. Culture fluids, which were sterilized before injection, always produced negative results. The other two were also cocci, differing somewhat in shape from the first, but were incapable of calling forth the same morbid process in the lungs. All the vagotomies

* "Handbuch der Chirurgie," Pitha und Billroth, i, S. 610.

† "Fortschritte der Medicin," 1835, No. 15, *Centralblatt f. d. med. Wis.*, 1886, S. 154.

were performed in a bloodless manner, and under strict antiseptic precautions, and the animals were kept in clean cages and received no food.

Now both of these experiments show that pneumonia may be called forth in animals without the agency of a single specific cause, *i.e.*, by the injection of simple febrile (non-pneumonic) blood, and by section of the vagi; and the latter demonstrates that after pneumonia is called forth by vagotomy it may be propagated in other animals through inoculation of the micro-organisms which accompany it. Now, when this evidence is taken in connection with the clinical proof above given, it clearly demonstrates how a disease not ordinarily infectious may become so under extraordinary unhygienic surroundings.

Anæsthesia Pneumonia: That ether and chloroform anæsthesia is frequently followed by irritation of the respiratory tract, and sometimes by acute pneumonia, is well known, although these disorders are generally regarded as the result of a local irritation of the air passages, especially in the case of ether. It is doubtful, however, whether a local surface action of these poisons accounts for the source of pulmonary embarrassment in all these cases; for it is well known that these anæsthetics exert a profound depression on the brain and nervous system, and hence it is quite possible that they produce the pulmonary disorder by impairing the integrity of the pulmonary nerve supply. At all events it is certain that lethal doses of these drugs, whether inhaled or ingested, have a special intoxicating influence on the respiratory organs of both animals and man. In some countries ether is used as a beverage in place of whiskey, and it is said that its poisonous action is similar to that of alcohol. Orfila* states that the administration of half an ounce of ether to a dog caused death in three hours. It was then found that the

* *Toxicology*, vol. ii, p. 531.

mucous membrane of the stomach was deeply inflamed, the duodenum slightly so, while the remainder of the alimentary tract was in a healthy condition. The lungs were engorged with blood and the heart contained black coagula.

Case 239. In the case* of a man whose death was caused by the inhalation of ether for about ten minutes, it was found that the mucous membrane of the whole extent of the bronchi was deeply injected, that the posterior part of the lungs was strongly congested with dark-colored blood, and that in the anterior part of the organs a frothy mucus was found, filling the air tubes.

Case 240. In another fatal case† from ether-inhalation, the membranes of the brain were found in a state of congestion and the lungs were engorged posteriorly.

Gerster‡ relates the following interesting cases in which acute pneumonia developed after the employment of ether as an anæsthetic:

Case 241. Female, aged 43, submitted to lumbar colotomy, under ether. Next morning she had a severe chill, which was followed by pneumonia of the entire right lung, and death on the fourth day.

Case 242. A male, aged 60, was castrated under ether, and two days later he was attacked by acute pneumonia. The temperature rose to 106° F., and death ensued.

Case 243. A male, aged 61, underwent the operation of inguinal colotomy under ether, and on the following day was attacked by pneumonia of the whole of the right lung.

Case 244. A male, aged 25, who had recently had pneumonia, took ether for the excision of the inguinal glands. Pneumonia reappeared in the same lung, in which it was located before, and proved fatal.

Case 245. A woman, aged 50, was operated upon under

* *Med. Gazette*, vol. xli, p. 432.

† *Med. Gazette*, vol. xxxix, p. 414.

‡ *New York Med. Record*, April 23, 1887, p. 453.

ether, for ruptured perineum. Pneumonia followed at once, and ended fatally.

Case 246. A male, aged 16, had the inguinal glands removed, under ether. Severe pneumonia followed, but ended in recovery.

That which is true of the pernicious influence of ether on the lungs applies with equal force to the behavior of chloroform, as may be seen from the following testimony:

Case 247. A male,* aged 42, swallowed two ounces of chloroform, and died in about six hours. After death the lungs were found much engorged with blood and subject to some apoplectic effusion. In Hausemann's *Jahrbericht*† it is stated that among the *post-mortem* lesions produced by chloroform are congestion and apoplexy of the lungs. Strassmann‡ found pulmonary and hepatic disintegration frequent complications in chloroform intoxication of animals. In Dr. Snow's§ collection of 50 deaths from chloroform in man, no account is given of the condition of the lungs in 20 cases, while in 5 cases these organs are reported healthy, in 13 they were moderately and in 12 they were markedly engorged.

It has also been shown in previous pages that alcoholic excess produces a strong predisposition to acute pneumonia, and it is also a common clinical observation that alcohol is an acute poison of the nervous system. Its characteristic toxic changes are observed in the brain, the oblongata, cranial nerves and throughout the peripheral nervous system. Delirium tremens frequently ends in pneumonia. Anstie says: ‖Nothing is more insidious than the occur-

* *British Med. Jour.*, May, 1866. *Amer. Jour. of Med. Science*, October, 1866, p. 571.

† 1872, S. 502.

‡ "The Fatal Effects of Chloroform," *Virchow's Archiv*, Bd. 115, Heft 1, p. 4, 1889.

§ "On Chloroform and Other Anæsthetics." By John Snow, M.D. London, 1858.

‖ "Reynolds' System of Medicine," vol. ii, p. 164.

rence of pneumonia in a subject whose nervous system is deeply poisoned with alcohol . . . . and it is most important, in every case of delirium tremens, that the chest should be periodically examined with care."

That alcohol is capable of producing pneumonia by poisoning the pneumogastric nerves is shown by a number of instances in the section which is devoted to a consideration of the action of this specific poison in its relation to pulmonary phthsis (see p. 61).

Symptoms.

Acute pneumonia is marked by symptoms of a decided character. Its onset is usually sudden, although in some instances it is preceded by a transient bronchial catarrh; and it shows itself in a decided rigor, almost as pronounced as that of a malarial chill. This is followed by fever, flushing of the face, nausea or vomiting, headache, delirium, frequently by convulsions in children, and sometimes in adults. The fever rises at once, and on the evening of the first day it may ascend to 103° or 104° F., and even higher, especially in children. The pulse, as a rule, is slow, labored and full, which, in the course of a few days, becomes weaker and more frequent. On the other hand, the respiration, in marked contrast to the pulse, is accelerated and may reach 40 or 50 a minute, or a higher rate in children. In the beginning there may be very little or no cough, and which is husky and dry. The expectoration during the first few days is small in quantity and consists of frothy mucus.

On the third or fourth day it becomes thick, viscid and tenacious, and contains a varying number of red-blood corpuscles, which give it a rusty color, varying from light-red to brownish-black. In marked adynamic states, as in the pneumonia of alcoholics, or in that of the aged, the ex-

pectoration may assume a prune-juice color, which indicates a serious prognosis. In cases of marked pulmonary engorgement severe hemoptysis may take place, and even in milder cases pure blood is not infrequently expectorated. The sputum may also contain fibrous casts of the smallest bronchial tubes. Expectoration may be wanting entirely in cases of great nervous depression, as in the insane, and in the very aged. Pain, which is usually found in some part of the chest, is a constant concomitant of pneumonia. If pleurisy co-exists the pain is of a stitchy character, and is aggravated by deep breathing. It is not always confined to the chest, however, for it may be located in the lumbar or in the iliac region, and if in the latter place the real nature of the disease may be overlooked and be mistaken for typhoid fever. In the beginning the tongue is usually covered with a whitish coat, which, in the course of a few days, changes to a dry, brown color. If the nervous symptoms predominate actively, as is the case when stupor and delirium supervene, the coating of the tongue changes to a thick, black, dry crust. The condition of the tongue is a good indication as to the progress and gravity of the affection. In severe cases the countenance becomes flushed and the lips and cheeks assume a bluish color. The skin is usually hot and dry, and very frequently remains so until the crisis is reached. In some cases the skin continues moist throughout the fever-period. The urine is scanty, its color somewhat dark, and of a high specific gravity. The alkaline chlorides, especially the chloride of sodium, diminish, and during the most severe period of the disease they vanish.

During the course of the disease the fever, with a slight morning remission, continues at a uniform elevation, the cough becomes more pronounced, the respiration accelerated, the pulse more frequent, weaker and irregular, attaining a rapidity of from 120 to 140, and in children exceeding these

figures; twitching of the muscles occurs frequently, urine and fæces are voided involuntarily in bed; delirium and coma may supervene, until the crisis is reached, from the fifth to the eleventh day. Just before this point the temperature may rise, and many of the leading symptoms may assume a more threatening aspect than before; when suddenly the patient breaks out in a profuse general sweat or a copious discharge of urine or diarrhœa may occur. The temperature drops to a point near, or below, the normal, and the whole outlook of the case is changed. In the course of a few hours the pulse becomes regular again and declines in frequency, the pain disappears, the breathing is comfortable, the expectoration changes to a lighter color, the chlorides return in the urine, and the countenance returns to its wonted appearance.

Not every case of acute pneumonia that ends in recovery terminates in crisis. Quite a considerable number of patients get well by passing through one or two apparent relapses, in which the pneumonic infiltration lights up afresh in some hitherto uninvaded area, and in which there is a recrudescence of fever, an extremely frequent pulse, a blunted condition of the senses, stupor or delirium, and a gradual recovery by lysis in two or three weeks. Other cases get well in a shorter time by lysis.

Acute pneumonia in the aged, or in those of a greatly depraved nervous constitution, is accompanied by symptoms of a very adynamic type. Very often the symptoms representing the pulmonary affection are entirely overshadowed by those pertaining to the nervous system. Cough and the characteristic expectoration are probably wanting, and no marked dyspnœa or pain exist. The increased respiration-rate may be attributed to the rise of temperature, and unless great care is exercised in regard to the physical signs of the lungs the case may be mistaken for one of typhoid fever.

The symptoms of acute pneumonia in many alcoholic sub-

jects resemble those of delirium tremens more than those of pneumonia from the very outset of the disorder. Even in cases in which pneumonia occurs in persons of intemperate habits, and in which it begins and pursues an ordinary course, it is a rarity not to find symptoms of delirium tremens developing before the termination of the illness. Patients of this kind are restless, cheerful, talkative, restrained with difficulty, suffer no discomfort, often have to be fastened to the bed, observe all kinds of small animals, converse with visionary friends or enemies, in the ceiling or beneath the bed, pick incessantly at the bed clothing and shriek and struggle until the point of exhaustion is reached. Usually there is very little cough or expectoration, the fever is low, the pulse, if at all firm in the beginning, becomes weak, the tongue is dry, crusty, and black, and coma supervenes and death takes place frequently on account of pulmonary paralysis.

Physical Signs.

On inspecting the chest it will be observed that there is less freedom of respiratory motion on the affected side. When both bases are implicated in the pneumonic process the costal breathing is exaggerated. Palpitation demonstrates increased resistance, exaggeration of vocal vibration and the intensified transmission of the cardiac impulse over the affected lung area. Vocal resonance is also more pronounced over the part of the lungs which is involved. During the first stage of pneumonia the percussion note is usually of a dull, tympanitic quality, while in the second stage, or in that of hepatization, it becomes flat and there is also decided increased resistance to the plexor finger. In children dulness is frequently wanting. As a rule, prolonged expiration is one of the earliest signs heard in acute pneumonia, although crepitation precedes it sometimes. Bronchial respiration is also present in most cases. Bronchophony ex-

ists from the beginning, although it is a less reliable sign than prolonged expiration or bronchial breathing. If an accumulation of catarrhal secretion fills up the bronchial tubes to such an extent as to prevent the admission of air all the auscultatory sounds will be suppressed in the region involved, but will reappear so soon as the tubes are permeable again. In complete consolidation, crepitation ceases, if it existed; while prolonged expiration, bronchial respiration and bronchophony persist. During the stage of resolution a fine, moist râle appears in places which were previously in a state of complete consolidation, and which is called the "crepitatio redux." Although pleurisy is almost a constant accompaniment of acute pneumonia, in one stage or another, pleural-friction sounds do not show themselves except in cases where the pleurisy is of sufficient extent to give rise to an effusion.

Differential Diagnosis.

Acute pneumonia is often overlooked in children. This is partly due to the fact that in the latter the disease is often ushered in with convulsions and vomiting. The diseases with which it is most liable to be confounded are pleural effusion, acute tuberculosis and pulmonary œdema, the prominent differential symptoms and signs of which are given in the following tabular view:

Acute Pneumonia.

1. Decided initial chill.
2. Convulsions in children, sometimes in adults.
3. Fever, high from beginning.
4. Pulse slow at first, cardiac apex not displaced.
5. Breathing accelerated.
6. Expectoration rusty, bloody, prune-juice color.
7. Delirium prominent.
8. Vocal resonance, and fremitus increased over affected area.

9. Dulness in adults, absent in children.
10. Crepitation, prolonged expiration, bronchial breathing.
11. Râles not movable by cough.
12. Redux crepitus, appears in resolution-stage.
13. Ends in crisis or lysis.
14. No tubercle bacilli present.

Pleural Effusion.

1. No decided initial chill.
2. Nervous disturbance rare.
3. Fever, medium and irregular.
4. Pulse frequent throughout, cardiac impulse displaced.
5. Breathing accelerated.
6. Expectoration scant, not discolored.
7. No delirium.
8. Vocal resonance and fremitus diminished over seat of effusion.
9. Dulness constant.
10. Respiratory sounds and râles suppressed.
11. Hypodermic exploration shows fluid.
12. Tubercle bacilli may be in fluid.

Acute Pulmonary Tuberculosis:

1. No decided chill, chills irregular throughout.
2. Nervousness marked.
3. Fever, irregular and high.
4. Pulse frequent.
5. Breathing increased.
6. Expectoration profuse, puriform, nummular and bloody.
7. Delirium may be present.
8. Vocal resonance and fremitus uncertain.
9. Dulness may be entirely absent.

10. Large, moist and crepitant râles; roughened respiration, usually bilateral.
11. Tubercle bacilli present.

Pulmonary Œdema:

1. No decided chill.
2. No special nervousness.
3. Fever rarely present.
4. Pulse frequent.
5. Dyspnœa marked.
6. Expectoration; frothy serum, may be bloody.
7. No delirium.
8. Vocal resonance and fremitus unsatisfactory.
9. Dulness present, usually bilateral.
10. Bubbling and sibilant râles on both sides.
11. No tubercle bacilli present.

It will then be seen that acute pneumonia differs from pleural effusion, acute pulmonary tuberculosis and pulmonary œdema in its decided initial chill, its high fever from the outset, its slow pulse in the beginning, its frequent cerebral complications, especially in children, its rusty and sometimes prune-juice expectoration, its crepitation, its prolonged expiration and bronchial breathing. In common with pleural effusion it has unilateral dulness, but in the latter affection the cardiac apex is displaced toward the opposite side, and exploration with the hypodermic needle will make the diagnosis absolutely certain.

Prognosis.

The prognosis of acute pneumonia is governed largely by the degree of fever, pulse and respiration-rate, the extent of pulmonary complication and the general condition of the nervous system. Fever, after all, is only a relative sign, inasmuch as it is, in great part, an indication of the degree of

vital resistance on the part of the patient's constitution. This seems to be true when it is excessively high, as well as when it is too low; and it appears that the safety point lies between these two extremes. A pneumonic adult, with an average temperature of 100° F., or less, has a less hopeful outlook than another with the same amount of pulmonary disturbance and with a temperature of 104° F. A low temperature in acute pneumonia indicates serious exhaustion of the nervous system, and this is found in the pneumonia of alcoholics, that of the insane, and in that of others whose nerve integrity is greatly impaired.

This deduction in regard to the prognostic influence of fever in this disease is practically corroborated by Dr. Wilson Fox's statistics.* He shows that out of a total of 353 cases of acute pneumonia that the mortality from 107° to 110° was 100 per cent., from 106° to 107°, 42.8 per cent., from 105° to 106°, 18 per cent., from 104° to 105°, 7.4 per cent., from 103° to 104°, 17.6 per cent., and under 103°, 36.9 per cent.

