The student's manual of medicine / by John James Eberle.

THE STUDENT'S

MANUAL OF MEDICINE.

BY

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PART I.

DISEASES OF THE RESPIRATORY ORGANS.

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TO

THE MEDICAL STUDENTS

OF

GREAT BRITAIN AND IRELAND,

THIS LITTLE WORK

IS RESPECTFULLY DEDICATED

BY THEIR WELL WISHER,

THE AUTHOR.

PREFACE.

MEDICINE, as a science, has so rapidly progressed during the last few years, as to render the smaller manuals in use among students practically useless.

It is impossible rightly to understand and appreciate disease, without a sound anatomical, physiological, and pathological knowledge; and therefore my aim has been to supply as much information in a concentrated form, as the limited time and space at my disposal would allow. This work will contain everything of importance that is to be found in other books on the subject, and is therefore necessarily, to some extent, a compilation; but it is hoped that in the grouping of facts, and in directing the reader's mind to just conceptions and conclusions, this manual may be found to be something more than a mere collection of details.

The wording and general style, I am perfectly aware is, to say the least, peculiar, and frequently defective; yet, in my wish to supply an acknowledged want, I have been led to convey my meaning in as few words as possible, thus obtaining brevity and simplicity, at the expense of correctness of style and expression. As a medical tutor, my examinational experience has enabled me to give prominence to those points which afford material for questions; and I trust that this little work may enable the student to concentrate the knowledge he has gained in the lecture room, and promote a better understanding of the contents of the more extended treatises.

JOHN JAMES EBERLE.

Easingwold, May, 1876.

DISEASES

OF

THE RESPIRATORY ORGANS.

GENERAL RULES FOR DIAGNOSIS.

The Pulmonary Region extends from one-half to two inches above the clavicle, the apex of the right lung being slightly higher than that of the left. Anteriorly on the left side it extends down as far as the upper margin of the seventh rib, but on the right only to the upper margin of the sixth. Posteriorly as low as the ninth or tenth rib.

I.—Inspection (a.) *as to size and shape of chest.* The right side is normally about half an inch larger than the left.

Bilateral enlargement,	*e. g.*	Emphysema.
Unilateral ,,	*e. g.*	Pleural effusion.
Bilateral diminution	*e. g.*	Phthisis.
Unilateral ,,	*e. g.*	Cirrhosis, or result of Pleurisy.

(*b.*) *As to play of chest.* Normal Respiration, 16 to 20 per minute. Respirations are diminished in frequency in cerebral affections, but increased in all diseases of the lungs.

The respirations may be wholly *Thoracic*, when anything impedes the action of the diaphragm, *e. g.* Peritonitis, or *Abdominal*, *e. g.* Pleurisy.

The play of one side may be diminished. *In upper part*, *e. g.* Phthisis. *As a whole*, *e. g.* Pleurisy and spinal hemiplegia. When the air in the lung is rarified, the soft parts above the clavicles sink in, the ribs are drawn inwards

towards the mesial line, and the epigastrium is sucked in with each inspiration, owing to the greater external atmospheric pressure.

Intercostal depressions are effaced in pleural effusion.

II.—Palpation, *Vocal Fremitus, or Vibration, augmented* in all diseases which increase the density of the parenchyma of the lung, *e. g.* Pneumonia, Phthisis, etc.

Diminished or annulled, e. g. Pleural effusion, etc.

Friction Fremitus. In health none, but when the surfaces are dry or roughened, a sensation of a rubbing movement is conveyed to the hand.

III.—Percussion. Resonance is in direct proportion to the amount of air in the part percussed. *Increased* in Emphysema (but not always, as the parietes in old subjects have been stretched as much as possible, and therefore cannot yield to the wave of sound); also in Pneumo-thorax. *Decreased* when there is fluid in the pleura, and in all conditions which render the parenchyma of the lung solid.

Percussion Sounds of a Special Character. *Amphoric Resonance*, similar to that occasioned by striking an empty cask, is heard when air is contained in a cavity of the lung or pleura.

If the cavities contain a small quantity of fluid, drops falling from the upper part to the fluid below will give rise to a sound known as *Metallic Tinkling*.

Cracked Pot Sound. The patient being directed to open his mouth, the chest must be sharply percussed. It is heard when there is a superficial cavity communicating with a bronchial tube, and is caused by the air and fluid being suddenly driven out.

It may also be heard in cases of bronchitis, where the secretion is thin, and has gravitated to the lower parts of the lung (Stokes),—or where there is solid lung posteriorly and a thin layer of healthy lung in front.

IV.—Auscultation (*mediate and immediate*).

Healthy Respiratory Sounds. *Vesicular murmur* is a diffused, soft, breezy, sighing sound, whose probable site of production is the airsacs of the lungs (Hyde Salter). Expiration is shorter and less audible than inspiration. *Bronchial breathing and voice* is heard even in health, between the scapulæ and near the sternal end of clavicle.

Morbid Sounds. *Weak Vesicular Breathing, e.g.*, commencing Phthisis.

Prolonged Expiration, caused by anything which prevents the exit of air from the lung, *e. g.*, phthisical deposits, cancer (rare), or in emphysema, on account of the want of resiliency in the lung.

Puerile Breathing, or compensatory breathing, caused by obstructive disease in some other part of the lung.

Bronchial Breathing. Hollow, e.g., solidification of lung. *Tubular, e.g.*, pressure from pleural effusion.

Cavernous Breathing, when it resembles the sound produced by blowing into a small cavity, *e.g.*, pulmonary cavity, or a very globularly dilated bronchus.

Amphoric Breathing, when it gives the sensation of blowing into a large cavity with thick walls, and is diagnostic of pneumothorax with pulmonary fistula.

Friction sound.—A too-and-fro creaking sound, due to a roughened state of the pleura.

Rales or Rhonchi, Dry Sounds.—*Sonorous* (snoring or cooing sound), in large tubes; *Sibilant* (whistling or piping) in smaller tubes.

Indicate that some of the bronchial tubes are abruptly narrowed at some particular part, either by spasm, as in asthma, pressure of cancerous and other deposits or tumours, or the presence of tenacious secretion or thickened mucous membrane, the result of inflammation.

Dry Crackle or Crepitation (similar to the sound produced by crumpling tissue paper) is heard in emphysema, especially the interstitial form.

Moist Sounds, *Cavernous Rhoncus* (gurgling or bubbling), indicates the passage of air through fluid in a cavity of the lung, or in a dilated bronchial tube.

Mucous Rhoncus, caused by the bursting of air bubbles in the fluid present in the larger bronchial tubes. *Submucous Rhoncus*, when it takes place in the smaller tubes.

All these sounds are unequally and occasionally evolved, and are heard both with inspiration and expiration. *Dry or fine crepitation*, resembles the sound produced by rubbing a lock of hair between the fingers, close to the ear. It is equally and rapidly evolved and heard *during inspiration only*. It is indicative of a commencing pneumonia, and is caused by the air entering and separating the

agglutinated-together walls of the air cells. *Crepitatio redux*, heard during the resolution of pneumonia *during inspiration*, and also to a less extent *during expiration*, is caused by the bubbling of air and liquid in the minute bronchi and air cells.

Voice Sounds.—*Bronchophony.*—Bronchial voice, as heard over the bronchial tubes in health, being audible over portions of the lung where it is not naturally heard, and caused by a condensation of the lung, in the vicinity of large bronchial tubes.

Pectoriloguy.—When the voice articulates itself into the ear (as it naturally does over the trachia), from some portion of the lung where it is not normally heard, indicates a cavity communicating with a bronchial tube, or a dilated bronchus, surrounded by consolidated lung.

Ægophony.—A tremulous modification of the voice compared to that of the exhibitors of Punch. *Laennec* considered it pathognomonic of pleurisy with effusion, but it may, although rarely, be heard in simple consolidation of the lung.

THE LARYNX.

It is absolutely necessary that the student should be perfectly acquainted with the natural anatomical characters of the part and functions of the organ before he attempts to inquire into its diseases. The larynx, the organ of voice, is made up of several cartilages, connected together by muscles and ligaments, and lined by a ciliated mucous membrane.

Now this mucous membrane particularly interests us, for it is here that most of the diseases have their seat, and it must be evident that if it be swollen, the walls being unyielding, obstruction must ensue. Again, it is connected to the cartilages by areolar tissue, which nearly absent below is abundant above the vocal cords, especially in the adult.

The opening into the larynx, or rima glottidis, in the adult is triangular in shape, about an inch in length, and a quarter in width at the base, and separates widely during inspiration, returning in expiration; these movements being reflex. The *trachea* is similarly constituted, and terminates in the two bronchial tubes which dichotomously dividing end in pulmonary vesicles.

ACUTE SIMPLE LARYNGITIS.

Definition.—With the view of basing the pecularities of a disease upon its pathology, I have chosen this term, because by it we must understand an ordinary inflammation of the mucous membrane lining the tubal organ of voice.

Pathology and Morbid Anatomy.—*In the first stage, that of active hyperæmia,* we find the membrane swollen, and gorged with blood, and its ordinary secretion scanty and tenacious. After a time, the epithelium becomes detached, younger cells are incompletely developed, become indistinctly granulated, and with the cells from the mucous follicles, are thrown off as soon as formed, constituting an abundant yellow muco-purulent secretion. The membrane may become flabby, effusion occur into the submucous areolar tissue, and the muscles rendered sodden with serous infiltration.

Causes.—*Acute Simple Laryngitis as an idiopathic affection is a most rare disease,* being most frequently traumatic from the inhalation of irritating vapours, and in children from sucking boiling liquid and steam from the spout of a tea kettle. *A subacute catarrh,* or common cold, is among the most frequent of diseases, and is often secondary to small-pox, measles, and other infectious diseases.

