brandy, two to eight drachms, with water or in a milk-punch or eggnog.

Remedial Treatment.—Various drugs, methods and serums have been advocated as possible specifics from time to time, particularly during the last few years. They have received the attention of the profession under circumstances to give them a thorough trial. While each of these has been found to possess virtue with limitations, not one of them has demonstrated sufficient specific action in tuberculosis to warrant its being depended upon as the soverign remedy in this distressing malady. The best treatment resolves itself into careful prescribing for the symptoms or special conditions as they arise, coupled with the best, dietetic and hygienic measures.

For the special symptoms the following remedies are variously indicated, the relative demand for their use seeming to be in the order mentioned:

Pre-Tubercular State.—General hectic condition, ill-defined and suspicious, but without well marked physical signs calls for *Bacillinum* or *Tuberculinum*.

The Totality of a case of tuberculosis seems most often covered by *Phosphorus*, *Arsenicum iod.*, *Stannum iod.*, *Ferrum phos.*, *Ferrum ars.*, *Iodine*, *Antimonium iod.*, *Kali carb.*, *Chininum ars.*

Fever.—Baptisia, *Ferrum phos.*, *Arsenicum iod.*, *Chininum ars.* *Aconite* is contra-indicated because of the liability to contract cold after its use.

Cough.—Stannum iod., *Antimonium iod.*, *Phosphorus*, *Sanguinaria*, *Rumex crisp.* Heroin $\frac{1}{12}$ to $\frac{1}{20}$ grain or Codeine $\frac{1}{4}$ to $\frac{1}{12}$ grain, in tablet or vehicle, may be used as a palliative if necessary.

Sweat.—Dilute Phosphoric acid, ten to twenty drops in four ounces of water and taken in divided doses during the day, is excellent. *Cinchona*, *Ferrum ars.*, *Pilocarpine* 2x. *Agaricine* 1x, one grain at bedtime. Atropine, $\frac{1}{200}$ grain hypodermatically at bedtime. A cup of weak chamomile tea drank upon retiring is often very efficacious as is also a cool sponge bath with water acidulated with vinegar.

Hæmoptysis.—Millifolium, *Hamamelis*, *Geranium mac.*, the latter in mother tincture, five drop doses. Absolute rest, cold liquid food, ice cap to the chest, heat to the feet. See chapter pulmonary hæmorrhage.

Pain.—If pleuritic, *Bryonia alba.*, *Aconite*, *Kali carb.*, *Squilla*. Exposure to the arc light is valuable to relieve the pain of the pleurisy. If myalgic—*Bryonia alb.*, *Actea rac.*, or Acetanalid five grains as a palliative. Paint the painful region with tincture of iodine or employ massage.

Laryngeal Inflammation. — Causticum, *Phosphorus*, *Drosera*, *Iodine*, *Spongia*, *Arsenicum iod.*

Intestinal Tract.—Papoid one to three grains or pepsin three to five grains, alone or with Bismuth subnitrate five to ten grains, taken after eating is excellent in indigestion or diarrhœa. A good pleasant formula is ℞. Bismuth subnitrate ʒiv, Essence of pepsin ʒvi. Mix. S. Take one dessertspoonful after eating. Burned brandy one or two drachms frequently repeated is of value. Opiates are contra-indicated. Persistent diarrhœa due to intestinal ulceration is benefited by colon flushing with hot boric acid or carbolized water or weak flaxseed tea. Of the remedies to be used are *Cuprum ars.*, *Cinchona*, *Ferrum ars.*, *Veratrum album*.

Special Suggestions.—Cod liver oil given in one drachm doses after each meal is excellent in its effect upon nutrition. It acts best in children and especially in bone and glandular tuberculosis. Gastric irritability or fever contra-indicate its use. A dessertspoonful of emulsion of mixed fats or thick cream is a good substitute.

Strychnine nitrate or sulphate $\frac{1}{60}$ to $\frac{1}{100}$ of a grain three times daily aids digestion and gives nerve and cardiac strength.