A pulse-rate of 120 in the adult is to be regarded as a danger sign, especially if the fever is moderate. In children in whom the pulse is very impressible no special anxiety need exist unless it reaches a rate of 150 or 160 in the minute. Intermittency and irregularity of the pulse are serious signals, although they are not infrequently found in the aged.

A frequent respiration-rate is not such an unfavorable indication as is a laborious dyspnœa, in which each inspiration is accompanied by pain and distress in the epigastrium.

When the expectoration is wanting from the beginning, excepting in children, who frequently swallow it, and especially when at the same time there are heard moist râles in the lungs, or when this is of a prune-juice color, a grave

* "Diseases of the Lungs and Pleura," p. 352.

prognosis must be given. Bloody sputum, or even hemoptysis, have a much less serious indication.

A moist, although coated, condition of the tongue is a favorable sign, and a dry, black and crusty coating of this organ must always be regarded with suspicion. The latter condition often indicates serious cerebral complications.

CHAPTER XXI.

A CLINICAL VIEW OF ACUTE PNEUMONIA FROM THE STANDPOINT OF NERVOUS DEVELOPMENT.

In discussing the pathology of acute pneumonia sufficient reasons were given for believing that disintegration of the pulmonary nerve supply bears a causative relationship to this disease; and in furtherance of the same idea an effort will be made here to show that the development of the various phases of acute pneumonia is largely governed by a lack of stability on the part of the nervous system while this is going through its periods of evolution and dissolution. In other words, the object is to demonstrate* (1) that acute pneumonia has its age-periods, which are largely governed by an instability or a want of resistance on the part of the nervous system; (2) that other diseases arise as complications of pneumonia, because the morbid impulses of the latter radiate through and compromise neighboring nerve tracts; (3)that the relative frequency with which these complications arise depends probably on the difference in the biologic ages of the nerve centers which are involved; and (4) that the pathology, physical signs and symptoms of pneumonia vary at different ages on account of the varying irritability of the nerve tracts that are implicated.

It is hardly necessary to remind the reader of the fact that the brain and the higher nerve centers are extremely complicated organs, and that the oblongata, to which frequent reference is here made, is composed of parts which are

* The substance of these remarks was published in the *New York Medical Journal*, Dec. 31, 1898.

most diversified in structure and in function. The latter is the organ which Foster defines as the link between the brain and the spinal cord, and it comprises the respiratory center, the convulsive center, the cardio-inhibitory center, the vasomotor center, the deglutition center, the motor center for the stomach, and at least two or three heat centers. In a measure, each of these centers has an independent functional existence and controls the function of the organ over which it presides.

In this connection it must also be remembered that different organs of the body, as well as different parts of the same organ, mature or attain full development at different biologic periods, and that immature organs or parts of organs do not resist disease as readily as those which are more fully developed; and hence we find that the various organs, and structures of the body are more liable to disease at one time than at another. Thus, for example, before the age of seven, the period during which there is the greatest activity in brain-growth, convulsions, pneumonia, tubercular meningitis, hydrocephalus, rickets, etc., are most liable to occur; from seven to fourteen years, when, according to Clouston, "muscular motion becomes co-ordinated fully with emotion," chorea, asthma, epilepsy, megrim, strabismus, are most likely to develop; while, after fourteen, during the period of the beginning of reproductive activity, hysteria, chlorosis, insanity, phthisis, dipsomania, acute rheumatism, valvular diseases of the heart, etc., are most prone to come to the surface.

In confirmation of the statement that acute pneumonia has its age-liability it may be said that statistics demonstrate that by far the greatest number of deaths occur from this disease during the first five years of life. Among the records which illustrate this is the Report* of the Board

* Thirty-second report upon the births, marriages, and deaths in the State of Rhode Island for the year ending December 31, 1884, p. 44.

of Health of Rhode Island; of the 5,425 decedents from pneumonia during twenty years, from 1865 to 1884, 1,723, or 32 per cent., were under five years of age.

What is the cause of this excessive death-rate from pneumonia in early life? Is it because there is greater sensitiveness of the pulmonary epithelium, which entails a disposition to catarrhal disease of the lungs, or is it because the child's brain and nervous system are in a very unstable and impressible condition? That it is chiefly the greater impressibility of the infantile nervous system that plays a fundamental rôle in the production of pneumonia is probably shown by the fact that its greater prevalence runs in a line parallel with that of convulsions during the first two decades of life—the latter being, as is well known, a disease of an exclusive neurotic nature. In the following chart, which shows the distribution of acute pneumonia and convulsions in age-periods in the city of Philadelphia for a period of three years, it is illustrated that both of these diseases reach their maximum death-rate before the age of five, and their minimum death-rate between the ages of five and ten. Now the highest death-rate of these two diseases corresponds exactly with the greatest rapidity of growth in the child's brain; for according to Boyd the greatest increase in the brain of the infant occurs before the seventh year, and that at the end of this period it has attained 90 per cent. of its bulk. Now in keeping with the general biologic law that an organ is most immature, and most liable to disease, when undergoing structural and functional activity, the period of maximum liability to pneumonia and convulsions, as shown in the above chart, corresponds with that during which the brain and higher nerve centers are subject to the greatest developmental activity. After the tenth year other causes of pneumonia than those above referred to become operative and divert the line of pneumonic deaths from that of convulsions, as is seen in the illustration.

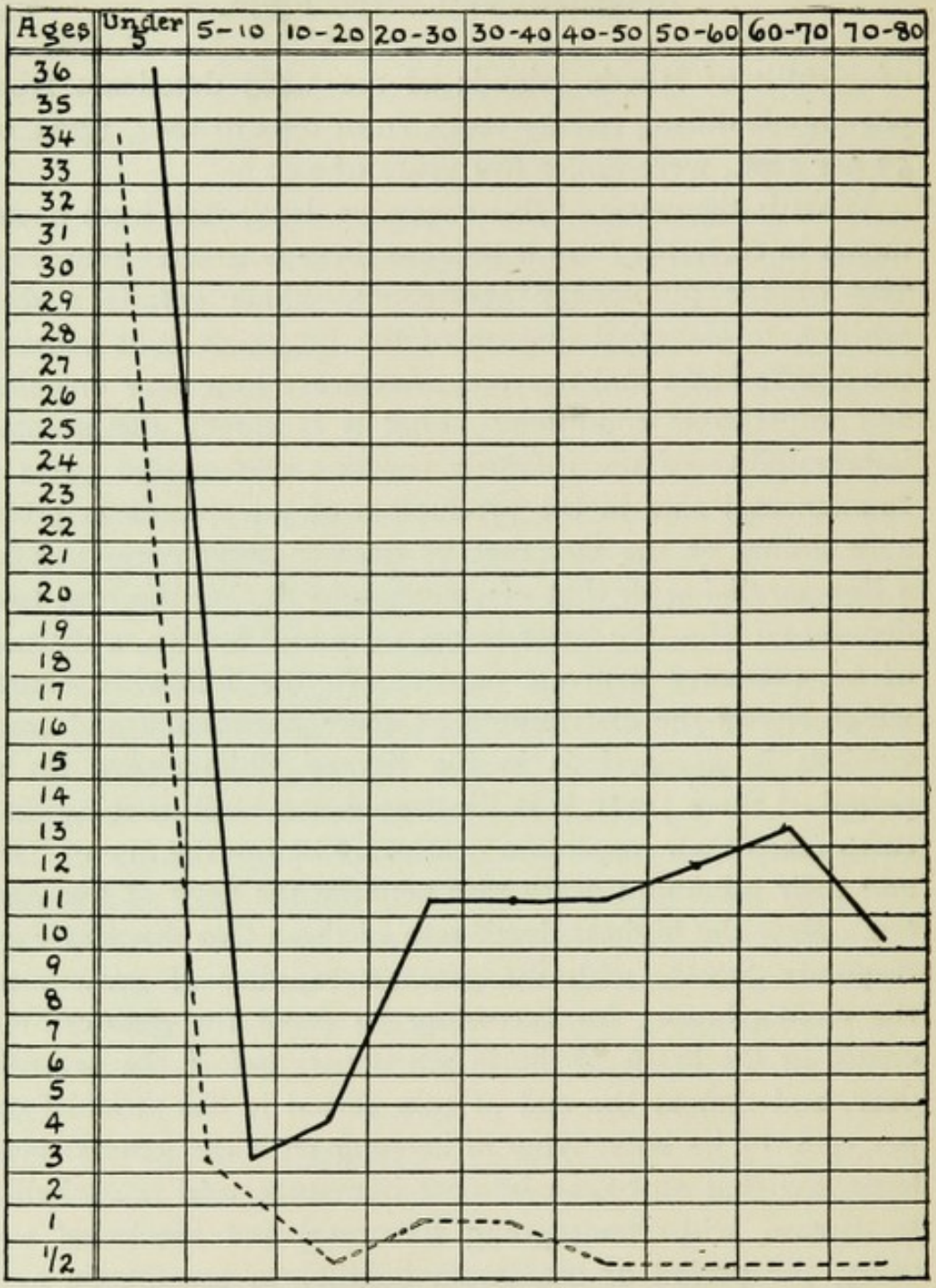

Fig. 41.—Chart showing the distribution of acute pneumonia and convulsions in age-periods in the city of Philadelphia for a period of three years, the number of cases in each age period being reduced to the least common multiple. The continued line represents that of pneumonia, and the dotted line that of convulsions.

It may be taken for granted, then, that both pneumonia and convulsions are closely bound up with the nervous system, and that both diseases prevail more extensively in childhood, because during that age-period the nervous system is most unstable and impressible. Now the interesting question arises, Why does this intimate relationship exist at this time of life? Is it not because anatomically the respiratory center and the center which coördinates muscular motion are in such close proximity to each other that disturbance in one will readily radiate to the other and produce a disturbance there simultaneously? If this conclusion is accepted we have an adequate explanation as to the reason of the frequent concurrence of the two diseases at the same time in the same individual. Whether the degree of instability is exactly the same in these centers, and whether a disturbance is propagated with equal facility from one to the other, are questions which will be considered hereafter.

In the second place, why is it that a sudden and often an enormous rise of temperature occurs as a constant concomitant of infantile pneumonia and convulsions? Now the argument which seeks to establish that the greater immaturity and irritability of the child's nervous system are responsible for the development of the two latter diseases applies with equal effect to the want of equanimity in the centers which control the production and dissipation of heat at the same time of life. The coördinating power of these centers—the most important of which are located at the base of the brain—is disorganized by the wave of disturbance which spreads from the centers already diseased, and results either in an overproduction or retarded dissipation of heat, or in both.

In the third place, the pulse is greatly increased in infantile pneumonia. In explanation of this pnenomenon it may be stated that electric irritation of either vagus produces temporary inhibition of the heart's movements. In a short

time, however, the inhibitory effects pass off, even though the irritation is continued, and the heart resumes its beats with greater frequency than before, although it sustans a loss in tone and vigor. Now that which is true of the heart's movements under these conditions is probably also true of its behavior in acute pneumonia. Irritation of the cardiac center, radiating primarily from the respiratory center, is reflected along the course of the cardiac branches of the vagi to the heart, but, owing to the greater excitability of the undeveloped nerve control of the infantile heart, the inhibitory effects are not pronounced and of short duration. In the adult, however, where the nerve influences are more stable, the inhibitory effects, as shown in the slow pulse, are usually pronounced in the early stage of pneumonia; but at a later period the inhibitory power is likewise lost, and, as in the child, the heart gives way to the predominating accelerator impulse.

In the fourth place the vomiting, which so frequently ushers in acute pneumonia, or accompanies its earlier stages, especially in childhood, must not be left out of consideration. Like many of the other symptoms of pneumonia it is very probable that this is of central and not of peripheral origin, for, according to Foster, the vomiting center is located closely to the respiratory center, and hence it is easy to perceive that the impulses of disease are readily communicated from the latter to the former.

In the fifth place, is the accelerated respiration-rate of acute pneumonia, and especially of infantile pneumonia, always due to extensive infiltration of the lungs, and to a consequent interference with the process of blood-aeration, or to some other cause? Taking into consideration the clinical fact that it is not always those cases of pneumonia with the most extensive infiltration in which the greatest difficulty of respiration exists, and that at the same time the respiratory center is involved in this disease, it is quite probable

that in the adult the quickened breathing depends as much on an irritated and semiparalyzed condition of the respiratory innervation as it does on pulmonary infiltration or any other discernible cause; and that in the infant the greater rapidity of breathing is accountable on the score of imperfect development of the respiratory nervous system.

INFLUENCE OF NERVE-CENTER-AGE ON THE DEVELOPMENT OF PNEUMONIA. Granting that the basal nerve centers in the child's brain are so irritable and unstable that disturbance of one may lead to pneumonia, and simultaneously involve the equilibrium of the convulsive, vomiting, fever, and circulatory centers, the interesting question arises whether in the adult, in which some of these accompanying disorders of pneumonia are wanting, or exist in a much milder degree than in the infant, some of these centers acquire a greater degree of resistance to the influence of morbid forces than others, and on this account become less liable to fall a prey to disease? Reference has already been made to the general biologic law that, by reason of a firmer equilibrium, the older organs are less liable to disease than those of less mature age. Now it remains to be shown whether there is any difference in the biologic ages of these centers? On the surface it seems as if nerve areas in such close structural proximity to each other could not differ much in respect to their ages. This may be true, or at least it is difficult to prove that it is not true, yet notwithstanding this it is quite clear that each center asserts its own functional independence, and that the various organs over the functions of which these centers predominate are of different biologic ages, and it seems that this difference must naturally be reflected on each controlling center.

It is well known that in skull-less, or brainless animals, the important organs of the body, like those of circulation, digestion and respiration are co-ordinated by separate ganglia with intercommunicating nerve-fibers; while in the

skulled animals these ganglia, although not being deprived of their influence on these organs, are brought into direct relationship with the brain through the medium of the pneumogastric nerves. The viscera, which are supplied by the latter, are, therefore, under the domination of a double nerve influence, *viz.*, that which comes from the ganglionic or spinal nervous system, and that which comes through the medium of the vagi from the highest co-ordinating center in the body. Now there is no question that biologically the organs of circulation and of digestion precede those of respiration, and hence, in accordance with the doctrine that the older organs are less liable to disease than the younger, provided other things are equal, it follows that the lungs offer less resistance to adverse forces than the heart and stomach. It is also very probable that this difference of vital resistance in these organs is impressed on their nerve-supply; for, while the nerve dominates the organ to which its fibers are distributed, the organ in a measure reciprocates this favor by modifying the growth and function of its nerve. This is certainly shown in the evolution of the phrenic nerve in the embryo of the higher animals. This nerve arises in the cervical division of the cord, but, according to the highest authorities, it is very short in its earliest stages of development, because the diaphragm is located very near the exit of the nerve at that time. Gradually the diaphragm recedes downward and is followed by the elongating nerve until it reaches its permanent destination. Here, then, is an instance in which the length, course and function of a nerve have been markedly modified by the change of position of the organ to which it is distributed, and there is every probability that other nerves, among which may be included the pulmonary branches of the pneumogastric, have also undergone a somewhat similar modification. For this reason it may be inferred that an organ, as a whole, reflects its impress on the nerve-center

from which it derives its impulses, and that the strength and weakness of the one become the strength and weakness of the other.

From what has been said thus far it is evident that the popular doctrine that the heart is very much more liable to embarrassment than the lungs is not supported by the facts of biology, and that instead of being more predisposed to disease it offers greater resistance to it than the respiratory organs. Neither is such a view favored by experimental or clinical experience. Ample evidence demonstrates that narcotics, like ether, chloroform, alcohol, opium, etc., the acute intoxication of all of which is confined more to the brain than it is to the peripheral nervous system, as a rule, arrest the action of the lungs before that of the heart. The same general relationship holds true between disease and injury of the brain and the respective vitality of these organs, as is shown by what is said on respiratory paralysis, on page 138.