Symptoms and Course.—May set in with or without the usual rigors and pyrexial condition. *The local signs* are at first, sense of soreness in the throat, aggravated by speaking, coughing, or swallowing; tenderness on pressure; the voice is hoarse and the cough husky, owing to the vocal cords being thickened, and incapable of vibrating sufficiently frequently, and regularly as to produce the natural, clear, and high-pitched tone. Violent fits of coughing and spasm, no doubt due to an irritable and hyperæsthetic state of the hyperæmic mucous membrane. Expectoration scanty, difficult, and painful, consisting of a glairy mucus, streaked with blood. If the inflammation be more intense, the obstruction due to the submucous infiltration will give rise to laboured respiration and dyspnœa. The voice and cough is noiseless, as the relaxed and swollen cords are incapable of any degree of tension. The noisy and stridulous inspirations less frequent than in health, are rendered more difficult by so-called spasm

of the arytenoid muscles, but perhaps in this stage more truly dependent upon, according to *Niemeyer*, a paralysed condition of the infiltrated and sodden muscles. The face and eyes are swollen and congested, the supraclavicular and substernal soft parts are depressed with each violent attempt at inspiration, the countenance is anxious and the face livid, and a fatal issue most frequently takes place either gradually or suddenly by apnœa. If the inflammation is mild, the first stage is followed by one of abundant secretion, with a diminution of all the symptoms, and a speedy return to health.

Treatment.—Although idiopathic laryngitis but rarely kills, being seldom more pathologically than an ordinary hyperæmia or catarrh, the traumatic variety very frequently does from *œdema* of the *glottis*. *In the first class of cases*, simple measures will suffice, such as hot applications to the throat; perhaps a leech or two to the upper part of the sternum; aconite or tartar emetic in small but frequently repeated doses, to reduce the tension of the blood vessels, and tendency to inflammatory exudation; inhalations of the volatile principles of benzoin, hop, or conium; combined with perfect rest in a warm and moist atmosphere; and a slop diet. *In traumatic cases*, where the œdematous infiltration is to be feared, topical applications of solution of argent nitrat, by means of a probang, or scarification with a guarded sharp pointed curved bistoury, with the aid of the laryngoscope. Drs. Bevan and Croly of Dublin have successfully treated cases by the application of leeches to the upper part of the sternum, an emetic followed by a cathartic, two grains of calomel every half hour combined with mercurial inunction, until slight salivation is induced. When these remedies fail, and dyspnœa increases, *tracheotomy should be performed before the case is too far advanced.*

CHRONIC INFLAMMATION AND ULCERATION OF THE LARYNX.

Chronic Laryngitis. Causes.—Often the sequel of the acute, or may have been chronic from the first, the result of functional excesses, the presence of ulcers or tumours, and syphilis, &c.

Morbid Anatomy.—Chronic inflammation and hypertrophy of the mucous membrane, with congestion and tortuosity of its vessels. Thickening of the vocal cords. Enlargement of the racemose glands (Green.)

Ulceration.—The ulcers due to *typhous poisoning* are supposed to be either analogous to those affecting Peyer's glands, or to a persistent hypostatic congestion. Those of *small-pox* to the spreading of the eruption from the skin. *Syphilitic* ulcerations usually involving the the epiglottis are irregular and ragged in shape, and show a tendency to heal at one part and advance at the other. *Tuberculous* deposits in the larynx, according to *Virchow*, are by no means rare, but owing to their superficiality and liability to accident from without, tend to break down and form an ulcer. All these ulcerations may lead to more or less destruction of the soft parts and cartilages, and the syphilitic variety is peculiarly prone to lead to necrosis of the cartilages.

Symptoms.—A cracked, hoarse, and perhaps inaudible voice, and barking cough. In mild cases, weakness of voice, fatigue after speaking, and an inclination to clear the throat and swallow. *If we have pain, difficult and painful deglutition, and sputa streaked with blood, the presence of ulceration may be inferred.* The laryngoscope alone will, however, reveal the true nature and character of the disease.

Treatment.—*Rest of function, warm atmosphere, inhalations* of muriate of ammonia, tannin, nitrate of silver, &c. Topical applications of solution of nitrate of silver, or chloride of zinc by means of a probang. In *specific ulcerations* we must at the same time treat the constitutional disease; *e. g., in syphilis*, preparations of *mercury* or *iodine*, according to the stage of the affection.

ŒDEMA OF THE GLOTTIS.

Definition.—By this we understand any effusion into the loose areolar tissue, abundant above the true vocal cords.

Causes.—Inflammation, ulcers, fevers, especially small-pox, scarlatina, erysipelas, and typhus, Bright's disease, sting of wasps, &c..

Symptoms.—Occurs almost exclusively among adults, and is usually a secondary affection. The tumour, which may be even as large as a pigeon's egg, gives rise to a sense of a foreign substance at the seat of obstruction, and a disposition to hawk and swallow. During inspiration it is sucked in, and thereby arrests that act; expiration is comparatively free. If laryngitis be absent, the voice will not be altered. *The tumour can also be felt* by the finger at the base of the tongue, and seen with the aid of the laryngoscope.

The dyspnœa increases rapidly, the patient becomes terrified, gasps for breath, the face is livid, the pulse small and irregular, a cold clammy sweat breaks out over the body, and death ensues by apnœa from œdema of the the lungs or paralysis of the heart.

Treatment.—Being aware of the fact that ordinary laryngitis is rendered fatal by the occurrence of œdema; that as a secondary affection in small pox and erysipelas, it frequently kills suddenly, it behoves us to be ready with an efficient treatment. Under œdematous laryngitis, I have enumerated the various modes of combating this affection, but I would just draw your attention to a few practical facts which I have taken from an able and practical pamphlet, written on the subject by Professor J. Stannus Hughes, of *Dublin*. He writes: "Effusion into the areolar tissue in *œdema of the glottis never extends below the true cords*, being limited at this point by the direct adhesion of the mucous membrane to the subjacent fibrous tissue." "That œdema below the glottis never co-exists with œdema above the glottis, and, therefore, in performing laryngotomy for the latter, no obstruction need be anticipated to interfere with the operation itself, or of its subsequent results." Œdema of the glottis so often kills suddenly, that the practitioner must always be prepared, even unassisted, to grapple with it at any moment." *When there are urgent symptoms of obstruction, tracheotomy ought to be performed at once, and not deferred until death from asphyxia appears imminent.*

LARYNGITIS WITH EXUDATION.

Etiology.—This disease, commonly known as *true croup*, most frequently occurs in children between the periods of

the first and second dentition. Delicate children are as liable to be attacked by it as are the robust, and it proves fatal in almost half the cases. It often prevails as an epidemic, and is most common in northerly, damp situations.

Pathology and Morbid Anatomy.—The exudation which characterises this form of laryngitis, consisting of fibrine entangling young cells, which rapidly coagulates, involves the epithelium only, thus differing from that of diptheria, which is developed in the substance of the mucous membrane itself, and sloughing, leaves an ulcer. In the vast majority of cases it commences in the upper part of the trachea, extends on to the epiglottis, and frequently into the pharynx. Under the exudation the mucous membrane is seen to be affected by the inflammation exactly as described under simple laryngitis.

Symptoms and Course.—May set in suddenly, or be preceded for two or three days by ordinary catarrhal symptoms. The little patient in the night awakes with a sensation of impending suffocation, the *voice is hoarse and harsh*, the *cough brassy, respiration* protracted, difficult, and accompanied by a *sawing, wheezing sound. There is more or less fever.* The symptoms due to the obstruction to the entrance of air become more and more marked, the child instinctively clutches at its throat in its endeavour to remove the cause, is restless, and terror is depicted on its countenance. The *face is livid*, every muscle is called into action to expand the chest, but without any result, the *alae nasi dilate*, and the voice is whispering and cough abortive and stridulous. The *air in the lungs becoming rarified*, atmospheric pressure causes the soft parts above the clavicle to sink in, the ribs are drawn inwards towards the mesial line, and the epigastrium sucked in with each attempt at inspiration. This is only seen in one other affection—atalectasis pulmonum. Either the membrane becomes loosened by suppuration and expelled, and the child gradually recovers, or the paroxysms become more frequent, the flushed face of the child grows pallid, and assumes a drowsy aspect, the respirations are diminished in frequency and strength, and loose their stridulous wheeze, and death takes place by slow apnœa; or, in other cases, *convulsions* or *paralysis of the heart may end the*

scene. The usual period of the disease is from one to three or four days.

Diagnosis.—The insidiousness of the attack, the presence of fever, great alteration in the voice, and signs of obstruction, with the presence of pharyngeal patches of croup membrane, or the expectoration of shreds, occurring in a child, will distinguish it from *acute simple laryngitis.* From *Laryngismus Stridulus*, by the sudden accession and departure of the fits, by the absence of dyspnœa, cough, and change in the voice during the intervals, and the non-existence of fever in that affection.

Treatment.—That of acute simple laryngitis, *a warm, moist atmosphere, of* 90°, two to twelve leeches, according to the age and strength of the patient. *Emetics* are useful at intervals to expel the accumulations of mucus and the obstructed membranes. *Hot applications* to the throat, *inhalations* of spray from lime water, ether, *topical applications* of solution of nitrate of silver, to hasten the separation of the false membrane, two or three grains each of *Calomel and James's Powder* every two or three hours, until the bowels act; expectorants, such as Ipecac and Senega may be given, combined with the *Iodide of Potassium* or *Chlorate of Potash.* In the *later stages stimulants must be freely administered.*

LARYNGISMUS STRIDULUS.

THYMIC ASTHMA, SPASM OF GLOTTIS.