Ichthyol combined with glycerin in equal parts and given two drops after each meal, increasing one drop per dose daily, until twenty to thirty drops are given at a dose, gives good results in cavity cases but not when associated with enteric troubles.

Creosote or Guaiacol have been favorites for their effect in diminishing the cough and expectoration. They are given in one drop doses after eating and gradually increasing to eight or ten drops.

Inhalations of Guaiacol, Terebene, Creosote Benzoin, Iodine or Eucalyptus, one drachm to the pint of boiling water, are sometimes useful as antiseptics to the respiratory tract and sooth the cough.

Sunlight.—The patient stripped to the waist and exposed to the direct rays of the sun in a warm room will be benefited as to the cough, sweats, fever and local pains, but this should not be tried if there is much exhaustion.

Electricity.—The ozone inhalations from static electricity are claimed to stimulate and improve nutrition. The X-ray is of value as a diagnostic means. The X-ray and high frequency currents are as yet too much in their infancy to draw definite conclusion as to their action in

tuberculosis. They have benefited many cases, apparently by improving nutrition and general cell tone, thus inhibiting the disease. Upon the bacilli their effect is to cause rapid growth and over-development with great increase in numbers, leading to ultimate attenuation and extinction from overstimulation.

FIBROID PHTHISIS.

Definition. — Chronic Interstitial Pneumonia. Cirrhosis of the Lung. Consists in a gradual fibroid change in which fibroid tissue takes the place of normal or tuberculous lung tissue, with consequent contraction and induration.

Etiology.—Most cases supervene upon an arrested tuberculous process, though some are of simple pneumonic nature. In the cases of tuberculous origin, the fibroid change is more common in the apex, where it surrounds the cavity formed or invades areas of caseous degeneration, arresting the active process and producing shrinkage and hardening, changing the area, lobe or lung into a mass of tough, grayish, fibrous tissue. This retraction leaves the bronchi dilated and inelastic, forming bronchiectatic sacs.

Fibrous phthisis of non-tubercular origin may be the method of termination in unresolving lobar or broncho-pneumonia. In the first the walls of the alveoli and the fibrinous deposit filling them undergo fibroid change. In the catarrhal pneumonia the finer bronchi, the alveoli and their contents are affected by the fibrosis and the lobules become hard fibrous masses, with no trace of

normal lung tissue left. Fibroid change in the lung may take place after attacks of plastic pleurisy with extensive adhesions and resulting lung compression.

Symptoms.—This disease is essentially chronic, lasting ten to twenty years, during which the patient may enjoy a fair degree of health. The patient is usually thin, anæmic and suffers dyspnœa upon slight exertion, but with little or no serious constitutional disturbance, *i. e.*, fever sweat, etc. There is much cough with purulent expectoration, sometimes fœtid, from the existing bronchiectasis.

Physical Signs.—These are characteristic. The chest on the affected side is sunken, flat, retracted and immobile. The shoulder is drawn down and the spine bowed. When the right lung is affected the heart may be drawn by the retraction toward the affected side; when the left lung is the seat of trouble, the area of cardiac impulse is greatly increased. Percussion shows variation from flatness over apex or base, to amphoric or tympanic resonance over bronchiectic cavities. Auscultation gives cavernous breathing sounds at the upper part and feeble sounds mixed with rales at the base.

Treatment.—Is only for intercurrent affections or aggravation of the cough. The primary condition is not amenable to treatment. The patient should seek a mild climate and avoid exposure to inclement weather. The remedies and means of service are those usually required for chronic bronchitis, bronchiectasis and emphysema.

BRONCHO-PNEUMONIA.

Definition.—Known also as acute catarrhal pneumonia and lobular pneumonia.

Broncho-pneumonia is a catarrhal inflammation attacking the air cells in various lobules of the lungs. It is usually bilateral and is the result of an extension from a bronchitis of the smaller tubes and is therefore secondary in character.