The practical question of the relative viability of the heart and lungs in acute pneumonia is one of greatest importance, because at present there seems to be a tendency to direct more attention towards a prevention of cardiac than of pulmonary collapse in the treatment of this disease. Indeed this trend is so strong that one might be led to believe that pneumonia is a disease pertaining to the heart rather than to the lungs, and that all that is required to resolve the pneumonic consolidation is to goad the heart with stimulants in order that it may pump the blood through the obstructed and impervious pulmonary capillaries. It is obvious, however, from a comprehensive study of the lethal tendency of this disease that its most threatening danger comes from a defective supply of nerve force to the lungs—from a pulmonary nerve exhaustion, which manifests itself not so much in a simple frequency of breathing, as it does in a frequent, laborious and shallow respiration.

That which is true of the greater resistance of the heart to disease on account of its greater biological age than the lungs is also true of the stomach and the balance of the digestive organs. This is very well shown by the fact that, as a rule, after the first wave of disturbance accompanying pneumonia has passed over these organs and their nerve supply, and has caused vomiting and diarrhœa, they settle back to their wonted stability and discharge their functions almost as perfectly as they did before the attack, while neighboring functions continue in a state of perturbation.

Influence of Nervous Resistance in Modifying the Symptoms and Physical Signs of Acute Pneumonia.

It is well known that the symptoms and physical signs of acute pneumonia vary widely under different conditions. Thus latent or senile pneumonia is as far apart in this respect from the pneumonia of childhood as night is from day, and the difference between the pneumonia that accompanies influenza and that of a typical croupous or catarrhal pneumonia is also frequently pronounced. Let us contrast, for example, the symptoms of pneumonia in the aged with those of the same affection in infancy in these respects. In the former, as a rule, the temperature is low, and of irregular rise and rhythm; the cough is slight and inconsequent, the expectoration is scant and of a rusty or prune-juice color, breathing is not greatly accelerated, the pulse is usually slow and weak, and if delirium is present, which is generally the case, it is of a low muttering and moaning character. In the latter the fever rises suddenly and mantains itself at a constant elevation, the cough is persistent and frequent, the expectoration may be wanting, but if present is of a frothy character and rarely discolored; respiration is exceedingly active, often reaching a rate of sixty

or seventy a minute, the pulse is small and so rapid sometimes that it becomes uncountable, and active delirium and convulsions are common complications. In the one the nervous system seems to be narcotized, as if overcome by opium intoxication, or laboring under a profound shock, and all its symptoms appear to be subdued and passive; while in the other the nervous system is apparently suffering from a wave of excessive irritability and instability, and behaves as if it were under the control of large doses of strychnine, and all the varied manifestations of the disease are presented in a violent and active form.

That the difference between these two forms of pneumonia is largely accounted for by a difference in irritability of the brain and nervous system at the two extremes of life is confirmed by the fact that in the pneumonia of alcoholism, of the chronic insane, of the feeble-minded, of the deaf mutes, etc., the symptoms run a similar course to that which is found in those of senile pneumonia. The poisonous action of alcohol destroys the irritability of the nervous system and reduces its physical level to that which obtains in chronic insanity, idiocy, etc., and which is analogous to that which is found in the aged. Want of nerve irritability also accounts for other clinical variations in pneumonia. Thus, for example, gangrene and abscess of the lungs, slow convalescence, etc., are more liable to follow pneumonia in persons with a depraved and vitiated nervous system than in those of opposite conditions.

From what has thus been so imperfectly said it is obvious that by viewing acute pneumonia from the standpoint of development of the nervous system and not from that of its local origin in the lungs, its true pathological relation, together with many of its therapeutic bearings, receive a scientific and truthful interpretation. So far as the author is able to see no other theory can unify and harmonize the varying factors of this disease so well, can so satisfactorily

account for its preponderating death-rate in early infancy, can so definitely explain the evolution of many of its prominent symptoms and the orderly succession of many of its nervous concomitants, can so clearly show why some of these appear early and others late in the course of the disease; why some are associated one with the other at one time and not at another; why its form varies at different ages and under different conditions; why some of its symptoms are prominent at one age and perhaps latent or absent at another; and, finally, no other theory points out so forcibly that the fundamental position of pneumonia naturally belongs to the family of neurotic diseases.

CHAPTER XXII.

THERAPEUTICS OF ACUTE PNEUMONIA.

WHAT, then, are the therapeutic indications of acute pneumonia, when viewed from the standpoint which has been advocated in the foregoing pages? If the primary fault of this disease lies in a disordered and discordant state of the nervous system, it is quite evident that this defect should receive immediate attention; but the preliminary stage of acute pneumonia is, as a rule, ushered in so precipitately and is usually of such short duration that engorgement and even infiltration of the lungs may, and generally, occur before the disease is properly recognized, or before medical aid is sought. Obviously, therefore, its secondary effects demand as much urgent therapeutic attention as its primary lesion, and our remedial efforts resolve themselves accordingly (1) into those which seek to allay perturbation of the brain and nervous system, and (2) into those which counteract and prevent pulmonary engorgement and infiltration, support the nervous system in general, and nourish the patient.

1. MEASURES TO ALLAY NERVOUS IRRITABILITY.

The nervous symptoms which mark the initial period of acute pneumonia are rigor, headache, fever, restlessness, dyspnœa, vomiting, convulsions, etc., and whenever a number of these are associated, especially in children who are entering on or who are passing through dentition, the likelihood is that pneumonia is impending. Now, what measures

are most appropriate to combat this tendency? Both experimental and clinical data show that cold has the power to reduce nervous excitability, and that no remedy meets this indication better and more effectively than cold applied to the head and chest in the form of ice. At least two rubber bags, filled with ice, should be applied to the head, and an equal number, or more, to the chest—one or two on each side—as a preliminary step. In the adult an injection of morphine, ¼ of a grain, may be given at the same time to produce sleep. As a result of this treatment the fever abates, the respiration-rate is reduced, sleep follows, restlessness subsides and there is good practical reason for believing that in certain cases where the cold is applied early enough the disease is strangled in its incipiency.

2. Measures to Control Pulmonary Engorgement and Infiltration.

No matter how divergent the external manifestations of acute pneumonia may be, there is, as has already been demonstrated, one pathologic groundwork which is primarily present in every form of this disease, *viz.*, engorgement of the pulmonary capillaries. Under the influence of varying internal and external conditions this may be said to be the soil from which develop the different forms of this affection. Now, in addition to the direct sedative influence of cold on the nervous system, which has been noticed already, it possesses the property of contracting the caliber of the blood-vessels and of allaying circulatory excitement and for these reasons it is the most efficient measure to dissipate and to resolve the local fulness and infiltration of the lungs when so administered. Objections have been urged against the local use of cold to the chest in treating pneumonia on the ground that its refrigerating effects do not extend deep enough to influence the pulmonary infiltration and that the

reflex action of cold general baths are preferable. Any suspicion that cold does not have sufficient penetrating power to reach the lungs should be allayed, however, by the evidence coming from the experimental work* of Dr. Schlikoff, which shows that if a thermometer is introduced into different organs and cavities of the body, and ice applied externally at the same time, the local temperature sinks a number of degrees in most instances, while the axillary temperature either remains stationary or falls but slightly, as will be seen from the following references to her work. Thus, by applying the ice-bag to the cheek, while the thermometer was in the mouth, the temperature fell nearly four degrees in one hour. In the hollow of the hand, while the ice was applied on its back, it fell five degrees in an hour. Introduced though a fistulous opening into the pleural cavity, while ice was applied to the chest, it fell over three degrees in an hour. In the vagina, when the ice-bag was applied over the pubic arch, it fell nearly a whole degree in an hour and a half.

Cold has been employed in the treatment of acute pneumonia for many years in Germany, and, so far as the author is aware, the illustrious Niemeyer was the first to apply this agent immediately to the chest for the purpose of reducing the activity of the local inflammatory process in the lungs. In his great work† this author makes use of the following language in describing the influence of local cold applications in the treatment of pneumonia:

"I have made extensive employment of cold in the treatment of pneumonia and, relying upon a large number of very favorable results, can recommend this procedure. In all cases I cover the chest of the patient, and the affected side in particular, with cloths which have been dipped in

* *Deutsches archiv f. klin. med.*, vol. xviii, p. 577.

† Niemeyer's "Text-Book of Practical Medicine," vol. 1, p. 185. New York: D. Appleton and Company, 1874.

cold water and well wrung out. The compresses must be repeated every five minutes. Unpleasant as this procedure is in almost all cases, yet after a few hours the patients assure me that they feel a material relief. The pain, the dyspnœa, and often the frequency of the pulse, are reduced. Sometimes the temperature goes down an entire degree. My patients often retain this surprising condition of improvement throughout the entire duration of the attack, so that their outward symptoms would hardly lead one to imagine the grave internal disorder. The relatives of the patient, too, who do not fail to perceive the improvement, now readily assist in the treatment to which at first they were opposed. In a few cases, and only in a few, the use of cold affords no relief, and the troublesome manipulation for its application increases the distress of the sufferers so much that they refuse to keep it up. In such cases I have not insisted upon the further application of cold.

"In the hospital at Prague every pneumonia is treated with cold compresses and, according to the statements of Smoler, it is exceptional for a patient not to feel material relief from this treatment. As, however, I have never succeeded in cutting short a pneumonia by means of cold applications, I should only ascribe a palliative influence to their use had not the duration of the disease in many instances been decidedly shortened and the convalescence hastened by means of their energetic and methodical employment. In fact, in but few cases have we seen the disease delay its departure until the seventh day. Many have improved on the fifth, and a very large number as early as the third day; nay, I have repeatedly found it impossible to keep patients with recent pneumonia in hospital for a longer period than a week. Cold is rightly regarded as one of the most efficient antiphlogistics in inflammation of external organs. Its action is directly tonic upon the relaxed tissues and dilated capillaries."

For several reasons it seems that the ice-bags are more practical than the cold compresses, which are recommended by Niemeyer. In the first place they are dry, and do not wet the bed clothing; second, their cold effects are more uniform and decided, because they retain a low temperature longer; and, third, it is not necessary to change them oftener than every two or three hours unless the patient has a high degree of fever. This not only saves work, but obviates the "troublesome manipulation" referred to by this writer.

How, then, is cold to be applied, and how long must it be continued? The affected area is to be surrounded with ice, contained in rubber bags, which are wrapped in thin towels. If there is a great deal of fever the towel is to be omitted. The number of ice-bags which are to be applied in any case depends on the degree of fever which is present and on the size of the area which is inflamed. If the fever is not very high and the disease is confined to the front or lateral base on one side only, two bags should be applied over this area, and as a precautionary measure another bag should be applied to the opposite base; for pneumonia appears to have a tendency to involve the corresponding area on the other side. If the exudation extends to the side and back, and for a considerable distance upwards, three or four bags should be applied front, laterally, and as far back as possible. If the affection is extensive, and involves both lungs, put on as many ice-bags as are necessary to cover the whole area. One of the worst patients in the author's experience required nine bags to cover the whole chest, sides and front, and at the same time two bags were applied to the head. The length of time during which cold is to be applied depends in a great measure on the degree of fever which is present. If the temperature falls to or near the normal point, and shows a tendency to remain there, the ice may be gradually removed. It is best, however, not to remove all the ice-bags, even though the temperature is nor-

mal, unless the crisis is at hand without question, because if they are all taken away before the proper time the temperature will rise again and it is brought down with greater difficulty the second than it was the first time. There is more risk in taking the ice off too soon than in allowing it to remain too long. Sometimes the application of ice to a certain area is followed by amelioration of the physical signs and symptoms, when all at once the temperature makes an ascent. Care should be taken to find out whether or not the inflammation has extended to a new field, and if this is found to be the case it should be followed up by applying more ice-bags to the newly invaded territory, and the temperature will in all probability fall.

What, then, is the local action of cold on the pneumonic process? The most apparent lesion in the lungs is, as has been observed already, an enormous distention of the pulmonary capillaries, with partial or complete stasis of blood in these vessels, exudation of fluid-constituents of the blood and proliferation and accumulation of epithelial cells and diapedesis of red and white blood-corpuscles in the alveoli and bronchioles. Experience teaches that cold not only contracts the small blood-vessels, but that it also dissolves an exudate, or an infiltration. Such at least is the impression which has been made on the author's mind. For example, the application of ice to a pneumonic area which is wholly devoid of vesicular sounds, and which has a flat percussion note and bronchial breathing, indicating beyond doubt that the process has passed beyond the stage of engorgement and into that of exudation or of infiltration, will, in the course of twelve or eighteen hours, develop a new group of physical signs, such as crepitation, reappearance of the vesicular murmur, diminution of flatness, etc. This has not only been observed by the author, but by others, notably among whom is Dr. F. Lees, who had a very extensive experience in the use of ice in this disease and to whom the author feels deeply

indebted for his early contributions to this subject. He says:* "In many cases I noticed a striking arrest in the development of the physical signs," and that the ice-bag "distinctly tends to repress the inflammatory process in the lung."

Supposing that in a very grave case of acute pneumonia cold had been applied after the manner here prescribed for thirty-six or forty-eight hours without giving relief to the difficult breathing, the distress in the chest, the cyanosis, the pulsation in the neck, and perhaps œdema of the extremities—marked indications of an obstructed pulmonary circulation—what can be done to reinforce the influence of the cold applications? In such an emergency there is no question that the old and almost forgotten art of venesection will give more assistance than anything else and should be employed without hesitation. Its effects under these conditions are similar to those which are sought to be brought about through the agency of cold, *viz.*, an unloading of the over-distended cardio-pulmonary circulation and thus affording an opportunity for the contraction of the pulmonary capillaries.

Niemeyer lays great stress on the importance of bleeding in such an extremely perilous stage of pneumonia. He says†: "When collateral œdema in the portions of the lung unaffected by pneumonia is causing danger to life the pressure of the blood is reduced by bleeding; and, by prevention of further transudation of serum into the vesicles, insufficiency of the lung and carbonic-acid poisoning are averted. Whenever the great frequency of respiration in the commencement of pneumonia cannot be traced to fever, pain, and to the extent of the pneumonic process alone, as soon as a serous, foaming expectoration appears, together with a respiration of forty or fifty breaths a minute, and

* *Lancet*, Nov. 9, 1889, p. 894.

† *Op. cit.*, vol. i, p. 186.

when the rattle in the chest does not cease for a while after the patient has coughed, we ought at once to practice a copious venesection, in order to reduce the mass of blood and to modify the collateral pressure.