This affection, although most usually placed nnder diseases of the nervous system, had better be briefly considered here on account of its being commonly known by the term False Croup. It is due to some affection of the Pneumogastric, either from centric cause, pressure on its course, or reflex from irritation of its periphery, *e.g.*, strong light (5th), bronchial catarrh, indigestible food in the stomach or alimentary canal, worms, teething, etc. If it is to be considered as a purely nervous affection, we must exclude spasm due to local catarrh. It is most common during the first dentition, especially from the fourth to the tenth month. The infant suddenly, from contraction of

the muscles closing the glottis, "holds its breath." Inspirations are violent and gasping, accompanied by a crowing sound. The eyes become fixed and glaring, and the countenance is expressive of great distress. The head is thrown backwards, and we may have spasms of the muscles of the hands and feet. In a few minutes, the spasm relaxing, a noisy expiration is followed by a fit of crying, and the child is well. Death may occur from convulsions. *There is no cough, no change in the voice, no fever, unless laryngeal catarrh be present.*

TREATMENT.—Remove the cause. *During the attack*—Plenty of air, warm bath, sinapisms to the calves of the leg, cold affusion, ammonia and ether to the nostrils, and antispasmodics, *e.g.*, asafœtida and musk.

CROUP.

Croup must now be understood *as a generic term*, signifying the presence of a certain set of symptoms, viz., change in voice (hoarseness or aphonia), brassy cough, with laryngeal obstruction and more or less spasm, *and not as any one peculiar disease.*

We can thus understand why Dr. Ware recognizes four varieties, viz.:—1, MEMBRANEOUS or TRUE; 2, INFLAMMATORY; 3, CATARRHAL; 4, SPASMODIC; for in all these affections the above symptoms are present as the results of the loss or modification of function due to some functional or organic lesion.

ACUTE PLEURITIS.

DEFINITION.—Inflammation of the two internal sides of the pleura, ending in exudation of plastic lymph or serum, in rare cases only terminating in suppuration.

CAUSES.—*Primary Pleurisy.*—Cold, injuries, excessive muscular action acting on a pleuritic diathesis. *Secondary Pleurisy.*—Albuminuria, acute rheumatism, scarlatina, measles, &c. It is a unilateral affection, and very rare under five years of age. A circumscribed pleuritis sometimes occurs in connection with phthisis.

Pathology and Morbid Anatomy.—I shall for the sake of convenience and clearness divide the disease into three stages, viz.: 1st, *That of inflammation;* 2nd, *Effusion;* 3rd, *Resolution.* First Stage—Inflammation.—The pleura (costal first) is reddened from hyperæmia, thickened from inflammation and proliferation of its connective tissue, dull looking from loss of its epithelium, and rough from papillary granulations of connective tissue elements. Second Stage—Effusion.—Coagulable lymph more or less abundant is now poured out upon the surface of the pleura, and is soon followed by, or accompanied with, serous effusion, which may be so great as to dilate the chest, compress the lung, and displace the diaphragm and neighbouring viscera. Third Stage—Resolution.—The serum becomes absorbed, the fibrin undergoes fatty metamorphosis, liquefies, and is also absorbed. More or less thickening and adhesion of the surfaces remain. Or it may become *chronic*, and after having rendered the lung useless by compression may suddenly be absorbed. If there is *purulent effusion*, it may evacuate itself by the parietes, or through the lung.

Physical Signs—First Stage.—Constrained movement of the affected side. Slight friction sound and fremitus. Second Stage.—Friction sound and fremitus increased as long as the surfaces are in contact, but when the effusion separates them are lost. Puerile breathing on the sound side. Dulness on percussion, varying in seat according to the position of the patient. Diminution of vesicular murmur. Decreased vocal fremitus. Ægophony. *If the effusion be great*, besides these we shall have bulging and immobility of the chest wall, the intercostal grooves are effaced, the heart, diaphragm, &c., are displaced, and the lung being compressed the vesicular murmur is absent, and we have bronchial breathing of a tubular character.

Symptoms and Course—First Stage.—Duration about 24 hours. Invasion sudden, but occasionally pain is felt for two or three days previously. Chills and perhaps frequently repeated rigors. Symptomatic inflammatory fever. Temperature seldom above 102 degrees. Headache, pain in back, thirst, anorexia, flushed face, suppressed secretions, scanty high-coloured urine, &c., &c. Sharp, lancinating pain in side referrable to the nipple region,

due to the stretching of the inflamed pleura. Full, hard, incompressible pulse, from ninety to one hundred per minute. Patient lies on sound side or back. Respirations abdominal and increased in frequency, on account of inspiration being checked before completion in consequence of pain. Suppressed cough, with little or no expectoration.

Second Stage.—Symptoms continue, but are modified as the effusion increases. Pain lessens, or if the effusion is great, it is of a dull character, due to pressure on the diaphragm. Fever diminishes. Decubitus on the the affected side. Dyspnœa, in direct proportion to the amount of effusion, and compressed and congested state of the lung.

Third Stage.—Gradual recovery. Absorption may commence immediately, but sometimes is not completed for several weeks. Uncomplicated pleurisy is rarely attended with a fatal result. Death in rare instances takes place from rapid effusion, or in broken down constitutions, from asthenia.

Treatment—First Stage.—Rest in bed, &c., &c. If the patient is plethoric, and the pain and other symptoms are acute, general blood-letting to ten or twenty ounces is recommended, or until the pulse is softer and less frequent, and the breathing less oppressed. But as a rule, a few leeches, followed by hot linseed poultices or other warm, moist applications, will be the better practise. We are able nowadays to produce the same amount of depletion by therapeutic agents, and thereby avoid the permanent impairment of the vital powers. *Veratria* $\frac{1}{12}$ gr., *Tartar emetic* gr. $\frac{1}{8}$ to gr. $\frac{1}{2}$, *T*r. *aconite* m. 1 to 3, all act as powerful vascular depressants. The tincture of aconite B. P. in drop doses every quarter of an hour, until the face pales and the pulse is influenced, is a most potent and valuable remedy. As pain alone is sufficient to cause increased afflux of blood to a part, we must diminish it by *opium* in frequently repeated doses. *Calomel* gr. $\frac{1}{2}$, with *opium* gr. $\frac{1}{3}$ every half hour, combined with inunction, until slight mercurialization is produced (Walshe.) Cold, wet compresses (Niemeyer.)

Second Stage.—As the first, according to the severity of the symptoms, in order to hasten absorption, we must regulate the ingestion of liquids, administer diuretics,

diaphoretics, and purgatives, sorbefacients, *e. g.*, mercury and iodine, and apply blisters. If there is any tendency to exhaustion, we must support the strength by stimulants and nutritious diet. Should the fluid not diminish, or should it become excessive, causing extreme dyspnœa, we must perform *paracentesis* behind the angles in the 5th or 6th interspace.

Operation.—A small incision is to be made through skin and fascia with a lancet close to the lower rib, and then the trocar and canula, warmed and oiled, and fitted to a tube placed under water, an aspirator or an india-rubber suction bag, is to be introduced. When the chest wall is perforated, the trocar should be withdrawn and the canula pushed well home. Dr. Fuller considers that it is frequently impossible to evacuate the fluid without the introduction of air, as the lung is unable to expand sufficiently to expel it by itself.

EMPYEMA OR PYOTHORAX.

Pus in the cavity of the pleura may be the result of a suppurative pleuritis, or derived from the bursting of an abscess, or by ulceration from a tuberculous cavity of the lung. If from the latter causes, we have suddenly the symptoms of pleurisy developed in a subject who has been suffering from some lung affection. It is difficult to say when we have pus in the pleura, but we may assume its presence if there is hectic fever, creamy tongue, with red tip and edges, quick pulse, exacerbations of fever, loss of flesh, absence of pain, profuse sweating, flushed face, clubbing of finger ends; but we can only be certain by puncturation with a grooved needle. It will be unaffected by sorbefacients. The physical signs will be those of chronic pleurisy. Perforation of the lung may ensue, marked by the symptoms of a slight pneumonia, and sudden purulent expectoration. Or spontaneous perforation of the chest wall may take place, termed "Paracentesis by necessity." Paracentesis ought not to be delayed too long, as the lung will become carnified by compression.

Treatment must be palliative, with nutritious diet. In *chronic cases* a probe may be introduced through the opening, and made to press against the lower and back

part of the chest wall, its point cut down upon and brought through. A perforated indiarubber drainage tube, as recommended by *Chassaignac*, is then to be introduced, and brought out at the lower hole. The pus will then drop away as secreted, and the cavity may even be washed out.—*(Drs. Goodfellow and De Morgan.)*

HYDROTHORAX

Signifies *a passive dropsical transudation into the cavity of the pleura*, and ought not to be considered as a special disease, but merely as a symptom of a general cardiac or renal dropsy. The fluid, consisting of water, albumen, and the salts of the serum of the blood, differs from that of pleurisy in containing no fibrinous coagula or other inflammatory exudation product. *It is a bilateral affection*. The symptoms will be those of chronic pleuritis, without any pyrexia, and, with the physical signs, are in direct proportion to the amount of fluid present. We must treat the causal disease, and *palliate by performing paracentesis*.

PNEUMOTHORAX.

Air in the cavity of the pleura is commonly the result of the bursting of a pulmonary cavity, or from the opening of a pleural effusion into the lung. It may, however, be caused by wounds of the chest wall, by ulcerative communications between the pleura and alimentary canal, or from evolution of gas from decomposition of pleural contents.

Symptoms.—Patient feels as if something had given way or burst in his chest *Intense dyspnœa*, due to the compressed state of lung, and, perhaps, collateral hyperæmia and œdema of the other. Decubitus on the affected side, or he has to sit up. *Pain* in region of lower ribs, due to pressure on diaphragm. Cyanosis and dropsy, *pulse small*, urine scanty, general congestion, *collapse*, and death in a few hours; or, in some cases, the patient may last days or weeks. As a pleurisy is most frequently set up, there will be pain, pyrexia, &c., in addition to the preceding signs of pure pneumothorax.