Etiology.—This form of pneumonia is due to an invasion of the air cells by continuity of surface, obstruction of the bronchioles or inhalation of irritating secretion, of an inflammation previously existing in the bronchial tubes. It may supervene in some cases in atelectatic lung tissue and in some rare instances the same irritating influence that causes the bronchitis may excite the catarrhal pneumonia spontaneously. Age is a most important factor, a great majority of cases occurring in infancy and old age, particularly when the constitution is debilitated or enfeebled. It is especially liable to occur by extension during the course of the various infectious diseases, whooping cough, measles, influenza, diphtheria and variola. Bad hygiene, improper nourishment, in fact, any debilitating influence are potent predisposing causes. The inhalation of irritating substances, vapors, and various kinds of dust frequently excite an attack.

Pathology.—The morbid changes are confined to various groups of air cells (lobules) scattered throughout the lungs. These may be here and there or several that lie close together may be involved, thus giving a larger area of consolidation and simulating pneumonic fever (croupous

pneumonia) by the seeming involvement of a whole lobe. These consolidated areas appear as nodules varying in size from that of a pea to a hazelnut scattered throughout the affected lungs. They may lie near the surface of the lung in which case they appear as small round elevations, or may be more deeply seated through the tissue. They do not fill when the lung is inflated, they are not as tough as healthy lung tissue and break down easily upon pressure, they vary in color from red to bluish or they may in some instances be firm, dry and whitish as if they had undergone purulent infiltration. When cut these nodules exude a reddish fluid or a few drops of dark blood. The lung tissue surrounding the inflamed nodule will be found to be congested, œdematous or emphysematous. In broncho-pneumonia we recognize three stages to the pathological process, though these are not as well defined as in lobar pneumonia. The *first stage* is one of vascular engorgement, the alveolar epithelium is swollen and turgescent, with more or less desquamation and exudation. The air cells are filled with serous fluid rich in cellular elements, blood and epithelium. The *second stage* is that of complete consolidation. The air cells are full of exudate rich in pus cells, blood cells and desquamated epithelium. The *third stage* is the time when this deposit undergoes fatty degeneration and softening and is absorbed or expectorated, with a subsequent restoration of healthy epithelium. In unfavorable cases the deposit undergoes cheesy or purulent degeneration leading to gangrene, abscesses, interstitial pneumonia or becomes infected with the bacilli of tuberculosis. Atelectasis frequently results from the broncho-pneumonia of children and localized pleurisy is not uncommon when the inflamed lobules lie near the surface of the lung.

Symptoms.—The earlier symptoms are essentially those of the accompanying bronchitis of the smaller bronchial tubes. It is sometimes very difficult to determine just when lung tissue becomes involved. But when in the course of a bronchitis, whether simple or associated with some other disease, there is a gradual rise of temperature to 103° or 105° (but not the chill and sharp rise of fever seen in lobar pneumonia), respiration becomes difficult and increases in frequency to 50 or 80 per minute, with the complaint of soreness and pain; the pulse grows rapid (140—160), loses force and is compressible; the soft, loose cough of the preceding bronchitis becomes dry, tight and hard with pain and deep soreness; expectoration is slight or absent—if present it is muco-purulent or blood streaked (not "rusty" as in lobar pneumonia), then the invasion of a lobular pneumonia may be diagnosed. Fatal cases are due to the respiratory condition and symptoms. The respirations become frequent, labored, feeble and short, the extraordinary muscles of respiration with the alæ nasi are brought into play, the heart systole grows weaker with feeble pulse, clammy extremities and cyanosis, death occurring from heart failure due to respiratory failure. In favorable cases the temperature lowers, respiration becomes deeper and less frequent and labored, the cough loosens, expectoration becomes freer and the pulse grows stronger. In the chronic form the pulse and respiration gradually increase in frequency, the temperature is never as high as in the acute, rarely going above 102°. There is dyspnœa, loss of appetite, strength and flesh and a gen-

eral systemic failure. Death in such cases occurs from general exhaustion and cardiac failure, not from respiratory failure as in the acute form.