What is to be said of the drug-treatment in acute pneumonia? Notwithstanding the present tendency to regard acute pneumonia as a self-limited disease, the author believes that drugs are not so useless as they are supposed to be by many writers, but on the other hand, that they are most invaluable aids in tempering the violence of the symptoms of this disease. And one of the best of these agents is strychnine. This drug, owing to its stimulating action on the nervous system in general, and the respiratory nerve supply in particular, is especially well adapted for use in this disease, as it is in fact in almost all affections of the pulmonary organs. Over and above this influence on the lungs it is the equal of digitalis in enhancing the function of the heart, and in this manner it tends to overcome some of the most serious tendencies to death in this disease. But in order to get the best action of strychnine it must be given for tangible effects, *i.e.*, in doses large enough to approach the line of its toxic action, and for this reason it is useless when given in small doses. In the adult it is best to begin with a dose of $^1/_{20}$ of a grain four times a day, and reinforce this with a hypodermic dose of $^1/_{20}$ of a grain morning and evening. On the second or third day, after its use in this manner, or perhaps later, symptoms of twitching or restlessness may develop, when its hypodermic administration may be abandoned. The latter amount may be increased, and very liberally, too, if the case is one of alcoholic or latent pneumonia. Another valuable drug is digitalis. With very large doses, such as are prescribed by some authorities, the author has had no experience, but frequently gives a dessertspoonful of the infusion every four hours, with a view of obtaining its tonic influence on the heart-

muscle. Capsicum is also of the greatest utility. It is one of our most effective diffusible stimulants, and it is of special usefulness in that stage of pneumonia which is accompanied by a low muttering delirium, comatose tendency, picking at the bedclothes, etc., and which is very often associated with a dry, black, crusty tongue. It is to be given in doses of from ten drops to a teaspoonful of the tincture in water every three or four hours. The author has given a teaspoonful of the tincture of red pepper every hour with the best results in low alcoholic pneumonia. The addition of from five to ten drops of the tincture of the chloride of iron to each dose of capsicum, will improve its efficacy in the very adynamic form of acute pneumonia. Quinine should be given in doses of five or ten grains every four hours until some of its constitutional effects are produced. When a malarial element is present, as is frequently the case in low, damp climates, it must be administered in doses from twenty to thirty grains every four hours. Morphine given hypodermically in quarter-grain doses at the outset of the disease, and at night, will add comfort and secure sleep to the patient. Sleep is very important in this disease, and a ten-grain suppository of asafœtida at bedtime will frequently produce a quieting effect. Oxygen given by inhalation is of immense service in cases of great dyspnœa and cyanosis. It is, of course, only a temporary measure, but it often assists greatly in bridging over the most critical period of the disease. If the dyspnœa is pronounced it must be given more or less constantly. In pleuro-pneumonia or in grip-pneumonia, or when an ordinary pneumonia is complicated with painful joints, or if pneumonia occurs in persons with either a personal or a strong family history of rheumatism, the salicylates of cinchonidia and soda, each in five-grain doses every three or four hours, with a teaspoonful of liquor ammonia acetate, is a useful combination. In case there is constipation, especially in the beginning of

the disease, calomel and bicarbonate of soda should be administered every hour in small doses until the bowels are relieved.

The question of food is also of much concern in the management of this disease. The food should be of the most nourishing character, concentrated in bulk, and easy of digestion. Such food we find in freshly expressed beef juice, of which two ounces, properly seasoned, should be given alternately every two hours, with a glass of milk containing a teaspoonful or a tablespoonful of whiskey or brandy. If the stomach is rebellious, as is very often the case in the beginning of the attack, the beef juice and milk may be given by the rectum.

Comparison of Results between Pneumonics Who Were, and Those Who Were Not, Treated with Cold Applications to the Chest.

Whatever opinions we may hold in regard to the value of any treatment, it is quite obvious that in the long run the verdict will favor that one which shows the smallest mortality rate. It is often said that nothing is more fallacious than statistics when applied to medicine, yet nowhere are figures more often resorted to, or serve a more useful purpose than in the determination of therapeutic results. As a rule, a drug is prescribed either consciously or unconsciously, because it has given a satisfactory response in a number of similar affections. In the light of the principle of numbers let us therefore inquire into the efficacy of local cold applications in the treatment of acute pneumonia.

Dr. Osler reports that out of 1012 cases of pneumonia treated in the Montreal General Hospital, 20 per cent. died, and that in the Charity Hospital of New Orleans the mortality rate was 20.01 per cent.; of 1000 cases of pneumonia treated in the Montreal General Hospital 20 per cent. died,

and that in the Charity Hospital of New Orleans the mortality rate was 20.01 per cent.; of 1000 cases of pneumonia treated in the Massachusetts General Hospital from 1822 to 1889, the death-rate was 25 per cent. Dr. Hartshorne states that the mortality rate from this disease in the Pennsylvania Hospital during the years 1884, 1885, and 1886 was a little more than 31 per cent. M. Louis treated 107 cases antiphlogistically, of which 32, or about 30 per cent., died. He found that in those who were bled during the first four days the death-rate was less than half of what it was in those who were bled during the first nine days of the disease. M. Grisolle, who used the lancet more moderately, had a mortality of 16 per cent. in 232 uncomplicated cases. In consonance with the statement of M. Louis, this writer declares that when bleeding was performed early the mortality was one in ten, while if it was performed late it was one in about six. Professor Rasori treated 648 cases of pneumonia with large doses of tartrate of antimony, of which about 22 per cent. died. With the same medicine, Grisolle lost 18.8 per cent. of 154 cases; and Dietl 20.7 per cent. of 106 cases. During a period of sixteen years previous to 1861 Dr. Huss treated 2616 cases with a death-rate of 10.74 per cent. During the first eight of these years the antiphlogistic treatment was carried out with considerable activity, while during the last eight only local bleeding was employed. The results were practically the same. Of 129 cases treated on the restorative plan by Dr. Hughes Bennett of Edinburgh, only four died, giving a mortality of 3.1 per cent.

Now, what conclusions are to be drawn from a death-rate of pneumonia which varies all the way from thirty to three per cent.? Are we to believe that nearly all pneumonics recover under certain methods of treatment, and that a large proportion of them die under other forms of medication? No one would be justified in making such a reck-

less deduction, yet it must be admitted that the truth lies somewhere between these two extremes; and in order to get at least a proximate solution of this problem, it is important to find the natural recovery-rate from this disease. What proportion of cases get well, and what proportion die under no treatment whatever? After having ascertained this point we will be in a better position to estimate the worth or worthlessness of any treatment. Data supplying this information have been furnished by Dr. Dietl, a Viennese physician, who in 1844, '45, and '46 treated 189 cases of pneumonia practically without medicine, and with a death-rate of 7.4 per cent. From 1847 to 1850 he treated a second series of 750 cases, with a death-rate of 9.2 per cent. Of Dr. Dietl's cases, 319 were complicated. That this probably represents the rate of natural recovery from pneumonia very nearly is confirmed by the results of pure homeopathic practice, the effects of which may be regarded as equivalent to the expectant or let-alone treatment. Thus, out of 94 cases of pneumonia treated in the homeopathic section of Leopoldstadt Hospital in Vienna, in six years, 9.57 per cent. died; and out of 24 case of pneumonia treated in the same institution by Drs. Wurmb and Caspar, in 1850, 12.55 per cent. died. Now if these figures are reliable, and there is no evidence that they are not, and allowing for the influence of food, nursing, and care which these patients received while in the various hospitals, it appears that from 85 to 90 per cent. of all pneumonia cases recover without any medical treatment.*

About eight years ago the author began making a collective investigation of the local influence of cold in the treatment of acute pneumonia among the members of the medical

* For the above quoted statistics in reference to the treatment of pneumonia in foreign hospitals, the author is entirely indebted to the interesting work by Dr. James Rogers, "On the Present State of Therapeutics," published by Churchill and Son, London, 1870.

profession, and succeeded in gathering four hundred cases. This collection includes cases of all ages, ranging from three weeks to eighty years; and many that were complicated with alcoholic, malarial, rheumatic, and cardiac conditions. Out of the total number there were 17 deaths, or a death-rate of 4.25 per cent. Now, when we compare these results with those which have been obtained from other forms of treatment it is seen that they make a very favorable showing. It is true that they are not quite so good as those of Dr. Bennett, but in saying this it must not be overlooked that, so far as the author knows, Dr. Bennett's results have not been duplicated by any one else on the lines laid down by him; while on the other hand this does not hold good of the cases in the author's collection. Here the results have not been secured by a single individual, but at least by forty-two different observers, among whom are at least two who have seen a score, and eleven others who have seen half a score of cases in succession without a single death. In itself this speaks volumes in favor of the cold applications, for it shows that the personal equation of the practitioner does not enter very largely into the success of the treatment.

CHAPTER XXIII.

ACUTE BRONCHITIS.

DEFINITION. An acute catarrhal affection of the mucous surface of the bronchial tubes.

SYNONYMS. Bronchial catarrh; Catarrh of the chest; Bronchialentzündung (German), Bronchite (French).

CAUSES. These depend on the individual conditions and on the external surroundings of the patient, and will be considered in the following order:

Age. No age-period is exempt from bronchitis, although infancy, in which there is a great susceptibility to acute pulmonary diseases, is exceedingly liable to it. Old age is also excessively prone to it.

Social State. The ill-fed, poorly-clothed, and badly-sheltered are more liable to bronchitis than those who are well-to-do, and who are provided with the essentials of civilized living.

Season and Climate. Bronchitis appears most often in winter, and in the autumn and spring—the periods of the year when extreme variations of temperature prevail; although a sudden change in the weather from warm to cold at any time of the year is conducive to an attack, especially if there is present any diathetic influence, like gout, rheumatism, malaria, etc.; so too, the inhabitants of a damp climate, especially when this is associated with a low and variable temperature, are very liable to this disease.

Occupation and Habit. Any local irritation of the bronchial tubes such as is brought about by the inhalation of air laden with foreign particles of matter will lead to bronchitis,

hence the liability to it of those who are engaged in dusty employments like spinning and weaving wool and cotton, working in iron and metal, cutting stone, milling, grinding scissors, etc. Any habit or excess which undermines the general health, and particularly that of the nervous system, like alcoholism, venery, and other sexual abuses, etc., will pave the way for the advent of the disease, and, indeed, may act as its exciting causes.

Intoxication. Many acute diseases like smallpox, scarlatina, measles, diphtheria, whooping-cough, typhoid fever, influenza, malaria, rheumatism, gout, etc., are frequently followed by bronchitis—the latter being in all probability due to the power which the toxins peculiar to these diseases have in setting up disordered action in the pulmonary organs.

PATHOLOGY. The morbid changes of bronchitis are confined to the structure and function of the bronchial mucous membrane. The normal histology of this membrane has already been given (p. 252), where it was shown to consist of an epithelial layer, a basement membrane, an inner and an outer fibrous coat, between the latter two being situated the muscular layer. Now the earliest morbid changes in this disease take place in the capillaries of the inner fibrous coat which become turgid and project into and diminish the caliber of the bronchial tubes. In a few hours the basement membrane becomes œdematous and swells, and in the course of twenty-four hours the epithelial layer begins to desquamate, and within three or four days is entirely thrown off. This is well illustrated in the following figures which are taken from the work of Dr. D. J. Hamilton, which has already been largely quoted, and which will be taken as a guide in everything that pertains to the pathology of bronchitis in both its acute and chronic form.

On account of the œdema which is present in the early stage of bronchitis, the expectoration is mostly of a serous

character, but after the mucous membrane has lost its epithelium the embryonic cells of the flat layer of Debove begin to proliferate so rapidly and abundantly that they furnish the chief supply of the yellow catarrhal excretion in the later stages of this disease. (See Fig. 42.) The rapidity with which these form, does not allow sufficient time for their development into epithelial cells, as is the case in health, and so they revert back to their original type, *viz.:* pus cells. This is shown in figure 43.

In the course of three or four days the columnar epi-

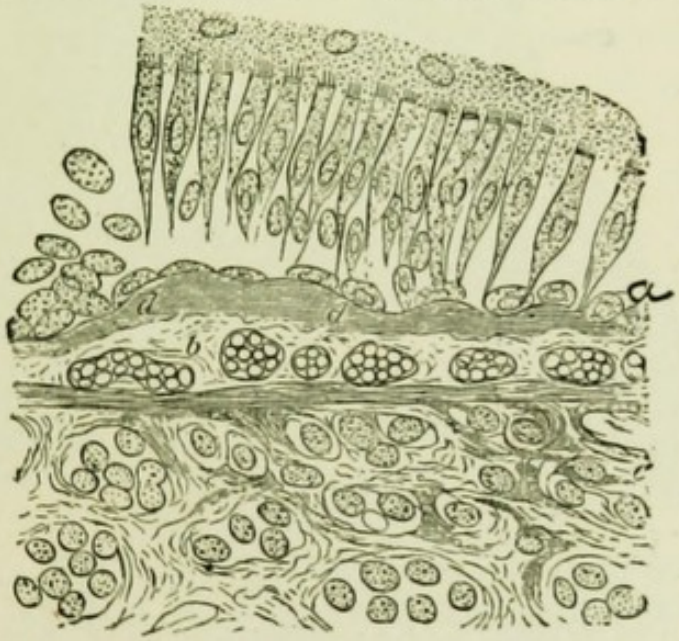

Fig. 42.—Transverse section of small bronchus. Commencing acute bronchitis. x 480 Diams. *a*, flat epithelial layer seen on section; *b*, inner fibrous coat; *c*, outer fibrous coat; *d*, basement membrane becoming œdematous.—*Hamilton.*

thelium is swept away entirely, the bronchial wall is red and engorged with blood, and its surface is uneven and rough; the inner and outer fibrous coats are infiltrated with leucocytes and large nucleated cells; and the spaces between the muscular fibers are also filled with leucocytes although the fibers themselves have not undergone any morbid changes. The mucous glands of the bronchial tubes are also implicated in the catarrhal process. They are distended with mu-

cus and the epithelial cells lining the duct desquamate and undergo fatty degeneration. The lymphatic glands at the root of the lung are always deeply congested and enlarged. Hemorrhage is frequently found in the outer fibrous coat and in the basement membrane. Sometimes these hemorrhagic spots are large and their contents break into the bronchus. According to Hamilton the nerve ganglia which are abundant around the large bronchi at the root of the

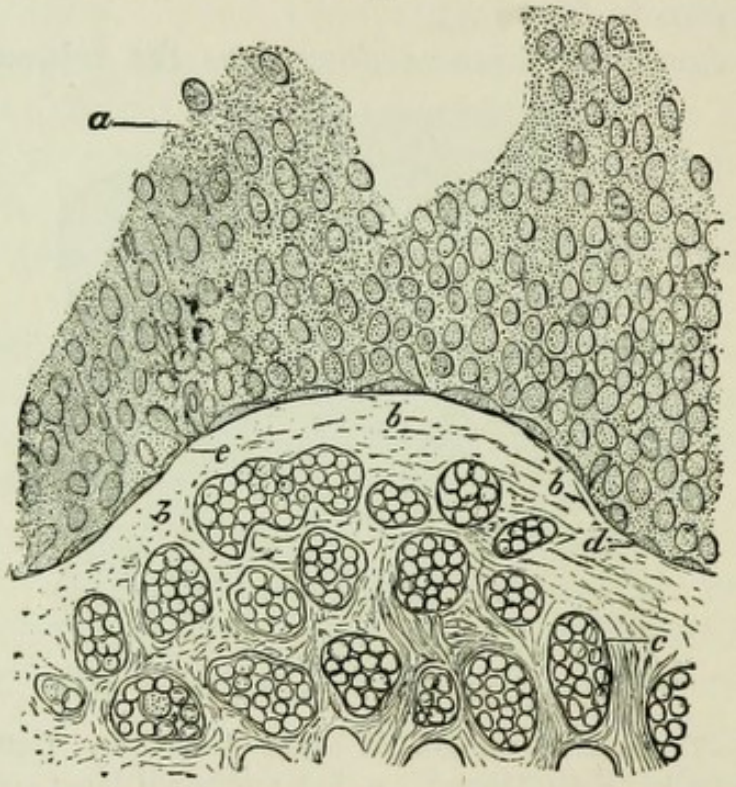

Fig. 43.—Transverse section of part of mucous membrane. Acute bronchitis x 450 Diams. *a*, Catarrhal secretion; *b*, swollen basement membrane; *c*, congested blood-vessels; *d*, flat epithelial cell layer seen on section ; *e*, flat epithelial cell layer germinating. —*Hamilton.*

lungs, as well as their nerve fibers, are very much involved in this disease. "The most intense congestion of the surrounding vessels of the ganglia is always noticed, and the minute capillaries which run into the ganglia and nerve trunks are all engorged with blood."

PLEURA. The pleural membrane is very rarely involved

in acute bronchitis. The right side of the heart becomes distended in the severer forms of this disease owing to an obstructed passage of blood through the lungs.