Physical Signs will be diagnostic of fluid in the pleura, causing *distension, displacement, etc.*, and that fluid being air, there will be *clear tympanitic resonance on percussion, amphoric voice and respiration.* As in ninety per cent of the cases, pneumothorax is the result of the rupture of a cavity in the lung, containing liquid, there will be an escape of fluid with the air; and even if not, some will soon be effused as the result of pleuritis. We shall then have either empyo or hydro pneumothorax, recognized by the additional signs of metallic tinkling, splashing sound on succussion, dulness below, varying in seat with the position of the patient, and tympanitic above. It is hardly necessary to observe that this must be a unilateral affection. If not the result of phthisis or gangrene, but of emphysema, a cure may follow by a circumscribed pleuritis being set up and sealing the orifice.

Treatment can only be palliative. Small stimulating doses of opium, combined with ether and other stimulants, to overcome the tendency to collapse, and relieve the pain and dyspnœa, turpentine and other fomentations. Puncturation of the chest not only relieves the compressed lung but also the sound one.

BRONCHITIS.

Etiology.—Bronchial catarrh, commonly a bilateral affection, is most frequently caused by cold, especially in the weak, and in those in whom a cardiac lesion (aortic or mitral regurgitant) keeps the lungs and bronchial mucous membrane in a chronically congested state. It is sometimes a symptom of typhoid fever, measles, and small pox, the result of the morbid state of the blood in those affections.

Irritants, *e.g.*, dust, cold air, vapours, &c. As an epidemic disorder, it is known as influenza.

Pathology and Morbid Anatomy.—In the 1st stage, *or that of hyperæmia*, we find the mucous membrane more or less swollen by œdematous infiltration, reddened by injection and ecchymoses, and the calibre of the tubes lessened. It is covered by a scanty, glairy, tenacious, transparent secretion, composed of detached epithelium, and a few young immature cells.

In the 2nd STAGE, *or that of rapid cell development*, the membrane is bathed in a more or less thin opaque secretion, of a yellowish colour, containing great numbers of young, immature epithelial cells, and of granulated leucocytes.

On opening the chest the lungs bulge out, on account of the air being unable to escape from the air cells through the narrowed tubes. In young children and weak or aged persons, the lobules sometimes are found collapsed. This is caused by the air entering with difficulty by reason of the secretion being forced into a narrower part, being able to escape in expiration by the side of the obstructing body, as the matters are driven into a wider part of the tube. The air cells therefore gradually empty and collapse.

SYMPTOMS AND COURSE.—Average duration, ten to twelve days. The inflammation usually commences in the mucous membrane of the nasal passages, constituting coryza, and travels downwards, occupying from a few hours to two or three days.

THE SYMPTOMS *belonging to the* 1st STAGE *are*: Inflammatory symptomatic fever, evidenced by chilly sensations, furred tongue, thirst, anorexia, suppressed secretions, lassitude, pulse full and strong, rarely over 100; increased temperature, 100° to 102°; pain, substernal, of an obtuse character; sense of constriction and tightness of the chest. The respirations are slightly increased in frequency; violent, irrepressible, paroxysmal, and painful cough; expectoration absent or scanty, consisting of glairy, tenacious mucous, sometimes streaked with blood. After two or three days the 2nd STAGE *is evidenced by the expectoration becoming abundant and muco-purulent*: the cough is said to be loose, and is followed by a sense of comfort. The fever and constitutional disturbance subside, and the patient gradually recovers. In the debilitated and aged it is a dangerous affection, as fever of an adynamic type is apt to be present with a quick, feeble pulse, brown, dry tongue, great debility, and low, muttering delirium. The tubes become blocked up, and death takes place from slow suffocation. In young children also this second stage frequently kills by inducing atalectasis, and it may be suspected if the respirations become frequent, the alae nasi dilate, and the face livid—the symptoms and signs of pneumonia being wanting.

Physical Signs.—First Stage.—Sonorous rhoncus, if large tubes are affected; sibilus, if the small ones. Second Stage.—Mucous or submucous rhoncus. No other modifications of the normal sounds.

Treatment.—When coryza appears, ten grains of Dover's powder at night, with a warm bath and glass of spirit, and a saline in the morning, will very frequently abort an impending attack. First Stage. — Small diaphoretic doses of tartar emetic ($\frac{1}{3}$ to $\frac{1}{2}$ gr. every four hours for an adult), or ipecacuanha; tincture of aconite, as directed under pleurisy; warm baths, or the cold sheet pack. Leeching over the sternum in some cases is good practise, followed by warm, moist applications, sinapisms, medicated inhalations of turpentine, conium, hot water, &c. Light liquid diet of beef tea, milk, &c.

Second Stage.—When expectoration is abundant, we may assist it by the administration of ipecacuanha, squill, senega, etc. Emetics are useful in children. If there is spasm, ether, henbane, belladonna, etc. In the feeble and aged, stimulating expectorants, such as the carbonate of ammonia and senega, may be given with plenty of beef tea and stimulants. In Chronic Bronchitis, when the secretion is very tenacious, *alkalies* are very serviceable, especially the muriate of ammonia or liq. potassæ.

Blisters, counter irritation by croton oil, etc., change of climate, inhalations of iodine, chlorine, etc. *Opium must be used with very great caution*, and although it frequently aids the secretion in the first stage, and eases the cough, it in the second stage, *especially in the aged, debilitated, and children, tends to promote the accumulation of the morbid products in the tubes.*

CAPILLARY BRONCHITIS, PERIPNEUMONIA NOTHA.

Definition.—Is a simple inflammation of the smaller (not smallest or capillary bronchial tubes, which, with the air cells, are affected in pneumonia) bronchial tubes, not differing in its anatomical character from that just considered, save in its seat. *It is a most grave affection*, mostly proving fatal in young children and the aged and debili-

tated. The symytoms are similar to those of ordinary bronchitis, save in their greater severity. The inflammatory fever is more intense, the temperature frequently reaching 103°. There is orthopnœa ; the respirations are increased to perhaps 50 per minute; the cough is difficult, and ineffectual ; expectoration small and tenacious, and, although specifically heavier than water, floats therein by clinging to the frothy mucous from the larger tubes. The face becomes swollen, the lips blue, and general cyanosis results from non-aeration of the blood.

In the adult recovery generally takes place ; but in children the face becomes livid, the air in the lungs becomes rarified, causing the jugular and epigastric regions and lower ribs to be drawn inwards, and death takes place from asphyxia. In the aged and feeble, fever of an adynamic type accompanying the inflammation, leads to general paralysis, coma, and death.

The average duration of this disease in fatal cases is from three to five days.

Treatment.—The tendency to death from imperfect oxygenation, due to the morbid state of, and accumulation of diseased products within the tubes, demonstrates the necessity of endeavouring to support the strength of our patient until the danger is past. We must, therefore, from the first, administer strong beef tea, eggs, milk, and wine. *Carbonate of ammonia* and iodide of potassium, *inhalation of oxygen*,, dry cupping, and *stimulants*, are the chief remedies at our disposal. *In children, emetics* are useful to empty the loaded tubes, but they must not be carried so far as to produce exhaustion.

EPIDEMIC BRONCHITIS or INFLUENZA.

This affection, due to some atmospheric peculiarity, is essentially a specific fever, with general catarrh of the respiratory and digestive tracts. It is characterized by chills, coryza, pyrexia, marked lassitude, anorexia, debility, and general symptoms of a bronchial catarrh. The frontal and maxillary sinuses, lachrymal ducts, and eusta chian tubes, participate in the inflammation. These symptoms continue for three to six days, and end by diaphoresis or diarrhœa.

Treatment.—In the strong and vigorous, purges and diaphoretics and opium; in the aged and feeble, tonics and support.

BRONCHITIS, WITH FIBRINOUS EXUDATION

May be primary or secondary, from extension from the trachea or larynx, with which it is identical in pathology and morbid anatomy.

It may be chronic, thus differing from the always acute laryngeal affection. *It is generally limited in extent.* The symptoms are much the same as in ordinary bronchitis, save in the expectoration of *characteristic moulds of the bronchial tubes.*

PERTUSSIS—WHOOPING COUGH.

Etiology.—This infectious and specific disease has been by some classed as a fever, and by others as belonging to the neuroses, or a peculiar variety of bronchitis. It is most common in children, especially after the second year, and occurs, as a rule, but once in a lifetime.

Pathology and Morbid Anatomy.—All the anatomical lesions found after death, excepting bronchitis, are due to complications. Morbid affections of the pneumogastric nerve, the brain, the bronchial mucous membrane, and the lungs, have all in turn been considered causes of this affection. *Niemeyer* views it as a catarrh of the mucous membrane, combined with intense hyperœsthesia of the air passages, which fully accounts for the ready occurrence and great violence of the reflex paroxysms of coughing.

Symptoms and Course — First Stage. — *Invasion* or *catarrh.* The primary symptoms *are those of ordinary catarrh or bronchitis*, except that in the majority of cases the cough is more violent and paroxysmal than in the ordinary affection. There is more or less fever for the first few days. The duration of this stage is from two or three days to as many weeks. Gradually the cough becomes more obstinate and persistent, and the Second Stage, or that of *development* is entered upon, which is characterised by the peculiar long-drawn, piping inspira-

tion, followed by a series of short spasmodic expiratory coughs, succeeded in turn by the *pathognomonic crowing, long-drawn inspiratory whoop.*

These paroxysms, depending on reflex spasmodic contraction of the glottis, usually last five or fifteen minutes, and terminate by an *abundant expectoration* of a tenacious viscid, transparent secretion. No air is heard entering the lungs during the fit; and the blood accumulating in the right side of the heart, causes acute cyanosis, evidenced by blueness of the lips, swollen and pallid face, and in some cases by hemorrhages from the mouth, nose, or even ears.