Physical Signs.—Percussion shows isolated points of consolidation, vocal fremitus being increased over these areas. Auscultation shows fine mucous rales over the affected areas, heard during inspiration and expiration and of a metallic character, indicating pulmonary consolidation.

Complications and Sequelæ.—Bronchitis is present in all cases. Pleurisy in many cases is a complication, accounting for the respiratory pain, especially if the lobules involved are near the surface of the lung or the area of inflammation is extensive. Intestinal catarrh is a common complication in children and infants and is a serious factor. Convulsions may occur and are of grave import; in fact, the brain symptoms in severe cases may resemble those of meningitis.

Diagnosis.—The diagnosis of a lobular pneumonia, supervening as it does upon an existing diseased condition, is often difficult, especially in mild cases with a limited invasion. The inflammation being limited to small and scattered areas of lung tissue surrounded by healthy lung tissue renders the detection of consolidation most difficult. Thus one of the most reliable signs of the disease is not available, and for the same reason bronchial or broncho-vesicular breathing is not marked. The physical signs are those of bronchitis with the added evidence of limited localized areas of consolidation. The crepitant rale is a reliable sign, but is naturally masked in these cases by the moist rales of the co-existing bronchitis of the finer tubes. From croupous or lobar pneumonia, catarrhal pneumonia is distinguished by its bilateral and

scattered development, the gradual onset, supervening upon another preceding disease, its lobular and not lobar character, and in adults by the mode of invasion. From a rapid tuberculosis which it often resembles, the bacilli should be looked for, their presence or absence deciding the diagnosis.

Capillary bronchitis and catarrhal pneumonia so nearly resemble each other clinically that some recent writers and teachers do not treat of them separately. The clinical diagnosis between them is usually well-nigh impossible. In simple capillary bronchitis the temperature runs a lower course as a rule, the prostration is more marked, defective aeration is very pronounced and we fail to find any areas of consolidation. Capillary bronchitis, like catarrhal pneumonia, is the result of an extension into the finer bronchioles of the inflammation starting in the larger tubes, but unlike catarrhal pneumonia there are not areas of consolidation nor the general aggravation of all the fever symptoms that mark the development of the latter.

Prognosis.—In infancy, during old age or cases in which the patient is in an enfeebled condition the outlook is not favorable. The prognosis is affected by co-existing circumstances. Bad nutrition, unhygienic surroundings, rachitic children, chronic nephritis or cardiac complications render it unfavorable. The probable outcome is less encouraging when the pneumonia complicates whooping cough, than when associated with measles. The height of the temperature, the extent of the bronchitis and the amount of consolidation are most important factors. A temperature of 104° to 105° is unfavorable. The ultimate prognosis in cases not going on to

complete resolution is unfavorable, as such areas of consolidation form foci for tubercular infection or abscesses.

General Treatment.—As broncho-pneumonia occurs in systems already depleted by previous disease, the tendency is to exhaustion, hence the indications are to build up the general nutrition and strength, accompanied by remedies to overcome the acute inflammatory process. The patient should be kept at rest in bed; his position should, however, be very frequently changed. Light or liquid nourishment in concentrated form should be frequently administered. Evidence of impending heart failure should be promptly met by the administration of alcoholic stimulants or Strychnine in one-hundreth grain doses. Respiratory failure takes place from obstruction of the air vesicles and finer bronchioles, hence to combat this, employ friction and massage of the respiratory muscles and, when possible, respiratory exercises, deep breathing at intervals, several times daily, artificial respiration, or, best of all, the use of oxygen (as described under pneumonic fever). Emetics are of little value, as the depression resulting from their use more than overbalances the relief they afford. Alternating hot and cold water douches may be necessary in infants threatening suffocation. During the important and critical period of convalescence the patient should receive most careful attention to avoid relapse and to ensure complete recovery. Respiratory exercises, diet, exercise, clothing, hygiene, etc., should be carefully supervised. Cod liver oil, mixed fats, malt preparations, fresh air and change of scene with appropriate remedies constitute the treatment for this stage.