Symptoms. In its milder form bronchitis begins with a feeling of depression, lassitude, coryza, hoarseness, cough, expectoration, and slight fever, and passes off in the course of three or four days. Its severer attacks are ushered in by a chill, not as decided and as violent as that which introduces an attack of pneumonia, but a series of chilly sensations creeping over the whole body, especially if the latter comes in contact with cold bedclothes, or with currents of cold air; at the same time there is a rise of temperature which rarely exceeds 101° F. in the adult, and usually remains at or below 100° F. Dyspnœa is one of the earliest symptoms, and is generally accompanied with a tightness and oppression of the chest, especially in the region of the diaphragm. In the milder cases the respiration-frequency does not go above 30 or 40, but in the severer cases, and in children, it may exceed 50 or 60 per minute. Under the latter circumstances the face is pallid, while the lips, nose, ears, and finger and toe-nails are of a livid hue. The breathing is now intensely laborious, and the diaphragmatic distress is very pronounced. Throbbing of the neck and swelling of the veins and head, with general cyanosis, become prominent factors, and œdema of the lower extremities generally shows itself at this stage of the disease. The pulse is often frequent, but rarely rises above a hundred, or one hundred and twenty, in a minute, unless the case occurs in a child or is a very aggravated one in an adult. In the beginning the cough is harsh, dry, tickling, and worrying, especially in the evening before the patient falls asleep, and is associated with pain and soreness under the sternum; but after the second or third day these concomitant symptoms subside, and the cough becomes easy and in general parlance "loose." The expectoration, which is very sparse in the

early stage of the disease, becomes more abundant and puriform on the second or third day, and assumes a yellow color. Occasionally it is tinged with bright red blood. In its early stage bronchitis is associated with marked fatigue, coryza, hoarseness, muscular soreness, and aching, especially in the lower extremities. These disturbances generally abate with the subsidence of the fever. The skin is dry and hot, and the mucous membrane of the mouth, throat, and nose is parched. These symptoms also improve with the disappearance of the greater activity of the disease. The urine is scanty, highly colored, and occasionally albuminous. The function of digestion is deranged. There is thirst, loss of appetite and coated tongue. Frequently there develops catarrh of the intestinal tract, and diarrhœa supervenes. Vomiting is also of common occurrence. Headache, sleeplessness, and restlessness are always present, while delirium, convulsions and coma may occur in severe and aggravated forms of the disease.

Physical Signs. In acute bronchitis the lungs may be distended and enlarged, and the movements of the thorax are always accelerated, and the abdominal are in excess of the costal movements, yet the disease is bilateral; and hence the form, size, and motion of the chest are equal and symmetrical throughout its course. The heart, stomach, and liver may be displaced downward by the pressure of the enlarged lung from above, and in aggravated cases the right ventricle may be distended to such a degree that the heart assumes a horizontal position in the thorax, and its apex-beat may be found outside of the nipple line. In mild cases the percussion note does not vary from the normal. In severe cases the percussion note is wooden and hyper-resonant, and partakes somewhat of a tympanitic character which is due to the distention, and emphysematous condition of the air-cells. There is no dulness in uncomplicated bronchitis.

AUSCULTATION. Owing to the dryness, swelling, and narrowing of the bronchial mucous membrane in the early stage of bronchitis, the breathing sounds are rough and harsh during this period. In the course of twenty-four or thirty-six hours the secretion of mucus and muco-pus appears, which gives rise to moist râles. These râles when produced in the large tubes are large in volume, are called large mucous râles, and when produced in the bronchioles are of small size, and are called subcrepitant râles. Mixed with these are the musical, or so-called dry râles. These are named sonorous and sibilant râles—the former being produced in the larger and the latter in the smaller tubes. All these râles may be heard during inspiration and expiration, and may always be displaced or at least modified by the act of coughing, and so be distinguished from the gurgling râles of small cavities and of dilated bronchial tubes, which are also audible during inspiration and expiration. The roughness and unevenness of the respiratory sounds are heard more distinctly after the râles have disappeared, and remain until full convalescence is established.

DIFFERENTIAL DIAGNOSIS. Bronchitis may be mistaken for emphysema, pneumonia, and acute phthisis, and the following tabular view in which the salient differential points between these diseases are indicated by numbers, will serve as a means of discriminating between them. Asthma and bronchitis may also be confounded one for the other, but a careful inquiry into the family and personal history of the patient will help to overcome this difficulty. Bronchitis is the inevitable product of asthma and, therefore, asthma cannot exist without some degree of bronchitis.

Acute Bronchitis.

1. No well-defined chill at onset.
2. Hoarseness, at the beginning usually.
3. Cough, severe from the first.
4. Expectoration, copious and yellow.

5. Hemoptysis, rarely.

6. Pulse, not very frequent—100 to 120, except in children it is more frequent.

7. Respiration, frequent.

8. Fever, moderate, 100° to 101° F., except in children it is higher.

9. Emaciation, not marked.

10. Delirium, exceptional in adults.

11. Both sides affected.

12. Uniform motion on both sides.

13. Upper part of lungs prominent.

14. Vocal resonance and fremitus symmetrical.

15. No impaired percussion resonance.

16. Moist and musical (dry) râles during inspiration and expiration.

17. Râles movable by coughing.

18. No tubercle bacilli present.

Emphysema.

1. No initial chill.

2. No hoarseness.

3. Cough, moderate.

4. Expectoration, copious when associated with bronchitis.

5. Hemoptysis, very rare.

6. Pulse, small and weak.

7. Dyspnœa, marked.

8. Fever, absent.

9. No emaciation.

10. No delirium.

11. Both sides affected.

12. Movements uniform on both sides.

13. Prominence and roundness of shoulders, stooped.

14. Vocal resonance and fremitus symmetrical.

15. Percussion sound hyper-resonant.

16. No râles. Prolonged vesicular murmur; also, prolonged expiratory sound.

17. ———.

18. No tubercle bacilli present.

Acute Pneumonia.

1. Decided initial chill.

2. No hoarseness.

3. Cough, harsh, not marked in most cases.

4. Expectoration, not copious, rusty, prune-juice color, may be bloody.

5. Hemoptysis, marked sometimes.

6. Pulse full and slow.

7. Respiration, frequent.

8. Fever, high from onset.

9. Emaciation, comparatively marked.

10. Delirium a prominent feature.

11. Unilateral.

12. Impaired movement on affected side.

13. ———.

14. Vocal resonance and fremitus increased over affected area.

15. Dulness.

16. Crepitant râles, prolonged expiration, bronchial breathing.

17. Râles, not moved by cough.

18. No tubercle bacilli present.

Acute Pulmonary Tuberculosis.

1. No definite initial chill, severe and irregular chills throughout its course.

2. No hoarseness as a rule.

3. Cough, usually constant.

4. Expectoration profuse, puriform, nummular, blood-streaked.

5. Hemoptysis, usually present.

6. Pulse rapid and weak.

7. Dyspnœa.
8. Fever high, intermittent, irregular. Sweats profuse.
9. Emaciation, rapid and pronounced.
10. Delirium may be present.
11. Bilateral, usual.
12. No difference in chest movement.
13. ———.
14. Vocal resonance and fremitus uncertain.
15. Dulness may be entirely absent.
16. Large moist râles. Roughened breathing sounds.
17. Râles, not moved by cough.
18. Tubercle bacilli present.

The diagnosis of acute bronchitis from diseases which resemble it is comparatively easy. Its bilateral location, its copious yellow expectoration, its moderate fever, its normal percussion resonance and its movable mucous râles distinguish it at once from acute pneumonia with its decided initial chill, its high fever from the beginning, its rusty or prune-juice expectoration, its slow pulse, its one-sided location, its immovable crepitant râles, and its prolonged expiration. There is greater difficulty in separating it from acute phthisis or tuberculosis, which has, in common with bronchitis, a profuse puriform expectoration, quickened respiration, a bilateral position, an absence of decided dulness, large moist râles, and roughened breathing, but differs from it in having a nummular expectoration, a weak and rapid pulse, a high intermittent and irregular fever, rapid and pronounced emaciation, and the stationary character of its large râles.

Course and Termination. Uncomplicated bronchitis in the adult is not a serious disease, and usually terminates in health in the course of a week or ten days. When it occurs in those whose vitality is greatly reduced either on account of constitutional causes, or of previous illness, it may terminate in the chronic form of the disease. When the inva-

sion is extensive or when it takes place as a complication of influenza, of albuminuria, or of cardiac degeneration, it becomes a grave disorder. Among the symptoms which indicate its gravity are cyanosis, pulsation of the veins in the neck, pain in the base of the chest, coldness of the ears, nose, knee-caps, and extremities, accelerated respiration, dyspnœa, frequency and irregularity of the pulse, cold sweats, delirium and coma.

TREATMENT. Simple and continuous rest in bed for three or four days, or until the fever subsides, combined with the local application to the chest of mustard or of some other counterirritant, the administration of calomel and soda to move the bowels and a stimulating expectorant composed as follows: ℞. Tinct, Benz. Comp. Ext. Euphorb. pil. Fld. aa fl℥ss, Tinct. Nucis vomicæ fl℥ii, Ammonii Chloridi gr. L, Syr. Scillæ, Syr. Tolu. aa℥. Aquæ, q. s. fl℥iv. M. Sig. One teaspoonful every three hours. This will in many cases suffice to give relief.

Cases in which the disease is very active, and in which there are marked fever, headache, restlessness, and nervousness there should be no hesitation whatever in applying one or two ice-bags to each side of the chest, and one or two bags to the head at the same time, which should be retained until the temperature falls permanently. The above-mentioned mixture, with the tincture of nux vomica omitted, may be given, alternated every two hours with the following: Strychninæ sulphatis, gr. 1¼, quininæ sulphatis, acetanilidi; sodæ salicylatis aa ℥i; pulv. digitalis, pulv. capsici, aa gr. viii. M. Ft. capsulas No. xxxii. Sig. One capsule every four hours. To secure sleep, a quarter of a grain of morphine sulphate, with a $^1/_{20}$ of a grain of atropine sulphate are to be given subcutaneously at night, or night and morning. If there is much difficulty of respiration a drop or two of a one-per-cent. solution of nitroglycerine every four hours will be of service. In patients who are addicted to the

excessive use of alcohol, or in others whose low vitality is shown by a muttering delirium, or by picking at the bed-clothing, the administration of a $^1/_{20}$ of a grain of strychnine two or three times a day subcutaneously, together with half-dram doses of tincture of capsicum every hour, is very advantageous. When cyanosis develops, oxygen must be given continuously by inhalation. In acute bronchitis accompanying influenza, salicylate of cinchonidia in five grain doses every four hours will be of great service. The diet must be rigorously nourishing. Two ounces of freshly expressed beef juice every four hours, a glass of milk every three hours will suffice to maintain the patient through the active stage of the disease. After the critical period has subsided, more solid food like soft-boiled eggs and bread, oysters, and scraped beef may be given. So soon as convalescence is assured the above medication may be advantageously substituted by the following, provided the doses of strychnine above advocated were given: ℞ Strychninæ Sulphatis, gr. iss; Syr. Acidi Hydriodici f℥i; Syr Hypophosph., f℥iii. M. Sig. One teaspoonful four times a day. Three grains of quinine sulphate, twice a day in capsule will reinforce the tonic action of the last mixture.

In the treatment of acute bronchitis in children the same general course which is outlined above is to be followed in a properly adjusted manner. As a counterirritant in very young infants, equal parts of amber oil and sweet oil are to be applied to the whole chest, and then covered with a layer of cotton wadding. In children of a year's growth the amber oil is to be used alone. In still older children a mixture consisting of croton oil ℥i and olive oil ℥xv is a very useful counterirritant. In all cases the counterirritants should be rubbed in once or twice a day.

CHAPTER XXIV.

CHRONIC BRONCHITIS.

DEFINITION. A chronic catarrhal inflammation of the mucous membrane of the bronchial tubes involving the deeper layers of this structure.

SYNONYMS. Chronic bronchial catarrh; Chronische brunchialentzündung (German), Bronchite Chronique (French).

CAUSES. Among these the following are the most prominent:

Age. Chronic bronchitis occurs at all ages, but is most common at the extremes of life. In childhood it develops out of an acute attack, or follows measles, whooping cough, diphtheria, etc., and in older persons it accompanies many of the diseases which are incidental to this period of life.

Sex. If there is any difference in the liability of the two sexes it is in favor of women.

Social State. It prevails most largely among the poorer classes of society.

Climate. This disease is most common in climates subject to sudden and decided changes of temperature, or in humid climates with a low temperature.

Season. It is most prevalent in the colder months of the year.

Occupation. Employments that necessitate the inhalation of irritating dust, such as that which arises from cotton, flour, stone, coal, charcoal, etc.; or entail exposure to sudden changes of temperature, like that of bakers, firemen, etc., favor the development of chronic bronchitis.

Complications. Diseases of the heart, especially of the mi-

tral and aortic valves, cancer and other chronic affections of the lungs, are strongly provocative of chronic bronchitis.

Intoxication. The poisons of alcohol, syphilis, rheumatism, gout, influenza, etc., are very prone to generate this disease.

Pathology.

The pathology of chronic bronchitis varies considerably and may be best considered under the following heads: (1) when caused by an acute attack of bronchitis; (2) when dependent on valvular lesions of the heart; and (3) when produced by the inhalation of foreign particles of matter.

1. *When Caused by an Acute Attack.* The bronchial tubes* contain a copious quantity of yellow catarrhal material. The mucous membrane of the lower part of the trachea and bronchial tubes is deeply congested, its surface is denuded of epithelium, and on its smooth and glistening basement membrane lie the embryonic epithelial cells, which constitute the flat layer of Debove's cells. The whole mucous membrane is much thickened and thrown into longitudinal folds, the lumen of the larger tubes is narrowed, while the smaller bronchi are in a state of dilatation, and the bronchial mucous glands are invariably enlarged, secrete a large amount of mucus, and sometimes form abscesses. Both the inner and outer fibrous coats suffer from marked cellular infiltration, and the cartilages and muscular layer weaken and become atrophied. The muscular coat of the small arteries is usually thickened, but the walls of the capillaries are thin and distended into large sinuses, the air-vesicles, as a result of the forcible coughing efforts incidental to chronic bronchitis, are overdistended, which leads to permanent emphysema. Fig. 44 describes the pathologic condition of the tube.

* For a description of the normal histology of the bronchi the reader will refer to p. 252.

It will be seen that the whole bronchial wall is thickened, thrown into folds, and is densely infiltrated. The basement membrane is easily made out, and on it are small bud-like projections, germinating epithelial cells. The air-vesicles around it are markedly emphysematous.