The abdominal muscles pressing the stomach against the fixed diaphragm, empties it during the paroxysm, and involuntary evacuations of urine and fœces frequently occur. Headache, convulsions, and apoplexy, the result of the venous congestion, and emphysema and hernia from violent expiration and muscular action, are frequently caused by this affection; whilst *pneumonia, atalectasis, capillary bronchitis*, and *infantile remittent fever may complicate it.* The fever disappears in the second stage. Death is rare from uncomplicated whooping cough.

The appetite is usually very good. The Third Stage, *or that of decline*, varies in duration from six weeks to as many months.

Treatment.—This being essentially a specific affection, abortive measures have proved ineffectual. We must therefore palliate the paroxysmal cough, and assist the expectoration of the viscid tenacious secretion by antispasmodics, small doses of opiates, expectorants, and an occasional emetic. The diet must be light and nutritious.

Trousseau recommends *cupri sulph.* as an emetic.

Alum gr. i. to gr. vi. every four hours, according to the age of the patient, by *Golding Bird. Belladonna*, gr. $\frac{1}{10}$ for one year, $\frac{1}{8}$ for four years, every twenty-four hours, and cautiously increased until the paroxysms decrease in severity and frequency (*Trousseau*).

Brown Seguard says it may be cured by keeping the child under the influence of *atropine*, in a state of delirium for three days! *Bromides of potassium* and *ammonium, strong coffee*, &c., &c., are among the numerous remedies advocated in the treatment of this tiresome and disagreeable affection.

INFLAMMATION OF THE LUNGS.

At the present time three diffrent forms of Pneumonia are recognized, and I have placed their peculiar pathological and anatomical characteristics before the student, in order that he may be enabled perfectly to appreciate the symptoms, physical signs, prognosis, and treatment of each variety.

Etiology.—Sudden chilling when the body is overheated, irritants, and wounds, are the chief causes of primary pneumonia. Typhus, measles, erysipelas, acute rheumatism, albuminuria, and extension of the inflammation from the bronchial tubes, those of secondary disease. As the mucous membrane of the bronchial tubes differs anatomically from that of the air cells, in having ciliated epithelium and mucous follicles, the air cells having no follicles and squamous epithelium, pneumonia does not produce bronchitis, and vice versa. It is rare under five years of age. Males are attacked more frequently than females. The lower lobe of the right lung is the part most usually affected in ordinary pneumonia.

I. CROUPOUS PNEUMONIA.

Is also termed Lobar, as it usually affects an extensive portion of the lung, consists in the exudation into the air cells of a rapidly coagulable albumino-fibrinous material, identical with that of membranous laryngitis, or true croup.

Pathology and Morbid Anatomy—First Stage.—Engorgement.—The lung is exceedingly vascular, of a purplish red colour, resembling the spleen. It is increased in weight and specific gravity, its elasticity is diminished, and it pits on pressure. The capillaries are distended with blood, and a thin, viscid, tenacious fluid bathes the walls of the vesicles. The inflamed tissue does not crackle much on being pressed between the fingers, and on section a reddish-brown, frothy liquid exudes.

Second Stage—Red Hepatization.—The lung, distended to the utmost, and marked by the ribs is of a reddish brown colour, resembling the liver, does not crepitate, and is friable under pressure, and sinks in water. The cut

surface presents a granular appearance, on account of the exuded liquor sanguinis and migrated blood corpuscles in the air cells having coagulated and become solid.

Third Stage—Grey Hepatization.—The lung is of a yellowish-white colour, owing to the capillaries being pressed upon by the over distended air cells, and also by the fatty degeneration and puriform character of their contents. Its tissue is soft and friable, and a puriform liquid exudes from its cut surface. The exudation increased by additional emigration of leucocytes, and proliferation of the epithelium, now consists of young cell forms and granular elements, in various stages of fatty metamorphosis.

Terminations—Resolution.—By far the most frequent sequence of red hepatization, and in my opinion ought to be considered as the ordinary third stage, and the grey hepatization as a more rare termination. The coagulated material liquefies and undergoes fatty degeneration, and is absorbed; the circulation is gradually restored, and complete recovery takes place.

In Abscess.—Extremely rare, except as the result of pyœmia. The cell production is so abundant as to cause the lung tissue to break down and slough from pressure. A thin, vascularized layer of lymph, and thickened, infiltrated connective tissue, encapsules this circumscribed collection of pus.

In Gangrene.—This results either from the formation of coagula in the pulmonary or bronchial arteries, from contact with some putrid substance in the lung, or from the virulence of the inflammatory process.

It consists in the death, disintegration, and chemical decomposition of a small (circumscribed) or large (diffuse) portion of the lung substance. If *diffuse*, death invariably results; but if *circumscribed*, the gangrenous portion may be expectorated, and the cavity formed contract and cicatrize.

Physical Signs—First Stage.—Perhaps comparative dulness on percussion; fine crepitation, heard during inspiration only.

Second Stage.—Dulness on percussion, increased vocal fremitus, bronchophony, bronchial respiration. The costal movements are diminished on the affected, and we have peurile breathing on the opposite side.

Third Stage.—The addition to those of the second stage of submucous bronchial râles.

Resolution.—*Crepitatio redux*, then broncho-vesicular and gradual return of the normal respiratory murmur.

Abscess.—When its contents are evacuated through the bronchial tubes, and a cavity is left, we may have cavernous respiration, pectoriloquy, cracked-pot sound, metallic tinkling, or even splashing on succussion.

Gangrene.—Depend upon the amount of tissue destroyed, and the size of the cavities left on the evacuation of the ichorous debris. We shall at first have moist râles, and afterwards signs of a cavity.

Symptoms and Course—First Stage.—*A well marked single and severe rigor*, after which there is a *rapid rise* in the *temperature*, so that in six hours after it may be 102°, in twelve hours 104°. Pain of a dull character referred to the nipple region; if it be acute, however, pleuritis may be assumed to be present. Cough, accompanied by the expectoration of, at first scanty, transparent, viscid, and tenacious sputa, becoming rusty coloured from the intimate admixture of blood.

There is thirst, furred, creamy tongue, constipation, scanty, high coloured urine, with diminished chlorides, and excess of urea, anorexia, &c., evidencing the pyrexial state. The *pulse full and hard*, averages from 100 to 120. The *respirations are increased* to forty or more per minute. *Wunderlich* considers all cases severe when the temperature is more than 104°, the pulse than 120, and the respirations exceed forty per minute. A sudden increase of temperature denotes the invasion of a new lobe, or some intercurrent affection. The *fever* is *remittent* in character. There is circumscribed redness of one or both cheeks. The *duration* of this stage is from one to two days.

Second Stage.—There is a *gradual diminution in severity* of all the symptoms just noticed, the pain lessens, the cough is not so troublesome, and is followed by easy expectoration of a sputa becoming less rusty and more yellow in appearance, the pulse becomes softer, smaller, and less frequent. *Herpes* frequently break out on the lips and nose, and their appearance is considered of favorable import.

Albuminuria is present in a large proportion of cases,

being due to the implication of the kidney in the general congestion, and is an unfavorable sign.—(Dr. Parkes.) On or about the seventh day, crisis takes place, evidenced by profuse sweating, passage of large quantities of lithates or fœces, great prostration, and a rapid fall in the temperature. The *respirations continue abnormally frequent for some time*, owing to the lung being still filled with exudation products.

Resolution is evidenced by *progressive improvement*, and gradual disappearance of all the morbid symptoms; and the lung is sound in most cases at the end of the second or commencement of the third week.

Albuminuria during this period is supposed by *Begbie* to be due to the excretion of the absorbed exudation matter by the kidney.

Third Stage.—The fever and symptoms do not decrease but are prone to assume a typhoid form. The pulse becomes frequent and feeble, the expectoration purulent, the strength fails, and death from asthenia ensues.

Pneumonia is particularly fatal to the aged and drunkards. Double pneumonia is very dangerous. During the first and second stages, if death occurs, it usually proceeds from hyperæmia and collateral œdema of the sound portion of lung, or rapid extension of the inflammation. More rarely to cerebral congestion or clot in the right ventricle, the result of slackened circulation from pneumonia, obstruction, and the excess of fibrin in the blood. In drunkards and debilitated persons, from exhaustion.

Abscess is characterized by the sudden expectoration of large quantities of purulent matter, and the physical signs of a cavity.

Gangrene.—Great prostration, pulse small, weak, and frequent, eyes sunken, face pinched and ghastly, breath foul, sputa of a blackish grey colour, with an intolerable fœtid gangrenous odour. Rapid sinking of the patient, and death from septic poisoning or apnœa. If resolution should take place the symptoms gradually abate.

Treatment—First Stage.—*Bleeding*, if the patient be plethoric and vigorous, and the fever, pain, and constitutional disturbance be severe. If there are symptoms of hyperæmia and collateral œdema of the unaffected portion

of lung, evidenced by intense dyspnœa, lividity, coolness of skin, and rapid sinking of the patient; and lastly, if there be symptoms of cerebral congestion. In some cases small diaphoretic doses of *tartar emetic* ($\frac{1}{4}$ gr.), with *calomel* (1 gr.) every four hours until the gums are touched or the inflammation modified. *Tartar emetic* with *brandy* (Todd).

Aconite in small frequently repeated doses, as recommended in the other inflammations, is extremely potent in reducing the violence of the heart's action and the inflammatory state. *Digitalis*, *opium*, *alkalies*, *veratria*, the *application of cold compresses* to the chest, have all been advised for the same end.