Remedies.—*Aconite.*—Chill or chilliness, sudden rise

of fever with thirst, restlessness and shifting pains. Cough is short, dry and hacking. General aggravation of all symptoms indicating that some new development is taking place. For the first hours of invasion.

Antimonium arsenite.—Valuable in broncho-pneumonia of the aged, with the loose rales and threatened suffocation of antimony associated with the thirst, restlessness and feverish prostration of arsenic.

Antimonium tartaricum.—The most frequently indicated remedy in broncho-pneumonia. Loose cough, chest full of mucus with fine rattling rales. Patient is too prostrated to raise the accumulated secretion which threatens to suffocate. Face pale or cyanotic and covered with clammy sweat, extremities cold, pulse quick and feeble, respiration rapid and oppressed, the whole ensemble one of distress and threatened suffocation.

Bryonia alba.—Cough dry, hard and deep-seated, breathing is oppressed, with soreness and sticking pains, indicating pleuritic involvement, some scanty mucous expectoration, with rawness and pain during coughing effort.

Ferrum phosphoricum.—Anæmic cases with asthenia. Less fever, thirst and restlessness than *Aconite*, but more debility. Hard, dry cough with oppression and expectoration of blood-streaked mucus. Not of use after cyanosis appears.

Iodine.—High fever, hard, croupy cough, marked areas of consolidation, strumous subjects. Valuable in stage of hepatization. Give in low potencies.

Ipecac.—Bronchial tract filled with mucus, loud rattling rales. Loose suffocative cough, with nausea, gagging or vomiting.

Lachesis.—Low condition. The cough is spasmodic and suffocative, waking suddenly from sleep and easily excited by touching the throat or laryngeal region. Difficult respiration, constantly obliged to take a deep breath. Threatened paralysis of the lungs with cyanosis and great distress for breath, especially after sleep. Extreme prostration.

Laurocerasus.—Dry cough or with copious expectoration. Cyanotic condition with great constriction of the chest, gasping for breath, small, feeble pulse, threatened paralysis of the lungs.

Lycopodium.—Cough, with grey, salty expectoration. Dyspnœa, with sense of constriction, shortness of breath and intense weakness. Great difficulty to breathe, with fan-like motion of the alæ nasi. Aggravation from four to seven each afternoon.

Opium.—Cough, with dyspnœa and blue face. Apathy, to a heavy stupor. Difficult, rattling, intermittent respiration, with hot, sweaty skin and drowsinesss and coma.

Phosphorus.—Especially suitable to the hectic type of fever and patient. Much oppression of breathing, with tightness across the chest. Hacking cough, with moderate expectoration of blood-stained mucus.

Squilla.—Chest shows much rattling of mucus which is expelled after violent coughing. The latter causes sharp sticking pains and is accompanied by involuntary micturition. Eyes suffused and watery, thin nasal discharge with excoriated nostrils. This remedy has the loose cough of *Antimonium tartrate*, but without its prostration, while the pains are like *Bryonia*, but the coryza and free secretion are different.

Sulphur.—May be used with advantage in protracted cases as an intercurrent. Also follows well after other remedies during the period of convalescence to promote resolution and absorption of secretion with return to the healthy state.

Veratrum album.—Deep, hollow cough, with much expectoration, suffocation, blueness of the face, weakness, with coldness of the surface and extremities and cold, clammy sweat on the forehead. Collapse and threatened heart failure.

ATELECTASIS.

Definition.—Atelectasis is a condition in which the walls of the pulmonary alveoli are collapsed. It may be divided, as to origin, into the congenital or acquired, and as to form, into the diffuse or lobular. In fœtal life atelectasis is the normal condition of the lungs and its persistence after birth constitutes the congenital form.

Etiology —*Congenital* atelectasis is due to lack of pulmonary development or power to expand the lungs. This may arise from the general feebleness found in children born prematurely; or an early separation of the placenta, compression of the cord or a protracted labor, may cause the child to make breathing efforts before it comes into the world, thus drawing liquor amnii or mucus into and obstructing the bronchioles.