2. *When Dependent on Valvular Lesions of the Heart.* Valvular lesions of the heart always interfere with the free

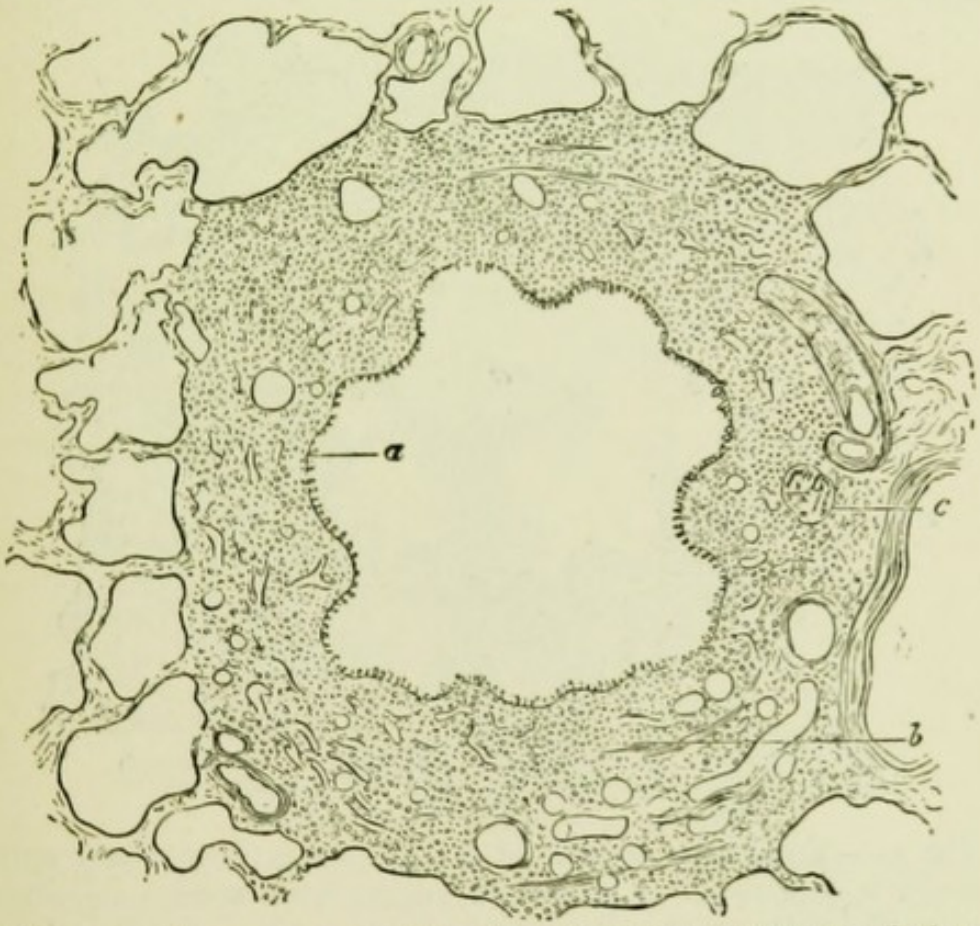

Fig. 44.—Transverse section of a bronchus in a state of chronic catarrh. x 50 Diams. (reduced one half) *a*, germinating epithelium, placed on basement membrane; *b*, remains of muscularis; *c*, a small nerve.—*Hamilton.*

circulation of the blood behind the lesion. Hence, in case of mitral regurgitation the blood-flow is interfered with in the pulmonary circulation, and this gives rise to chronic bronchitis and other pulmonary affections. Examination of a bronchial mucous membrane in such a condition shows it

to be intensely congested and of a deep cyanotic color, and the smaller sized tubes contain a frothy serous liquid. The lung substance is also much congested, and throughout it are noticed hemorrhagic patches. The lumen of the tube is usually narrowed, and its mucous membrane is thrown into folds and thickened, not, however, on account of cellular infiltration, as is the case with the previous form of

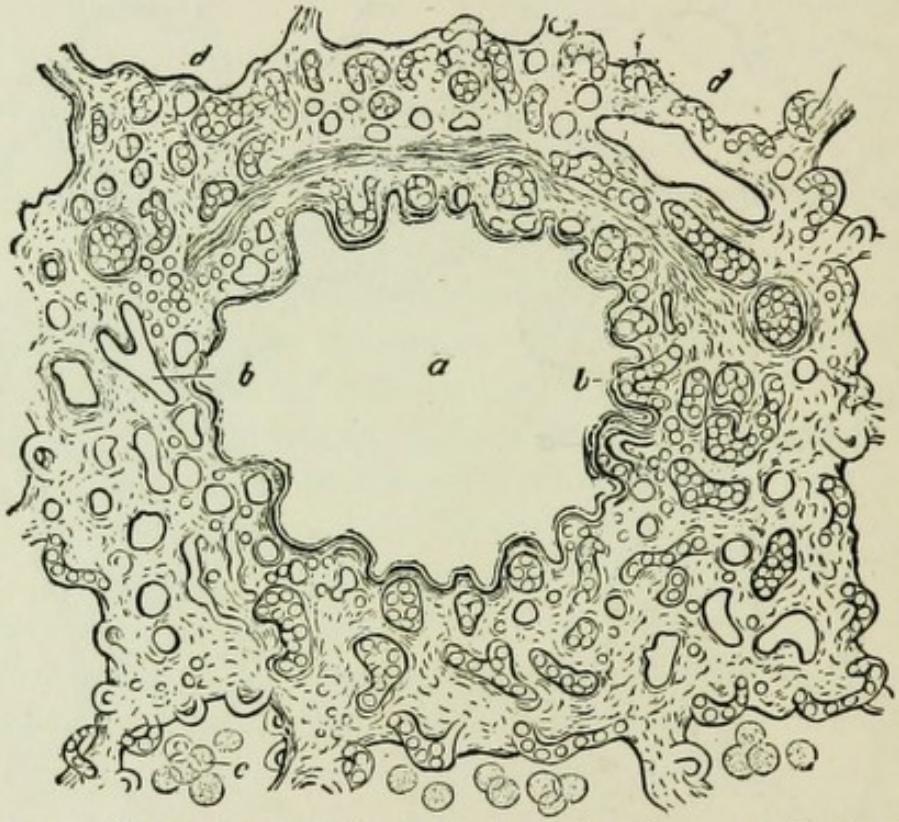

Fig. 45.—Transverse section of a bronchus in regurgitant mitral disease. x 300 Diams. *a*, bronchial lumen, with mucous membrane thrown into folds; *b*, congested capillaries; *c*, desquamated and pigmented alveolar epithelium; *d*, alveolar cavities of lung.—*Hamilton.*

bronchitis, but because its blood-vessels are distended and turgid. Every texture seems to be in an œdematous condition. As a rule, the epithelium is wanting, and on the free surface of the basement membrane, which is swollen and œdematous, are numbers of bud-like growths which seem to take the place of epithelial cells. Every minute blood-vessel is so distended with blood that rupture of their walls

and hemorrhages are of frequent occurrence. Figure 45 gives a good illustration of this pathologic product.

3. *When Produced by Inhalation of Foreign Particles of Matter.* This form of chronic bronchitis is extremely common among coal miners, and is brought about through the inhalation of particles of carbon. The mucous membrane of this affection is thickened, the epithelium has dis-

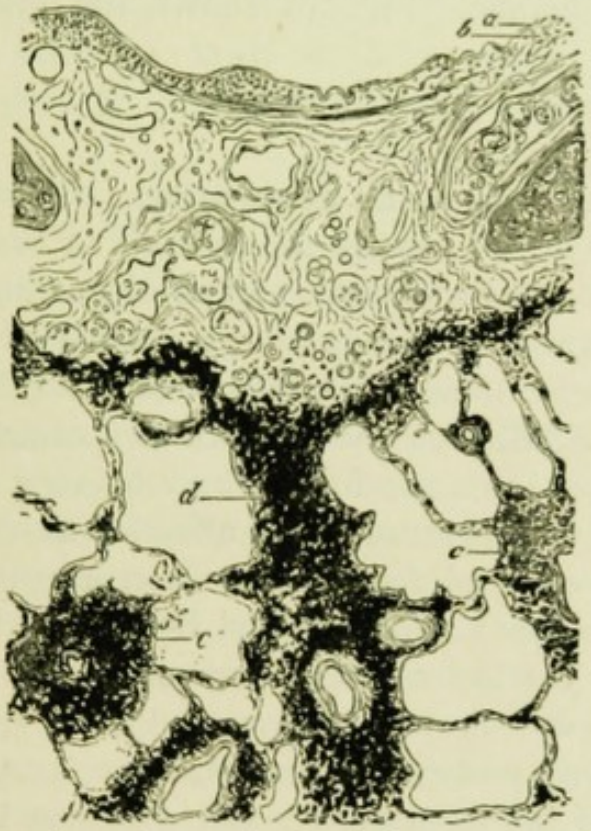

Fig. 46.—Bronchus of a coal-miner's lung. x 50 diams. *a*, Basement membrane; *b*, inner fibrous coat; *c*, pigmented nodules surrounding the arteries; *d*, pigmented lobular septum.—*Hamilton.*

appeared, the tubes contain a yellow non-purulent fluid, and the substance of the lungs is of a black color. The carbon particles do not penetrate the lungs through the bronchial mucous membrane, but gain entrance through the infundibula, air-vesicles, and bronchioles, whence they are carried by the perivascular and peribronchial lymphatics, into the lobular septa, around the arteries, into the deep layer of the pleura, and finally land in the bronchial glands. The irritation which

is set up by these particles leads to thickening of the fibrous interlobular tissue, and compression of the pulmonary blood-vessels, as is seen at *c*, Fig. 46. This serious interference with the blood-supply in connection with the clogged condition of the lymphatic vessels, produces a state of œdema and thickening in the fibrous coat of the bronchi, which finally leads to an accelerated and imperfect production of bronchial epithelium. Fig. 46 shows the bronchus of a miner's lung.

Symptoms.

Unless ushered in by an acute attack, chronic bronchitis develops slowly. It usually shows itself at first during the changeable months of the year, and subsides during the summer months until, in the course of a number of years, it becomes permanent. The cough may be dry and the expectoration scant if it takes on the form of dry catarrh ("catarrh sec" of Laennec); or the former may be very troublesome, and the latter very profuse, if the affection assumes a humid nature. The color of the expectoration is yellowish-white, or grayish, and may contain blood if the cough is very severe. When cardiac complications exist the color of the rather scant expectoration is gray and watery, and may be bloody. Indeed profuse hemoptysis may be anticipated in this variety of the disease. At first dyspnœa is inconsiderable, but becomes very pronounced when the mucous membrane is thickened and interferes with the usual circulation of air throughout the lungs. In cardiac bronchitis the dyspnœa is constant, and specially marked during exercise. Cyanosis appears in various degrees and localities when the pulmonary circulation is stinted with oxygen, as is the case when the bronchial tubes are narrowed, or impeded with an abundant accumulation of catarrhal material, or when cardiac complications are present. Fever, as a rule, is absent, but the temperature may rise slightly, especially in compli-

cated cases. Emphysema generally accompanies the latter stage of the disease. The constitution remains unimpaired in the milder attacks, but may become gravely affected when cough excites vomiting, and interferes with sleep, and is associated with profuse expectoration. In some persons chronic bronchitis may be the active manifestation of the rheumatic or gouty diathesis. The latter may show itself simultaneously, or alternately with the former, in articular pain of the shoulder, knee, wrist or finger, in gastralgia, lumbago or sciatica, or in psoriasis or eczema.

Physical Signs.

In recent cases the chest movements are symmetrical, but in cases of long standing its circumference becomes more circular, and the shoulders stooped and round on account of the emphysema which accompanies it. There is no change in the vocal fremitus. The percussion pitch is not altered except in old cases where it partakes the character of a high tympanitic, ringing pitch. The vesicular sounds are feeble, the breathing in the bronchi is roughened, expiration* is very much longer than inspiration, on account of the impaired elastic power of the air cells; mucous râles of different dimensions are heard if the disease is humid, and sonorous and sibilant râles are present when it is dry.

Diagnosis.

About the only disease with which chronic bronchitis may be confounded is pulmonary phthisis, especially when bronchiectasy exists in the former affection. As a rule, the finding of the tubercle bacillus determines the nature of the trouble, although this organism may also be present in bronchiectasy.

* This must not be mistaken for so-called prolonged expiration.

Course and Termination.

Chronic bronchitis is essentially a catarrhal affection of the bronchial mucous membrane, which, on account of the excessive coughing that accompanies it, causes dilatation of the bronchial tubes and of the air-vesicles, leading, on the one hand to bronchiectasy and on the other to emphysema. The danger of bronchiectasy as a source of tuberculosis has been dwelt on already, on page 276, and while this risk is not so *pronounced in bronchitis* as it is in phthisis, its importance as a factor in producing further pulmonary disturbance must not be overlooked. Emphysema terminates in pulmonary collapse, and in hypertrophy of the right side of the heart and in general dropsy. On the whole, however, the greatest danger of chronic bronchitis is an extension of the catarrh of the tubes into the alveoli and develop a chronic catarrhal phthisis.

Treatment.

The treatment of chronic bronchitis resolves itself (1) into local measures applied to the chest-surface; (2) into those which increase the vital resistance of the pulmonary and of the general nerve-supply; (3) into those which relieve special indications; and (4) into those which nourish the patient.

1. Local Measures. The application of mustard to the chest morning and evening, or of tincture of iodine once daily, or of hot flaxseed meal poultices from morning until evening, or of croton oil, either pure or with equal parts of olive oil, every other day, exert a beneficial influence on the progress of the local condition. The constant wearing of an ori-nasal respirator, carrying on its sponge a medium-strong solution of carbolic acid, ichthyol, or thymol is a useful procedure.

2. Measures Which Enhance the Vital Resistance of the Pulmonary and General Nerve Supply. Among

these strychnine unquestionably comes first, and should be given in the manner prescribed on page 335. The author has witnessed some very striking amelioration in chronic bronchitis when the drug was employed in the manner indicated, especially when given with the syrups of the hypophosphites and hydriodic acid, half a dracm of each to a dose. When marked dyspnœa exists, the addition of one or two drops of a one-per-cent. solution of nitro-glycerine and five drops of the tincture of strophanthus, every four hours, will increase the efficacy of the strychnine. The hypodermic injection of silver nitrate, according to the method recommended on page 349, has yielded some very excellent results in the hands of the author. Quinine, red pepper, and arsenic, may be given in combination with strychnine, with good effect, as follows:

℞ Strychninæ Sulphatis..... gr. i
Thermolis..... ʒ i
Quininæ Sulph..... ʒ iss
Pulv. Capsici..... gr. x
Acid Arsenici..... gr. ½.
M. Ft. Capsular No. xxxii.
Sig. One capsule four times a day,

The amount of strychnine is to be gradually increased in the above composition. The importance of cod-liver oil is not to be overlooked in the treatment of this disease. If the cough is very annoying, and the breathing is so short as to prevent the patient from lying down and going to sleep a quarter of a grain of morphine is to be administered, hypodermically, at bedtime, and continued as long as it is believed to be necessary to the patient's best interests. The following mixture may be given at the same time, but not in connection with the amount of strychnine in the above capsule:

℞ Ext. Euphorbiæ pil. Fld.
Tinct. Nucis Vomicæ
Tinct. Benz. Comp.
Tinct. Capsici } aa..... fl ʒ ss
Sol. Trinitrin (1 p. c.)..... gtt. xxxx
Syrup Tolu..... fl ʒ i
Syrup Sarsaparillæ Comp., q.s..... fl ʒ iv.
M. Sig. One teaspoonful every four hours in water.

3. Measures Which Relieve Special Indications. If the rheumatic or gouty diathesis is present, which is more often the case than is suspected, then the salicylates and the ammonia preparations are to be resorted to. The following combination may be given with benefit in such cases:

℞ Sodæ Salicylatis..... ʒ iss
Potassæ Iodidi..... ʒ ij
Liq. Ammon Acetatis.....fl ʒ i
Sol. Trinitrin. (1 p.c.).....gtt. L
Tinct. Strophanth.....fl ʒ ss
Elix. Lactopeptin } aa.....fl ʒ i
Infus. Gentian }
Syr. Sarsaparillæ, Comp., q.s.....fl ʒ iv.
M. Sig. One teaspoonful every four hours in water.

4. Food. Nutritious food of an appropriate character is of vast importance in the treatment of this disease. The gouty or rheumatic cases should eat meat sparingly, and should subsist chiefly on vegetables, fruit, oysters, fish and soups. A large amount of water should be taken. The various lithia waters serve a very useful purpose in such cases. Other cases should receive the most liberal nierogenous diet.

In order to select a suitable climate for cases of chronic bronchitis, the principles which have been laid down for the direction of the phthisical are to be applied in these instances.

CHAPTER XXV.

ASTHMA.

DEFINITION. Asthma, like epilepsy, is a convulsion. It consists essentially of a paroxysmal motor disturbance of the respiratory centers and of the pulmonary nerves, which causes spasm of the muscles of the bronchial tubes and catarrh of the mucous membrane of the same.

SYNONYMS. Spasmus bronchialis, Engbrüstigkeit (German), asthma nereux (French).

CAUSES. *Age*—Asthma is most liable to develop in the first decade of life—the period which marks the most rapid growth of the highest nerve-centers. Dr. Salter* collected 225 cases of this disease and found that it develops with the following relative frequency in the different age-periods:

During the first year		11
From	one to ten years	60
"	ten to twenty years	30
"	twenty to thirty years	39
"	thirty to forty years	44
"	forty to fifty years	24
"	fifty to sixty years	12
"	sixty to seventy years	4
"	seventy to eighty years	1
		225

From this table it is seen that 71, or 31 per cent. of these cases arose during the first decade of life.