Second Stage.—Any of the former remedies may be chosen according to the severity of the case and strength of the patient. To hasten the absorption of the exudation, *mercury* and *iodine* have been recommended.

Stimulants must be given if there be a tendency to death by asthenia or clot in the heart, or if delirium be present. *In the aged and drunkards*, combined with *quinine*, it is essentially necessary.

I have enumerated most of the agents employed in the treatment of this common and dangerous affection, in order that the student may be acquainted with them; but I would strongly impress upon him the necessity in this as in all other diseases, to appreciate the peculiarities of each separate case in its individuality, and not treat each by the same general rule.

Pneumonia has a tendency to end by crisis; therefore do not employ any severe remedies to prevent this. Perfect quiet in bed, and a fresh, moist, and warm atmosphere. Cover the chest with a large jacket poultice of linseed meal, with or without mustard; half-ounce doses of liq. am. acet., combined with half a drachm of sp. œth. nitr., to aid the lungs by increasing the secretion of the skin and kidneys. A saline laxative; small doses of opium, or hypodermic injections of morphia to *relieve pain*. Light diet of beef tea, milk, eggs, &c. If the patient be aged or debilitated from whatever cause, or if typhoid symptoms appear, or *should gangrene occur*, strong beef tea and stimulants, with ammonia and bark, should be freely administered. *Stimulants are best given just before and over the crisis.*

II. CATARRHAL PNEUMONIA.

This form of pneumonia always succeeeds bronchitis, and confined to small portions of the lung, is *also termed Lobular, or Broncho Pneumonia.*

It is often a complication of measles and whooping cough, and occurs in atalectic lungs. The air cells become filled with proliferated alveolar epithelium, in addition to that which has been inhaled from the smaller bronchi.

This may undergo fatty metamorphosis, and be expectorated or absorbed, with complete recovery; or the retained inflammatory products give rise to local increase in the connective tissue and consolidation, which may undergo caseous degeneration, soften, and a phthisis result.

Symptoms.—If in a child suffering from measles, bronchitis, or whooping cough, the temperature should rise from the ordinary 101° or 102° to 104° or 105°, with frequent pulse, flushed face, painful cough, and evidences of obstructed respiration, we must fear catarrhal pneumonia; and if we have physical evidences of consolidation in different portions of the lung, our diagnosis is rendered certain.

Treatment.—That of ordinary pneumonia.

III. CHRONIC INTERSTITIAL PNEUMONIA.

It is one of the elementary constituents of the pulmonary lesions included in the term "Phthisis." It is one of the terminations or complications of croupous and catarrhal pneumonia, especially the latter; and is often the result of the irritation of minute foreign particles, or chronic inflammatory deposits in the lung. The right lung is more frequently affected than the left.

Pathology and Morbid Anatomy.—As a primary disease, consisting in a hyperplasia of the connective tissue, it is very rare *(Cirrhosis.)* The lung is diminished in size, firm, and almost cartilaginous in consistence, and varies in colour with the amount of pigmentation. On section the diseased portion will be found riddled with bronchiectatic cavities, containing stinking secretion, or in a more or less ulcerated condition.

Pathologically the disease consists in the growth of an adenoid tissue (a delicate fibro-cellular tissue), within the wall of the air cell, and in the interlobular tissue, with a proliferation of the intercellular connective tissue rapidly becoming fibrous, and thus occupying less space, causing contraction. If the result of a croupous pneumonia, the exudation, instead of undergoing fatty degeneration and absorption, becomes vascular and fibroid.

Physical Signs.—Flattening or retraction of the chest wall, dulness, and the other signs of consolidation, afterwards being combined with those denoting presence of cavities (dilated bronchi.) There is puerile respiration on the opposite side.

Symptoms.—Gradual loss of flesh, breathlessness on exertion, constant cough, with at one time little or no expectoration, at others of enormous quantities of putrid muco-purulent secretion from a bronchiectatic cavity. In the early stages hœmophysis from the over worked and congested sound lung; later on, due to the ulceration or sloughing of the walls of a cavity, or to the arterioles thereon having lost their support on one side, bulging out, and after a time rupturing. When the disease is extensive and advanced, the right side of the heart being unable to overcome the obstruction, dilates, cyanosis appears, followed by general dropsy, and, at length, death.

Treatment.—Maintain the strength by attention to *hygienic* and *dietetic* measures. It is irremediable, and will sooner or later end fatally. We must *palliate* the the various symptoms, especially the cough, and endeavour to modify the expectoration. *Inhalations* of turpentine, chlorine, carbolic acid, and iodine, have each been in many cases followed by marked improvement.

Contrast between the Symptoms of

PNEUMONIA AND PLEURISY.

1. Single, well marked, severe rigor.	1. Chilly sensations, or repeated rigors.
2. Dull pain (usually in right side).	2. Acute sharp lancinating (usually in left side).
3. Temperature 103° or 104°, and frequently higher.	3. Temp. rarely exceeds 102°.
4. Expectoration, first mucous, then rusty, and at length muco-purulent.	4. No expectoration.
5. Fine crepitation, then physical signs of consolidation.	5. Friction sound, then physical signs of effusion.
6. No alteration in signs by change of position.	6. Seat of dulness varies in seat according to the position of patient.
7. No displacement of the heart or liver.	7. Displacement of the heart and diaphragm, with the abdominal viscera in direct proportion to the amount of effusion.
8. Respirations greatly increased in frequency.	8. Only slightly increased.
9. Tends to end by crisis.	9. Terminates slowly.

EMPHYSEMA.

DEFINITION.—An abnormal amount of air in the pulmonary vesicles, causing their dilatation and morbid enlargement (VESICULAR), or an escape of air into subpleural and interstial connective tissue of the lungs (INTERSTITIAL). When several air cells having broken down into one (ATROPHIC).

ETIOLOGY.—There have been numerous theories advanced to account for the production of this disease, the chief being the *Inspiratory Theory*, "that a portion of the lung having lost its function by consolidation or otherwise, the remainder vicariously having too much work to do, dilates; also if a portion of lung collapses, there is a space left in the thorax which must be filled, and the lung dilates to do so."

The *Expiratory Theory*, "that the air entering by the greater force of inspiration through tubes narrowed perhaps by bronchitis, is unable to get out by expiration, and

there is a resulting stasis in the air cells. If this inspiratory vesicular inflation be kept up for any great length of time, the continuous strain and expansion produce structural changes in the vesicular walls, they atrophy, grow thin, become perforated, several vessels blend into one larger cyst, and then emphysema really commences. Great muscular straining and blowing on wind instruments will also give rise to the same process. Freund considers a rigid distension of the thorax to be very frequently the primary disease, and emphysema the secondary result.

Pathology and Morbid Anatomy.—On opening the thorax we find that there is incomplete retraction of the lungs, which frequently bulge forwards; the lung is dry and anœmic, as the continued pressure on the walls of the air cells causes tension, and, at length, obliteration of their capillaries. This pressure upon, and obliteration of the pulmonary capillaries also causes dilatation of the right heart, with general congestion of the venous system. Evidences of bronchitis are almost invariably present.

Symptoms.—Their development is slow, and varies in severity according to the amount of disease. There is laborious breathing; inspiration is shortened, and expiration prolonged; sputa frothy, like soap suds; dyspnœa is abiding, varies but little, and has no wheeze. The accumulation of blood within the right ventricle causes at first its hypertrophy, but from the general venous congestion, want of oxygenation, and consequent malnutrition, the patient gradually weakens, and the heart structure sharing in the general emaciation thins and dilates. The cyanosis may become extreme, pulsation of the jugular veins evidencing incompetence of the auricular ventricular valves, and if the patient be not carried off by some intercurrent affection, general dropsy will terminate the case.

Physical Signs.—The upper and middle third of the chest enlarged, and the ribs and sternum are raised together in inspiration, the heart is pushed down; intense resonance on percussion. Sibilant and sonorous râles when present evidence the co-existence of bronchitis. The prolonged expiration of emphysema is intense, but natural; that of solidified lung in phthisis is comparatively high in pitch, and tubular in quality.

Treatment.—*Can only be palliative*, as the disease is incurable. Mild, warm atmosphere, plenty of warm clothing, and light nutritious diet. We must treat the complicating or causal affections, *e.g.*, whooping cough, bronchitis, asthma, &c.—

ASTHMA.

Definition.—A neuropathic affection, characterised by paroxysmal attacks of urgent dyspnœa, depending upon a spasmodic contraction of the smaller bronchial tubes.

Etiology and Pathology.—Varieties have been described depending upon some supposed special causation; *e. g.*, spasmodic, hay, hysterica, dyspeptic, cardiac, and emphysematous; but whether it is due to some local hyperœsthesia or reflex nervous action, it is impossible to decide. It may occur from infancy to old age, and is frequently hereditary. Asthmatics live to a good old age. Some consider it a constitutional disease, connected with the rheumatic or gouty diathesis. The urine succeeding the fit is remarkable for the diminution of urea and chloride of sodium, which would imply a considerable arrest, either of formation or elimination—(Ringer, Parkes) —or to the starvation generally enforced at the time—(Hyde Salter.)