Secondary atelectatsis or the *acquired* form is the most usual and generally is the result of bronchial obstruction, due to plugs of mucus entering the smaller bronchioles during an attack of bronchitis and permanently closing

them; the air in the terminal air cells beyond the point of obstruction is gradually absorbed and the cell walls collapse. This is especially apt to occur in the bronchitis secondary to measles or whooping cough, owing to the enfeebled condition of the inspiratory muscles. Patients enfeebled by rachitic or wasting diseases or bad hygiene are particularly liable to pulmonary collapse during a course of bronchitis. The pressure upon a bronchial tube of an aneurism, an enlarged gland or intra-thoraric tumor may cause collapse.

Pathology.—The lobules affected are depressed below the surface of the lung; they are darker than normal in color and do not crepitate. When cut they appear dense and tough. The lower and posterior portion of the lungs are most affected in the congenital form, while in the acquired the collapsed lobules are diffused throughout the lung tissue. The microscope shows the alveoli completely collapsed containing only a little secretion.

Physical Signs.—In the congenital form, atelectasis may be readily recognized, if it is well marked, by a distinct retraction and inactivity of the chest wall over the inferior ribs posteriorly, due to lack of expansion. In these cases percussion shows dulness, but in the acquired form such areas may be so small and so scattered throughout the lung tissue as not to be discoverable by percussion. Auscultation reveals the rales of the associated bronchitis only, but if large areas are involved in the collapse there may be an absence of respiratory sounds.

Symptoms.—In the congenital form the symptoms are more or less severe according to the extent of the area involved. Fever is absent and the symptoms are those of obstructed respiration. Rapid breathing with

cyanosis, cold extremities, feeble pulse, etc. In the acquired form, if during an attack of bronchitis the breathing becomes suddenly more rapid and difficult with developing cyanosis and depression of temperature with evidence of exhausting vitality, atelectasis may be diagnosed if the physical signs corroborate the symptoms. It is most apt to be confused with lobular pneumonia, and if the areas collapsed are small and scattered a clear diagnosis may be impossible as it usually is associated with lobular pneumonia. In the congenital form, if a newborn infant makes rapid, feeble efforts to breathe, is short of breath when nursing, and has a blue skin and feeble cry, *without rales or cough* (and the heart sounds are normal) atelectasis may be diagnosed.

Prognosis.—Is bad if an extensive area is collapsed. It is stated that one-fourth the mortality of early infancy is due to this condition. The outlook in the acquired form following measles and whooping cough is particularly grave.

General Treatment.—In the congenital form respiratory efforts should be encouraged by slapping, sudden application of cold, artificial respiration, etc. Crying should be encouraged. The physician may apply his mouth to the child's and distend its lungs with air from his own, holding the infant's nose while he does so. An emetic may be given if the bronchial tubes seem filled with mucus with inability to expectorate. Drugs are of little use in this form. In the acquired form of atelectasis, attend to the underlying diseased condition, nourish and build up the patient as rapidly as possible. To stimulate deeper respiration and thus aid in expansion of the collapsed alveoli, instruct in deep breathing, employ

massage, use cold douching, passive gymnastics or electricity. All these are helpful. In event of threatened suffocation inhalation of oxygen should be employed.

Remedies. –The remedies should be those for the associated condition:

Antimonium arsenite.—Restlessness, thirst, fever, sweat and perspiration. Great dyspnœa, threatening suffocation with cyanosis and fine rales.

Antimonium tartaricum.—Is particularly well indicated with its excessive mucous accumulation, rattlin grales, inability to expectorate, cyanosis and exhaustion.

Carbo vegetabilis.—Great oppression, wheezing and rattling in chest, extreme collapse, with bluish color and coldness of the breath and body surfaces.

Lachesis.—Short oppressed breathing with suffocative attacks, ropy mucous expectoration with great dyspnœa. Threatened paralysis of respiration, especially when waking from sleep.