Sex.—Males are more prone to asthma than females, particularly in the earlier periods of life. Thus in Dr. Salter's† collection of 207 caes there were 138 males and 69 females.

* "On Asthma: Its Pathology and Treatment," by Henry Hyde Salter, M.D., F.R.S.; first American edition, New York, 1882, p. 59.

† *Op. cit.*, p. 59.

Heredity. All observers practically agree that asthma is inherited, and evidence apparently points out, too, that, like in the case of pulmonary consumption, it has both a direct and an indirect inheritance. In reference to direct and indirect heredity it may be stated that among Dr. Salter's collection of 207 cases* there were 84, or 38 per cent., who had a distinct family history of the same disease; 45, or 27 per cent., with a phthisical history; and 21, or 10 per cent., with a gouty inheritance. Clouston† says that asthma is frequently found in children of neurotic families, that it often disappears when other nervous diseases appear; that an attack of epilepsy, hysteria, or chorea will often stop it, and that Pavel holds epilepsy, insanity (see p. 180), neuralgia, chorea and hysteria as being interchangeable with it in the same families.

Salter‡ relates an excellent example of alternation between epilepsy and asthma:

Case 248. M., aged about 50, had epileptic attacks about once in a fortnight. After one of his usual periods an attack of asthma came on in place of a paroxysm of epilepsy. The dyspnœa passed off and left him as well as usual. At the expiration of the next accustomed interval after the asthma the ordinary premonitory symptoms and the usual epileptic fit occurred. This was repeated on several occasions, the epileptic seizure being supplanted by the asthmatic attack.

The above evidence, therefore, tends to demonstrate that asthma may be transmitted by families that are entailed with strains of phthisis, gout, insanity, epilepsy and of other nervous disorders.

Climate. Asthma is a most capricious disease in regard to climate as a causative factor. As a rule a damp easterly wind is provocative of an attack, although it must be remem-

* *Op. cit.*, p. 60.

† "Neuroses of Development," p. 65.

‡ *Op. cit.*, p. 23.

bered that some asthmatics flourish in low and damp localities. A large number of asthmatics have greater immunity in a high than in a low locality. Sometimes neither elevation nor distance seems to have any influence. The author has known the severest attack to discontinue so soon as the patient was placed on a moving railroad train. An asthmatic is unable to sleep in the back of the house, but can do so with impunity in the front. Salter is of the opinion that, on the whole, country air and climate are more conducive to asthma than those of large and smoky cities, and the author has seen evidence that seems to confirm this view.

Odor, Dust, Etc. The respiratory mucous membrane in some asthmatics is so extremely sensitive to the action of certain kinds of animal and vegetable emanations that their exposure to the presence of dogs, rabbits, cats, horses, sheep, guinea-pigs, ipecacuanha and flower-dust, etc., incites an attack very readily. Indeed some asthmatics would be altogether free from their paroxysm if they could escape the pernicious influence of these agents.

Intoxication. Gouty, rheumatic and syphilitic intoxication of the nervous system very often predisposes to the development of asthma.

Peripheral Irritation. Causes of this kind are polypi and other affections of the nasal passages, dyspepsia, flatulence, overloaded rectum and uterine and ovarian disease.

PATHOLOGY. It is generally accepted that asthma is a neurosis of the pulmonary branches of the pneumogastric nerves. This view is fully confirmed by such pathologic evidence as that furnished by the case of Heberden, in which a bony outgrowth of the upper dorsal vertebra compressed one of the vagi; by that of Dr. Gardner,* in which a neuromatous tumor of the vagus was suspected before and found after death, and by that of Dr. Mercklen,† in which enlarged lymphatic glands compressed the left vagus.

* *Edinburgh Medical Journal*, 1850.

† *Deutsche medicinal Zeitung*, 1887.

Opinion seems to be divided on the question whether the intimate and essential pathology of asthma consists of a spasmodic contraction of the bronchial muscles or of a swelling or turgescence of the bronchial mucous membrane. In view of the facts, however, that physiologic research has demonstrated that the lumen of the bronchial tubes may be diminished and distended through the influence of the pneumogastric nerves; that during an asthmatic attack there is no dulness, but rather a hyper-resonant percussion sound; that very frequently an attack of asthma begins and ends suddenly; that the most severe form of bronchitis does not produce dyspnœa like that of asthma, while in asthmatics the slightest catarrh often leads to most violent dyspnœa, and that the nitrites and other drugs relieve most cases of genuine asthma by dilating unstriped muscular fiber, it seems clear that the element of muscular contraction is the chief factor in the genesis of this disease and that the bronchitis is, in many instances, merely a resultant of the nervous and muscular commotion. On the other hand, it is true that a bronchial catarrh may act as a focus for the lighting up of a fresh attack of asthma in persons who are predisposed to the latter disease.

Symptoms. The attack may be preceded by sneezing, nasal catarrh, headache, neuralgia, vertigo, diudesis, etc. Its invasion usually occurs during the small hours of the morning, even sometimes while the patient is asleep, and is marked by a sense of constriction and oppression of the chest. A short, dry, hacking and wheezy cough, a pronounced dyspnœa, an immobility of the chest, and a short, jerky inspiration, while expiration is much prolonged. During the attack the face is pale, the head is bathed in perspiration, the chest is bowed forwards, the shoulders are propped up by the elbows resting on some support, and every effort is concentrated by the patient towards the purpose of carrying on the utter misery of breathing. Yet as soon as the asthmatic grip

is loosened the patient, in an incredibly short time, is transformed into an apparently healthy and robust individual. A truce is then established until the next attack, when the patient is subjected to a repetition of the same procedure. In cases of long standing the attacks frequently lose their regularity, but become more severe and the interval between them shortens until the paroxysm is more or less a continual one.

There is usually very little if any expectoration during the attack. When it ceases the sputum is scant, and of a tough, mucous consistency, often partakes of the shape of small pellets, and in case there is present a considerable degree of bronchitis it is yellow in color and copious, and in severe cases it may contain blood. Octahedral crystals of varying sizes were discovered in it by Leyden.

There is absence of fever, the pulse is weak and slow, and may be intermittent, the veins of the head and neck are dilated, the urine is scant and high colored, and albuminuria is occasionally present.

Physical Signs. There is no dulness in asthma, but the percussion note is of a hyper-resonant, perhaps of a tympanitic, quality over both lungs. During the attack auscultation shows that the vesicular murmur is exceedingly feeble, or is veiled by loud, sibilant and sonorous râles, which are more pronounced during expiration than inspiration. When the attack ceases moist râles are added to those just mentioned.

Diagnosis. The dyspnœa of asthma bears a resemblance to that which is found in croup, œdema or spasm of the larynx, paralysis of the vocal cords, entrance of a foreign body into the bronchial tubes, cardiac dyspnœa, emphysema, pressure of enlarged glands on the bronchi and by the pressure of aneurismal or other mediastinal tumors on the vagi.

Croup is distingushed from asthma by the crowing respiration, by the hoarseness, presence of fever and by the age

of the patient, asthma being a very rare affection in early infancy. To differentiate between œdema and spasm of the larynx, and paralysis of the vocal cords from asthma, the local sensibility, the absence of sibilant and sonorous râles in the chest, the smoothness of the respiratory sounds in the lungs, and the laryngoscope must be depended on. In the dyspnœa which is due to the entrance of a foreign body into a bronchial tube there is a unilateral impairment of respiratory motion in the chest, an absence, or at least a partial suppression of the normal vesicular murmur in a limited lung area, and probably puerile breathing in the remainder of the respiratory surface. From cardiac dyspnœa it is differentiated by the breathing, which is retarded, weak and suppressed in asthma and is panting and exaggerated in cardiac dyspnœa; by the prolonged expiratory act, which is present in asthma and absent in cardiac dyspnœa; by the wheezing and sibilant râles in the former, and by the mucous râles and the frequent hemoptysis in the latter. Emphysema differs from it in the following points: Its dyspnœa is permanent, and is much aggravated by exertion. In it the chest is circular, the shoulders round, and the diaphragm low. Asthma, however, leads to emphysema. Pressure of goiter and of enlarged glands on the bronchi give rise to a localized, hissing, breathing sound in the bronchial tube. Evidence of tumefaction must also be looked for in the neck. The symptoms which are produced by the pressure of aneurism or tumors on the vagi differ from those of asthma in the following particulars: The breathing is difficult and persistent in the former, and is accompanied by a sharp, ringing, croupy cough; there is usually a perceptible impulse, a localized dulness, an increased transmission of the cardiac sounds over the involved area and there may be an inequality in the pulses and in the pupils.

Course and Termination. Asthma is essentially a chronic disease and may last throughout a long life. A par-

oxysm is seldom, if ever, fatal. One of the important questions is, do asthmatics die of this disease or of some secondary complication? The literature on this point is somewhat scant, but there is some evidence to show that asthma develops into phthisis. The author is able to trace four cases of this disease which moved in this direction. Two of these died of consumption and two had the physical signs of the latter disease, but became entirely well under treatment. The elder Williams* records four cases of asthma that passed into phthisis. Fuller† states that in spite of the belief that asthma and consumption are antagonistic to each other many asthmatics die of consumption. Other authors record a similar experience. It appears that the danger of asthma lapsing into consumption only begins when the attacks of the former follow each other in such close succession that the bronchial irritation produced by the preceding paroxysm is no longer allayed. It is, therefore, more or less an accumulation of the effects of asthma which leads to this danger.

TREATMENT. The treatment of asthma consists of measures (1) which give immediate relief to an attack; and (2) those which maintain immunity from the attacks.

1. *Measures to Give Immediate Relief.* To cut short an attack the author has found nothing more effective than the hypodermic injection of $^1/_{25}$ grain of strychnine, $^1/_{15}$ grain of morphine, and $^1/_{150}$ grain of atropine. Sometimes the strychnine and atropine give relief without the morphine. In a severe attack it is advisable to combine the morphine. After the attack is relieved the morphine is to be abandoned, and the strychnine and atropine continued every other evening, at bedtime. The strychnine is to be gradually increased.

If, in case the hypodermic injection just referred to gives no relief in the course of half an hour, give a quarter of a

* "Pulmonary Consumption," 1872.

† "Diseases of the Chest," 1862.

grain of morphine alone, subcutaneously, or allow the patient to inhale the fumes of burning, dry, nitre paper, or breathe a pearl of amyle nitrite crushed in a handkerchief. In addition to the above the author has witnessed an attack of asthma immediately improve after the hypodermic administration of silver nitrate and cocaine in the neck, as recommended in the treatment of phthisis and chronic bronchitis.

2. *Measures to Secure Immunity from Asthma.* Among the most important measures to secure freedom from asthma are:

First. Silver nitrate and cocaine hydrochlorate introduced subcutaneously over the course of the vagi in the neck, once every week or ten days, as previously recommended.

Second. Strychnine in gradually ascending doses until the physiologic action of the drug is developed. This is secured by giving $^1/_{25}$ of a grain with 2 grains of quinine, four times a day, for a week, when it is increased to $^1/_{20}$ of a grain for another week; for the following week it is increased to $^1/_{15}$ of a grain, and so on, increasing it slightly every week until nervousness or twitching of the extremities are observed. The action of strychnine is enhanced by giving it with the syrups of the hypophosphites and of hydriodic acid.

Third, the alkalies and salicylates. There is no question that the element of gout or rheumatism, which shows itself in stiffness of the joints or in lumbago, or in sciatica, or in eczema of various parts of the body, underlies the asthmatic outbreaks; hence the alkalies and the salicylates are strongly indicated in connection with the above recommendations. The following combination has been frequently employed by the author in this complication:

℞ Sodæ Salicylatis, Lithiæ Carbonatis, Potassæ Iodidi, aa ..... ʒ ij
Liq. Ammon. Acetatis ..... fl ʒ i
Tinct. Strophanth ..... ʒ iv
Sol. Trinitrin (1 p. c.) ..... gtt. xl
Syr. Sarsaparillæ ..... fl ʒ i
Infus. Gent., q.s. ..... fl ʒ iv.

M. Sig. One teaspoonful four times a day.

Fourth. Mercury in some form to relieve torpidity of the liver and to regulate the secretions of the alimentary canal is a useful adjuvant. Two grains of blue-mass, every evening, followed occasionally with a small dose of Epsom salts, answers this purpose very well.

Fifth. The matter of diet is one of importance. Food should be light and of easy digestion, and neither animal nor vegetable food should be eschewed unless there is present an underlying gouty or rheumatic diathesis, when a meat diet should be restricted. A large quantity of water should be consumed by such patients at all times.

CHAPTER XXVI.

PLEURISY.

DEFINITION. Pleurisy is an inflammation of the pleural membrane, which condition may be followed by an accumulation of fluid and air in, and adhesion of, the walls of the pleural cavity.

SYNONYMS. Pleuritis, Inflammation of the pleura, Das Entzündliche Seitenstechen (German), pleurésie (French).

CAUSES. *Age*—It is common at all ages, but is more liable to occur between twenty and thirty.

Sex.—Sex has no influence on its production in early childhood, but in adult life the male sex is more prone to it.

Climate.—It is most prevalent in cold climates and seasons.

Intoxication. The poisons of gout, rheumatism, beriberi, influenza, syphilis, septicæmia, smallpox and of other eruptive fevers seem to have a special tendency to irritate the pleural membrane and to engender pleurisy.

Injury to the Chest. Wounds and contusions of the chest-wall frequently lead to pleurisy, especially when there is present a gouty, rheumatic or some other predisposing diathesis.

Diseases of the Lungs. The early stage of acute pneumonia, especially the croupous form of this disease, is very prone to be complicated with pleurisy. The same is also true of chronic bronchitis, bronchiectasy and phthisis.

Other Diseases. Bright's disease in children is very liable to lead to pleurisy. Pleurisy may be an extension from pericarditis.

PATHOLOGY. The pleural cavity is formed by a serous

covering of the inner exposure of the thoracic cavity and by a similar covering of the outer surface of the lungs—the former being called the parietal, or costal, and the latter the pulmonary layer. Now it has already been shown that the pulmonary layer consists of an outer dense, and an inner loose, layer, the latter of which dips down into the lung and divides it into lobes and lobules, and really forms the fibrous framework of the lungs which supports the lymphatics, blood-vessels, etc. The pulmonary layer contains stomata, or free surface openings, which are in direct communication with the underlying lymphatic vessels, and is well supplied with unstriped muscular fibers.

Among the earliest pathologic manifestations of pleurisy are intense hyperæmia of the pleural membrane, which changes from its moist, glistening appearance to a dry, dull-brown color. Its epithelium desquamates rapidly, and fine red points are seen scattered thickly over its surface. In a short time there is a surface exudation of serum from the pleural blood-vessels, which may be deposited, wholly or in part, as fibrin or serum. If it is principally deposited as fibrin it is called fibrinous pleurisy, and if it accumulates chiefly as serum it is known as serous pleurisy. It is seen, therefore, that the exudation process in pleurisy is the same as that which obtains in croupous pneumonia, or in croupous laryngitis. The fibrinous deposit, as in acute pneumonia, may undergo fatty degeneration become absorbed, and leave the pleural membrane entirely intact; or it may form adhesions with the opposite pleural surface, and enter into an imperfect organization. Occasionally adhesions of this kind give rise to sacs of various sizes, in which is contained an effusion of serum or pus, etc. These isolated accumulations often render a diagnosis difficult. In serous pleurisy there is a limited deposit of fibrin, while the pleural cavity may be filled with straw-col-

ored serum, containing flakes of fibrin, leucocytes and red blood-corpuscles.

Again the tension in the circulation of the pleural membrane may be so great that the blood, instead of allowing its serum to filter through the walls of the capillaries, breaks bodily into the pleural cavity and accumulates there. This is the hemorrhagic variety of pleurisy, and it finds its counterpart in the hemoptysis which occasionally accompanies croupous pneumonia. Furthermore, the analogy between pleural effusion and acute pneumonia holds true in other respects. Just as the red blood-corpuscles transude into the air cells of the lung during the acute pneumonic process and give rise to the red or rusty expectoration, so is there more or less of a constant tendency towards hemorrhage from the pleural surface in both the serous and fibrinous variety of pleurisy, resulting in a brownish or bloody discoloration of the effused liquid.