Symptoms and Course.—It is essentially a paroxysmal affection, and may commence without warning, but is often preceded by drowsiness and mental excitement. Dr. Salter mentions itching of the chin and chest as a common symptom of an approaching attack. The paroxysm generally takes place at about three to five o'clock in the morning. The patient feels unable to get the air into his lungs. Every muscle of inspiration is brought into play, and frequently he rushes to the window, and imbibes the cold condensed air with some little relief. He rests his elbows upon some solid support to enable the pectoral and other extraordinary muscles to aid him in his efforts. The sterno-cleido-mastoid muscles are rigid and prominent, and the chest is fixed in a condition of extreme inspiration. The respirations are not increased. Inspiration is performed with a spasmodic effort, and *expiration* is prolonged, and *accompanied by a wheezing*

sound. The face is pallid and cyanotic, and the countenance expressive of distress. Owing to the obstruction to the circulation, there is accumulation of blood in the right side of the heart. The pulse is small and feeble, and the surface of the body cool. Suddenly or gradually, after a variable period, the spasms lessen, and the air enters the lung freely. There is now an increased secretion from the bronchial tubes, evidenced by the expectoration of a semi-transparent, jelly-like sputa.

TREATMENT.—Remove the cause. *Pure, dry, warm, compressed air*; *opium, smoking stramonium or tobacco, asa-fœtida, cannabis indica*; nauseating remedies, *e. g., Tartar emetic, lobelia inflata, conium, chloroform, strong coffee, fumes of burning nitre paper, hot spirit and water*, have all been recommended either to arrest, shorten, or mitigate the paroxysm. *Quinine*, preparations of *iron, zinc*, and *silver*, and other *nervine tonics* are in repute for the prevention of the paroxysms, and the radical cure of the disease.

PULMONARY HŒMORRHAGE.

Hœmorrhage may take place into the bronchial tubes (bronchorrhagia) or into the cells or interstitial tissue of the lung itself (pneumorrhagia.)

It is most generally thought to arise from an embolus blocking up one of the pulmonary arterioles, and either from collateral hyperæmia and consequent rupture, or from increased pressure in the blocked up artery itself, hœmorrhage results. In other cases it is due to the branches of the pulmonary or bronchial arteries situated on the wall of a cavity, being ulcerated into; or losing its support on one side it bulges out, becomes aneurismal, and at length gives way. The *symptoms* are those of interference with the function of respiration, and loss of blood, and in most cases there will be external evidences in the shape of an hæmoptysis. Coagulation of the blood may take place in the tubes, and thus cause atalectasis.

CANCER OF THE LUNG.

Very rare. Either the deposit is infiltrated replacing pulmonary structure, or exterior to the pulmonary organs in the mediastinum, bronchial glands, or the pleura.

It is usually secondary. The *encephaloid* or medullary *variety is the most common*, schirrus being very rare, and colloid practically unknown; in people over fifty. There are usually signs of cancer elsewhere, with the peculiar cachexia, combined with evidences of lung implication, and perhaps the expectoration of currant-jelly-like sputa.

PULMONARY CONSUMPTION.

I must confess I approach the task of condensing and elucidating this subject with great diffidence, because there exist among the writers and pathologists of the present day wide differences of opinion regarding even the origin and essential elements constituting this disease.

Definition.—By pulmonary consumption we must now understand a progressive wasting of the body, coincident with, or depending upon, some structural degenerative changes of the lungs.

Morbid Anatomy.—In the early stages we may find the *air cells* filled with epithelial cells in every stage of development, or, more rarely, a fibrinous exudation entangling young cells.

The alveolar walls are almost constantly found more or less thickened by a tissue composed of small cells, separated from one another by fine fibrils of a plastic material (adenoid tissue). *The interstitial areolar tissue*, by active cell proliferation, is increased in quantity and altered in structure. If the cellular elements are in excess, the part, on section, will present a dull looking homogeneous gelatinous appearance, and at a later period will be found to have softened into a yellow looking, structureless, granular debris, soon breaking down into irregularly shaped cavities. When the alveolar walls are occupied by more or less adenoid tissue, and there is increased growth of the connective tissue, we will find that the affected part, instead of being quite soft and friable, is hard and indurated. This induration is in direct proportion to the amount of fibrillation of the new growth. Soon this will present an appearance similar to the one just described, for all deposits in phthisis are characterised at first by progressive condensation, and subsequently by cheesy degeneration and the formation of cavities. In nearly

every case on section we will find combined with these deposits, or, in rare cases existing without them, firm, well-defined, spheroidal, semi-transparent nodules of a greyish-white colour, varying in size from a pin's head to a pea, situated in the walls of the aircells, the intercellular tissue, or the bronchial mucous membrane.

Microscopically these nodules are composed of lymphoid or adenoid tissue *(grey miliary tubercle)*; in others a similar lymphoid growth is found in a like situation; but the alveoli being filled with peculiar large, branched, multinucleated, and other cells, more or less undergoing granular degeneration, they have a yellow appearance (*yellow tubercle*).

Pathology.—I have neither time or inclination to confound the student with idle and exploded theory, but will endeavour to the best of my ability to glean from amongst the latest works on the subject the results of practical investigation, supply them to him in a concentrated form, and, having done so, draw our conclusions. *Laennec* and his disciples regarded as tubercle all growths which at first indurated, ultimately underwent caseous degeneration. All pneumonic deposits, at first grey, then yellow, indurated or crude, afterwards undergoing cheesy metamorphosis, *were* tubercular.

Virchow, however, has established the fact that numerous formations, having nothing in common, undergo caseation *and that therefore all pneumonic cosolidations are not tubercular*. *Niemeyer* contends that nearly all pneumonic deposits are the products of inflammatory action, and not the result of neoplasm.

We said that the most frequently found lesion in cases of pulmonary consumption consisted in an accumulation of cells within the pulmonary alveoli, exactly similar to that which takes place in catarrhal pneumonia, and in all cases some thickening of the alveolar walls by a small celled lymphoid tissue. *This implication of the lung tissue* forms the one great distinction between the morbid histology of pneumonia and that of the lesions constituting phthisis. The perpetual accumulation of young cells in the air vesicles, due to the proliferation of the alveolar epithelium, and inhalation of cells from the smaller bronchi, on account of their incomplete development,

mutual injury from crowding and deprivation of nutrition, from pressure on the capillaries, tend to perish and break down. The thickening of the alveolar walls, and the increase of the connective tissue will be found to be cellular or fibroid accordingly as the inflammatory process be acute or chronic in character, and it will simplify matters by at once stating the fact that *any of the various forms of pneumonia already described, which cause consolidation of the lung, may undergo destruction, and thus form the anatomical basis for pulmonary consumption.* "When pneumonia ends in resolution, the inflammatory product undergoes fatty metamorphosis, then liquifies, and is absorbed. When the disease is followed by caseous infiltration, the fatty metamorphosis is incomplete. The infiltration dries up, the cells are atrophied, they lose their rounded form, and shrink, through loss of their water, into irregularly-shaped clots."—(Virchow.) Now as to the grey nodules which we have described as co-existing with these inflammatory deposits, or regularly distributed through the lung, and which are termed miliary tubercular growths. *Tubercle is a growth consisting of lymphoid tissue, usually associated with large multinucleated masses of protoplasm incapable of further development, and exhibiting a tendency to degeneration, involving in its destruction that of the surrounding tissue.* One class of pathologists hold *that tubercle is an exudation from the blood, essentially morbid in character*, in which the vitality is so lowered that it tends to disintegration, and to produce the lowest kind of organic forms, *i. e.*, molecules, granules, and nuclei—(Hughes Bennett.) Another, *that it consists in merely a retrograde metamorphosis* of pre-existing structures, tissue elements, or morbid growths —(Williams, Henle, and others.)

Virchow considers tubercle to be a new growth, due to the proliferation of connective tissue cells, and leading to an exudation, which is poured out during what he termed a tuberculous inflammation.

Professor *Burdon Sanderson* and others, by experimental investigations, consisting in the inoculation of various chronic inflammatory products (caseous or not) produced in various organs miliary formations identical with tubercle, presenting a special tendency to become caseous at their centres. The general or local distribution varied

as the lymph or blood streams were the means of carrying the infective material We have therefore certain facts to guide us in this difficult subject. We now know that all caseous deposits found in the lungs are not tubercle; that most of them are the products of ordinary inflammations occurring in persons of a delicate constitution; that there is now only one primary form of tubercle recognised, namely, the grey miliary; that inoculation of a chronic inflammatory deposit produces it; that many learned physicians and pathologists consider *tubercle to be a secondary resorption disease* — (Niemeyer;) — and lastly, others, *that it is the local expression of a special constitutional affection, termed the "tuberculous cachexia"*—(Flint.)

Now whether tubercle is deposited as such, or as an exudation resulting from the absorption of chronic inflammatory deposits, is still sub judice.

The question at issue is, Is tubercle a primary or secondary growth?

Many clinical observers state that there are signs of a constitutional disease before there are the local manifestations of inflammation, and in my humble opinion, we are inclined to attach too much importance to theory based upon histological and pathological researches, to the exclusion of sound clinical observation.

It is peculiar that lymphoid tissue, of which tubercle is composed, has been discovered in all the situations in which that growth is usually found, even around the smaller capillaries as lymphatic sheaths. It may, however, be accounted for by *simply supposing it to be an excessive growth of adenoid tissue* (the lowest formed tissue found in the body) most commonly in that part of great delicacy of structure, and most exposed to atmospheric influences, and other mechanical irritations.

This growth (grey miliary tubercle) by inducing in the first place an excessive nutrition of the alveolar epithelium, gives rise to the presence in the alveoli of the peculiar large multinucleated branched cells, and to a general cell proliferation of the epithelium, completely blocking up the air cells, constituting, in fact, a broncho-pneumonia. After a time, these deposits, on account of the mutual pressure of the cellular elements, and partly by mechanical obstruction to the circulation, soften and break down

with the formation of cavities. Growths of this tissue in the alveolar walls, in the intercellular tissue, or in the mucous membrane of the capillary bronchi, may so generally and rapidly increase as to cause great constitutional disturbance, and alarming dyspnœa from mechanical pressure. The collateral hyperæmia caused by this local obstruction, may also tend to the production of inflammatory exudations. In a third class of cases, a primary catarrhal, or, more rarely, a croupous or interstitial pneumonia, either by direct irritation, by reflex action, or, more probably, by infection, gives rise to a more or less abundant and disseminated growth of tubercle.

Etiology.—All persons do not possess a phthisical disposition. There are, however, certain influences which weaken the constitution, and induce a state of the system predisposing to the development of low inflammatory affections, and the growth of tubercle.

The so-called *scrofulous diathesis*, which is characterised by general constitutional debility, accompanied by increased irritability; marked tendency to profuse cell formation, especially of the lymphatic gland tissue, and the hair and nails; peculiar albuminous state of the blood, and tendency to the infiltration of organs with albuminous material, may be termed the type of the *phthisical habit*.

Hereditary constitutional feebleness may proceed from other exhausting maladies of the parent instead of consumption, *e. g.*, syphilis, diabetes, cancer, &c., besides the influence of imprudent marriages.

Other debilitating causes render the system prone to the development of tubercle; *e. g.*, want of sanitary attention, moist, damp, changeable atmosphere, low situation, insufficient or unsuitable food, exposure to cold, &c., and anything producing fluxionary hyperæmia and bronchial catarrh; *e. g.*, dusty atmosphere, due to stone, knife, or file grinding, &c., &c. Then again, according to the infective theory, next to the products of pneumonia, the exudation of pleurisy and pericarditis, disease of the bronchial and mesenteric glands, &c., most frequently give rise to tuberculosis. It is most common between the twentieth and thirtieth years.

Persons with cardiac disease are very obnoxious to phthisis. Whether dependent or not upon the engorged

state of the lungs, is a point not yet understood, but which I consider to be of great significance.

Physical Signs—Incipient Stage.—In which the growth of tubercle gradually gives rise to a catarrhal pneumonia. Pure tuberculosis never causes consolidation sufficient to render percussion dull—(Niemeyer.) Feeble respiration, rough, wavy inspiration, prolonged expiration, succeeded by broncho-vesicular breathing, fine crepitation if pneumonia, sibilus if capillary bronchitis be present.

Stage of Deposition.—Here there is more or less shrinking of the apex, with subclavicular flattening and immobility. The muscles are thinned and irritable, rising up when struck with the finger. Dulness, increased vocal resonance and fremitus, bronchial breathing and bronchophony, squeaking râles, and, in fact, all the ordinary signs of consolidation.

Stage of Softening and Formation of Cavities.—Those of a cavity filled with softened matter and fluid. Bubbling rattles, at first small, afterwards large, then amphoric voice, cavernous respiration, metallic tinkling, pectoriloquy, &c. &c.

Symptoms and Course.—Perhaps with our present pathological knowledge, we cannot adopt a more convenient division of the varieties of pulmonary consumption than the following :—

1. Tubercular.
2. Pneumonitic.
3. Tuberculo-pneumonitic.

GENERAL SYMPTOMS OF PULMONARY CONSUMPTION.

These must vary according to the amount of interference with the functions of the lungs, disease in other organs, and the general health of the individual.

The general health is more or less deteriorated for some time before there are evidences of pulmonary affection, indicated by debility, loss of weight, capricious appetite, dislike to fats, dyspepsia, &c.

Increased temperature.—There can be no inflammatory process or widely distributed rapid cell growth in an organ

like the lung, without an elevation of temperature. This, therefore, is one of the earliest and most important signs of phthisis. *Niemeyer* states that the temperature of a pneumonitic phthisis is quite peculiar. In the morning perhaps normal, in the evening perhaps 102°. If tubercle be present, this difference is not so well marked.

Impaired nutrition evidenced by loss of weight, pallor and anœmia, and progressive emaciation.

Increasd frequency of respiration, the result of fever, loss of breathing surface, and pain.

Pains in the chest and shoulders, most frequently neuralgic, are sometimes due to a circumscribed pleurisy.

Cough, the earliest of the pulmonic symptoms, slight, dry, and hacking, is dependent upon the nervous irritability and hyperœsthesia of the air passages, the growth of tubercle, or a broncho-pneumonia.

Expectoration usually that of ordinary catarrh, but if we find on microscopical examination elastic fibres, phthisis is certainly present. Rusty sputa is diagnostic of a pneumonia, and in the third stage, when cavities are forming, it is muco-purulent and nummular.

Hœmoptysis may be a very early symptom, due to a natural weakness of the capillaries, or to a collateral hyperœmia. It is still sub judice as to whether hœmorrhage relieves the part, or by its presence sets up a pneumonia. In the later stages it is from ulceration into, or the bursting of a pulmonary arteriole running along in the wall of a cavity.

Heightened temperature, gradual loss of weight, with the physical signs of an apicial localised bronchitis or pneumonia, may be said to be diagnostic of incipient phthisis.

TUBERCULAR.

I shall under this heading briefly enumerate the symptoms which characterise the presence in the lungs of minute, semi-transparent, grey granulations, surrounded by more or less hyperæmia; in other words, of acute miliary tuberculosis.

Sypmtoms are those of a febrile affection, without any precursory catarrh, viz., frequent chills, hot skin, red tongue, nausea, anorexia, vomiting, and perhaps diarrhœa;

temperature 103° or 104°; pulse about 140; respirations 30 to 60, with rapid wasting from the first; tubercular laryngitis gives rise to distressing cough and aphonia. There may ensue signs of cerebral and intestinal tuberculosis, evidenced by symptoms of ulceration of the bowels and meningitis. Exhaustion is accelerated by profuse diarrhœa, and most frequently proves fatal in from twenty days to three months.

There may or there may not be unusual dulness on percussion; respiration is more or less morbid in character, as mentioned under *physical signs*, but in acute generally disseminated miliary tuberculosis of the lungs, uncomplicated by pneumonia or bronchitis. There can be no signs of broken down tissue and cavities.

PNEUMONITIC PHTHISIS.

An ordinary pneumonia, especially the apicial form, occurring in a person of a phthisical habit of body, may directly lapse into phthisis, evidenced by the physical signs of consolidation, mucous and gurgling râles, troublesome cough, and muco-purulent expectoration, containing elastic fibres, perhaps hœmoptysis, hectic fever, and rapid emaciation. Most commonly, however, this form begins very insiduously, with precursory catarrh, increased temperature, anorexia, &c., and loss of weight. The physical signs may be very slight at first, but after a time we find evidences of a localised catarrhal or croupous pneumonia, most frequently situated at the apex. These symptoms may entirely disappear, and the person regain his health during the summer months, but exposure to cold or the setting in of winter almost invariably restores them. Such alternations may go on for a number of years until little by little the lung becomes extensively diseased.

Any of the general symptoms may be present, and with the loss of weight, dyspnœa, and physical signs, &c., will be in direct proportion to the amount of pulmonary lesion.

The second stage, or that of caseation, and breaking down with the formation of cavities, is characterised by hectic fever, rapid loss of flesh, colliquative night sweats (perhaps

nature's attempt to ease the lungs by increasing the function of the skin), and diarrhœa; abundant muco-purulent, nummular expectoration, great debility, exhaustion, and death.

TUBERCULO-PNEUMONITIC.

Perhaps by far the commonest form of phthisis, whether the tuberculosis be the primary growth, or secondary to the pneumonic deposit. These cases are generally chronic, slight cough, increased temperature, gradual loss of weight, with physical evidences of tubercle, or a small, localised catarrhal pneumonia, being the signs most commonly met with in this variety. Slowly and silently, perhaps even year by year, as the deposits increase, as tubercle is deposited, giving rise to catarrhal pneumonia, with subsequent caseation, as other portions of the lung becomes implicated, do the symptoms and signs proceed, until these organs become diseased to an extent incompatible with life.

TREATMENT.

The tuberculous cachexia is irremovable, therefore we must endeavour to strengthen and invigorate the patient, and palliate the symptoms by every means in our power. All persons of a tuberculous habit of body need not necessarily die of pulmonary consumption; therefore by prophylactic measures, such as warm clothing, nutritious diet, codliver oil, moderate use of stimulants, exercise in the open air, a uniform dry atmosphere, in an elevated situation, mental recreation, etc., we must place them in a state of health and a position unfavourable to the growth of tubercle. When the disease is present we must endeavour to arrest its progress and effect a cure. *The temperature is the best indication of the amount of change* going on, and we may assume that in subduing it we reduce the disease. *Rest in bed*, stimulants, *digitalis*, *combined with quinine*, and milk diet, are the most successful antipyretics. *Codliver oil*, extract of malt, the hypophosphites and other *preparations of phosphorus*, preparations of *iron and iodine*, *inhalations* of chlorine, and other medicinal agents, are the most valuable therapeutic remedies at our

command to strengthen, stimulate, and relieve the patient. *Symptomatic treatment* is the most important part of the therapeusis of consumption. *The cough* must be allayed by suitable expectorants and sedatives; opium in some form or other is indispensable. *Hæmoptysis* is most easily relieved by the combination of acetate of lead and opium, *the night sweats* by dilute sulphuric acid, Dover's powder, oxide of zinc, and general antipyretic treatment. *The emaciation*, induced by the pyrexia, abundant muco-purulent expectoration, sweats, and diarrhœa, is, perhaps, impossible to arrest; but the use of emulsified codliver oil and other fatty and nutritious articles of diet are highly commendable.

In acute miliary tuberculosis the case is hopeless, and we can only palliate the symptoms and support the powers of life.

The following are the places most suitable for the winter residence of consumptives—*Torquay*, *Isle of Wight*, *Hastings*, *Mentone*, *Madeira*, *Algiers*, *Cairo*, *&c.*, where the atmosphere is warm and humid; but in other cases a uniform cold climate has been found to be the best.

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