On the other hand, an enormous number of colorless blood-cells, or leucocytes, may migrate through the walls of the dilated pleural capillaries, collect in the pleural cavity, and multiply there still further by division. These cells become incorporated at once with the serum that is effused simultaneously and give rise to what is known as a purulent exudation, or empyema. Rindfleisch* describes the mechanism of this process in the following language: "The exudation thus becomes purulent on its way from the blood-vessels to the free surface of the serosa, and thus we obtain the purulent exudation." Not every purulent effusion of the pleural cavity is such from the beginning of the affection, but is transformed into it during its course. According to Rindfleisch† a fibrinous pleurisy may apparently pursue a favorable career when suddenly the fibrous pseudo-membrane, in virtue of its luxurious vascularization, begins to generate an

* "Text-Book of Pathological Histology," p. 268.

† *Op. cit.*, 269.

abundance of pus cells, while at the same time large shreds of fibrin liquefy into pus, the pleural membrane becomes like the granulating surface of a wound. Under these circumstances large quantities of pus frequently collect in the pleural cavity in a short time.

Now when we inquire into the mechanism of liquid effusion in pleuritic inflammation it will be found that this depends in a great measure on the principle of inflow and outflow of the pleural cavity. Normally the quantity of liquid which is secreted in this sac is balanced by the amount that is absorbed, and hence an accumulation is impossible, but when the supply is increased and the rate of absorption is lessened, as is the case in the inflammatory and fibrinous stages of pleurisy, the normal equilibrium is disturbed and the liquid frequently collects in large quantities.

Independent of any inflammatory state of the pleura there is another serous effusion of the pleural cavity, which is engendered by obstruction of the circulation outside of the pleural cavity. Thus, for example, diseases of the heart, liver, or kidneys very frequently embarrass the circulation, and give rise to a passive hyperæmia of the pleural capillaries and a consequent accumulation of serous fluid in the pleural cavity, as well as in other serous sacs. An accumulation of this kind in the chest cavity is known as hydrothorax.

In extensive effusion of the pleural cavity the lungs become very seriously compressed and embarrassed, and according to Rokitansky the following changes take place in these organs and in the thorax: "The thorax is dilated in a manner more or less apparent, the intercostal spaces are widened and prominent, the diaphragm is forced down into the abdomen, the mediastinum and heart are displaced to the other side, or, when the effusion is symmetrical, lie in the middle of the chest. The lung itself is compressed to a degree corresponding to the amount of the effusion and unless

old adhesions offer resistance it is constantly pushed upward and inward against the mediastinum and backbone. We find it reduced to the fourth, sixth, and even to the eighth part, of its normal volume, and flattened into a cake; its color is pale-reddish or bluish-gray, or lead-color, and its consistence is leathery, tough, and void of blood and air, and in a state of atrophy at the edges and surface. It is coated externally by the coagulum of fibrin, which extends from the costal to the pulmonary pleura. In partial pleuritus the displacement and compression are limited to a portion of lung corresponding to its seat and extent."

Symptoms. The symptoms of pleurisy vary with the variety of the affection.

Dry Pleurisy. With the exception of a slight pain there may be no symptoms in this variety of pleurisy. Entire adhesion of the pleural surfaces may occur in persons who have never experienced any severe illness or pain in the chest.

Pleurisy with Sero-Fibrinous Effusion. This form usually pursues an acute course, and is ushered in by a severe rigor, fever and pain in the chest, head and back, and dyspnœa. The cough is of a short, hacking character and usually distressing, and the expectoration consists of a frothy mucus, but may be entirely wanting. If the rigors recur regularly every day or two during the course of the attack it is an indication of empyema, or tuberculosis. The fever is a continuous one, with an evening exacerbation, ranges generally from 101° to 103° F., and lasts for about eight or nine days. The pain is of a stitchy or lancinating character, aggravated by breathing and, as a rule, locates itself in the side which is affected, but may also be found in the back. The trunk is usually bent toward the side which is involved. As the effusion progresses dyspnœa becomes more severe, but, as in the crisis of acute pneumonia, there may be a sudden improvement in the dyspnœa, and in other symptoms concurrent

with the subsidence of the fever. As a rule before effusion takes place the patient rests the easiest on the well side, while afterwards he is most comfortable on the affected side. Cyanosis is present very often and persists in many cases throughout the attack, especially if the respiration is embarrassed by a sudden onset of a copious effusion. The urine has a high specific gravity and is loaded with urates.

In some cases of acute sero-fibrinous pleurisy the fever moderates at the end of a week or ten days, the exudation ceases, and the patient has the appearance of a favorable convalescence when suddenly he becomes short of breath again, begins to cough and expectorate bloody sputum, the fever returns, the effusion increases markedly, and the case may end fatally or drag along for months.

Empyema or Pyothorax. This variety of pleurisy cannot by its symptoms be diagnosticated from the serous form except that it is of greater duration, and is accompanied by frequent rigors and an irregular rise and fall of temperature.

Physical Signs. However significant the symptoms of pleurisy may be they are less reliable than the physical signs that may be elicited. On inspection there is found diminished respiratory motion on the affected side, and during the stage of effusion there is little or no motion on this side and exaggerated movement on the healthy side. The affected side now also becomes larger, and there is obliteration of the intercostal spaces, especially at the base of the chest. The apex impulse of the heart is displaced toward the opposite or healthy side. During the stage of absorption the mobility of the chest returns, the intercostal spaces become more normal, the enlargement disappears and the cardiac apex beat returns to its normal place. After recovery there is permanent contraction of the affected side. In hydrothorax dependent on circulatory obstruction there is bilateral effusion and dilatation of the chest. On palpation there is found tenderness of and increase of the sense of resistance on the

affected side and diminution or absence of the vocal fremitus below the level of the liquid; and an increase of the same above, as well as on the normal side. In some cases the apex beat of the heart may be felt on the right of the sternum, or beyond the left of the nipple line. During the stage of resolution the apex beat of the heart returns to its natural place or, indeed, may be drawn too far in the other direction by the suction. In chronic empyema of the side of the chest the impulse of the heart is transmitted through the effusion to the thoracic wall and gives rise to what is known as "pulsating empyema."

Percussion shows very little if any change in the first stage of pleurisy. In the second stage, or in that of effusion there are dulness or flatness and an increased sense of resistance in the lower part of the chest, which terminate abruptly at the level of the fluid (unless the side is entirely filled) and exaggerated percussion resonance prevails above the fluid. If no adhesions confine the fluid, the line of the level of dulness changes by altering the position of the patient. For example, the level of the fluid is marked with ink or chalk, while the patient is sitting or standing, and on assuming the lying position it will be seen that the line of dulness sinks to a lower level in front and rises again to the former level when he returns to the first position. Sometimes a cracked-pot sound is perceived in the summit of the lung, which is brought about by the vibration of air in the trachea and large bronchi. This may mislead one to believe in the existence of a cavity in this area. During absorption of the liquid the level of dulness gradually recedes towards the base of the chest, but impaired percussion resonance may remain almost indefinitely on account of a thickened condition of the costal pleura. In chronic pleurisy, or in empyema, the flatness is marked generally throughout the affected side. In hydrothorax, dependent on circulatory obstruction, there is dulness or flatness over both bases, the level of which also varies

with the change of the patient's position, if there is present a sufficient quantity of fluid. On listening to the chest there are found feeble and wavy respiratory sounds, and a to-and-fro friction murmur. Respiration is suppressed below the level of the fluid, and puerile breathing is heard on the well side. A broncho-vesicular, or a bronchial respiration, is heard over the lung when it is pushed upwards into the apex of the thorax. In cases where the lung is not compressed too much a hollow, amphoric breathing is heard just above the level of the fluid. During the process of absorption the respiratory sounds return from the top toward the base of the lung, although in many cases respiration remains absent at the base for a long time. In other cases crepitant and subcrepitant râles develop in this area, which may linger for months and months. In all cases of suspected pleural effusion, even though the diagnosis seems reasonably certain, it is advisable to make an exploratory puncture into the pleural cavity with an ordinary hypodermic syringe, carrying a long needle, and for the following reasons: (1) to determine a liquid accumulation from pneumonia, thickening of the pleura and mediastinal tumors; and (2) to discover the nature of the liquid. The syringe should have good suction power and be aseptic. The skin should be well cleaned with alcohol at the point where the puncture is to be made. When the needle is inserted to its full length the piston of the syringe should be partly withdrawn, and if no fluid appears the needle is drawn outwards for a short distance, and the piston is drawn out still farther. If no liquid is found, and a strong suspicion exists that fluid is present, it is good practice to puncture the chest in other regions before exploration is abandoned. Whatever the consistency of the discovered liquid may be, it is important that it should be examined for the purpose of finding whether or not tubercle bacilli are present.

Differential Diagnosis between Pleurisy and Pneumonia.

Pleurisy.

1. Localized, stitchy or lancinating pain.
2. No convulsions.
3. Dry, short cough.
4. Persistent chilliness for a few days.
5. Irregular fever, rarely above 103° F.
6. No sudden decline in fever.
7. Impaired chest motion of and trunk drawn to affected side; friction sounds.
8. Flatness, with feebleness or absence of fremitus and of vocal resonance after effusion has occurred, and no prolonged expiration or bronchial breathing.
9. After effusion the intercostal spaces are forced out, and the apex of the heart is displaced.
10. Liquid may be withdrawn by exploratory puncture.

Pneumonia.

1. If pain is present it is dull, deep and diffused.
2. Convulsions in children.
3. Cough, with rusty or bloody expectoration.
4. One severe rigor at beginning, exceptionally two.
5. Fever high from the beginning, usually above 103° F.
6. Sudden decline of fever in crisis, more gradual in lysis.
7. Probably no discernible difference in the chest movements.
8. Dulness, with increase of vocal resonance and fremitus, and bronchial breathing or prolonged expiration; crepitation.
9. No displacement of the thoracic organs.
10. No liquid to be withdrawn.

COURSE AND TERMINATION. Dry pleurisy is not a serious disorder. A general rule in regard to the prognosis of pleurisy may be laid down: that the more acute the affection is the more favorable its termination will be. The changes that occur in the course of pleurisy are not as sudden as those in pneumonia, and there is generally a slow return to health, or a gradual decline. The fever ascends sluggishly during the first six or seven days, and remains at a continual level for a short time. During the latter period the effusion occurs and after this the fever subsides. If absorption now takes place the symptoms markedly improve. The urinary secretion becomes more free and less high in color, the respiration is easier and the patient assumes an air of health.

But this amelioration may only be temporary, and may be followed by another attack of fever, by pain in the side and by further effusion, thus opening the way for the disease to become chronic. At this time the effusion is very liable to change from a serous to a purulent character, and if this takes place it will require months to recover from the disease. It must be remembered, of course, that the effusion may be purulent from the outset of the attack.

When the effusion occurs rapidly and becomes very copious it may seriously compress the heart and large blood-vessels and cause fatal syncope.

Empyema, unless relieved by thorough drainage, terminates in phthisis, or eventually causes death by the pus burrowing into other important cavities and organs.

TREATMENT. The treatment of a light attack of pleurisy without much or any fever resolves itself into a simple bandaging or strapping of the chest, the application of mustard plasters, of dry cups, or of fly-blister to the affected part. But if moderate or high fever is present it is very important that an ice-bag, or two, should be constantly applied over the involved area. It is safe to say that an early resort to the ice-bag in the treatment of this disease prevents, or at least

limits, the extent of fluid accumulation in the pleural cavity. Simultaneously with the use of the ice-bags four or five leeches, or a number of wet cups, may be applied, if the pain is not promptly relieved by the ice.

So much for the external therapeutics of pleurisy. On what line is internal medication to be conducted? It has already been stated that sometimes the rheumatic diathesis is associated with pleurisy. The pleural membrane forms a closed sac, and is a homologous structure to the closed synovial sacs of the joints, and in some cases of pleurisy the rheumatic poison concentrates its activity on the closed sac of the chest, and rheumatism of the pleura instead of rheumatism of the joints results. In some cases, indeed, the two co-exist. In both of these conditions there is an interference with the absorption of serum and the latter accumulates in consequence. It is advisable, therefore, in cases of pleural effusion to administer the salicylates and alkaline preparations, provided the affection occurs in individuals with a family or personal record of rheumatism. For this purpose the following formula is recommended:

℞ Sodæ Salicylatis
Lithiæ Carbonatis } aa ..... ʒ ij
Potassæ Iodidi
Liq. Ammon. Acetatis ..... fl ℥ i
Syr. Sarsaparillæ Comp. ..... fl ℥ iss
Infus. Gentian, q. s ..... fl ℥ iv.

M. Sig. One teaspoonful every four hours.

That which is true of the curative effects of the salicylates and of other alkalies in rheumatic pleurisy is also true of the action of the same drugs in pleurisy that is incidental to influenza. Moreover, the pleurisy, which is of traumatic origin, is often amenable to the influence of the same agents. For it must be borne in mind that not infrequently in such instances the injury is merely the exciting cause that calls into activity the latent rheumatic tendency. In other words, if the latter had not previously existed pleurisy would not develop. This is well illustrated in injury of joints in per-

sons who are predisposed to rheumatic pain. A comparatively trivial strain of a joint in such individuals often leads to prolonged pain, stiffness and even swelling of this structure, and the salicylates and other antirheumatic agents greatly assist other measures in curing this condition.

What is to be done after effusion has firmly established itself. Is early surgical interference advisable at this stage of the disease, or is it good policy to make further efforts to absorb the fluid through the instrumentality of drugs? In regard to surgical measures a few principles may be laid down as a guide: (1) Surgical interference is peremptory when the liquid shows no tendency to undergo absorption; (2) when the liquid is so voluminous as to hamper the function of breathing and circulation; and (3) when the fluid accumulates very rapidly. Under other circumstances a preliminary attempt ought to be made to produce resorption by giving the salicylates, potassium iodide, calomel, lithium, etc., although it is inexpedient to place too much reliance on this procedure. If this produces no tangible results in the course of a few days, aspiration is to be resorted to.

Where should the aspirating needle be introduced, and how much of the liquid should be withdrawn at once? The aspirating needle should enter the pleural cavity in the sixth or seventh interspace, preferably the latter, in the axillary line. The skin is cleaned and disinfected and the index finger is pressed in the intercostal space, and the needle is quickly thrust through the chest wall immediately in front of the finger tip. The liquid should be allowed to flow slowly into the aspirating bottle, and if pain or cough or constriction of the chest occur aspiration must be stopped until these symptoms subside, and then be resumed. No further efforts at aspiration should be made when the fluid becomes bloody, when there is constant pain and coughing, or great difficulty of breathing.

Sometimes the effused liquid is isolated or encapsulated by

adhesions, and under these circumstances the aspirating needle must be introduced at the spot where the fluid was discovered by the hypodermic needle. It is never safe to assume that the liquid is located anywhere else.

If the effusion is purulent, is it best to aspirate or to make an incision and drain the cavity? That aspiration in empyema is occasionally successful is true, but the author believes that time is saved by making an early and a free incision. When the empyema is general an incision large enough to admit a good sized drainage tube is to be made into the seventh intercostal space, in the axillary region. Rib section is, as a rule, unnecessary and if the interspace is not wide enough it is better to trephine than to sever a rib for this purpose. The tube should just pass into the pleural cavity and no more, be sutured to the edge of the wound or have a fixed flange, and its free outside end should be covered with a layer of antiseptic cotton sufficiently thick to absorb the pus which drains into it. As the discharge diminishes another and a smaller tube should be inserted from time to time, and finally be abandoned altogether.

The internal medication in effusion of the chest cavity should be supporting in character. Cod-liver oil, syrup of the iodide of iron, of the hypophosphites and of the hydriodic acid, quinine and strychnine, as well as a liberal and nutritious diet are to be administered.

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