Lycopodium. — Emaciation with bloated abdomen. Atony and apathy. Cachectic children, cough, dyspnœa, labored effort to breathe with motion of alæ nasi. Relief from warm food and drink and 4 to 8 P. M. aggravation.

Sambucus.—Suffocative cough with presence of much mucus. Child turns blue and gasps for breath. Worse just after midnight.

EMPHYSEMA.

Definition.—Emphysema is over-distension of the air vesicles accompanied by loss of elasticity and power to contract, with ultimate atrophy of the alveolar walls, resulting in permanent dilatation of the aveoli.

Varieties.—There are several varieties.

Compensatory.—Whenever one lung or a part of one lung is prevented by disease from fulfilling its functions the neighboring healthy tissue or the opposite lung takes up the double work and the vesicles become distended from the extra effort. This is the case with broncho-pneumonia, tuberculous areas, pleural adhesions, cirrhosis of the lung or pleurisy with effusion.

Vesicular.—The acute distension from bronchitis of the finer tubes with threatened cyanosis, in cardiac asthma, angina pectoris, or pressure on the pneumogastric nerve; the violent efforts to get air in any of these may result in sudden distension and even rupture of the air cell walls.

Interstitial. — During violent coughing, straining at stool or after tracheotomy, air may appear in the interlobular or subplural tissue.

Atrophic.—Senile changes involving atrophy of the alveolar walls with distension of the vesicles. Most frequently seen in withered up old people.

Hypertrophic.—This is the usual variety, characterized by enlargement of the lungs due to distention of the air cells and atrophy and relaxation of their walls, with resulting dyspnœa and evidence of imperfect æration of blood.

Etiology.—Emphysema results from persistent high intra-vesicular pressure acting upon congenitally weak lung tissue. Due to defective nutrition in the alveolar structure and deficiency in development of the elastic fibres. Heredity plays as usual an important part. As exciting causes may be mentioned anything which induces forced inspiration or expiration. Asthma and chronic bronchitis are the most frequent causes, whoop-

ing cough, violent prolonged exertion, players on wind instruments, glass blowers, etc.

Pathology.—The thorax becomes capacious, barrel-shaped, and the cartilages are calcified. The lungs are large and have lost their elasticity. The air vesicles are much distended, many are coalesced, producing greatly enlarged cells with atrophy of the frame work and absence of elastic fibres. In the bronchi the mucous membrane is rough, thickened, and the finer bronchial tubes are much distended.

Symptoms.—Dyspnœa is constant or is produced by slight exertion. Respiration harsh, wheezy and much prolonged. Cyanosis is present in varying degree, sometimes very marked, even startling, and out of all proportion to the patient's apparent comfort and ability to get about. This feature is characteristic, for the same degree of cyanosis if due to heart disease or other variety of lung trouble would mean that the patient would be in bed and near death. Bronchitis with cough is a constant symptom, with relief in summer and aggravation in winter. Asthma is frequently present. As age increases recurrent attacks of bronchitis occur and the condition grows worse. Death occurs from intercurrent pneumonia, cardiac dropsy, or cardiac distension and cyanosis.

Physical Signs.—Inspection shows the chest to be barrel-shaped, the antero-posterior diameter being equal to the lateral. The sternum and costal cartilages are prominent, the inter-costal spaces are widened, with immobility during respiration.

Palpation. The apex beat is rarely felt, but there is pulsation in the epigastrium. Percussion gives greatly increased resonance, full and drum-like. Heart dulness

obliterated and liver line of dulness lowered. Auscultation gives breathing sounds enfeebled with a prolonged expiration (4–1 instead of 1–4) harsh and rough, accompanied by coarse rales.

General Treatment —Emphysema is incurable as far as correcting the morbid changes already established in the air vesicles; the process, however, may be arrested. Establish a sound digestion and give a rich nitrogenous diet, avoiding starch and sugar. Alcohol and tobacco are injurious and should be forbidden. A milk diet, peptonized or with lime water, may be necessary in those of perverted or feeble digestion. Protect the patient from cold and dampness. Outdoor exercise is beneficial, avoiding, however, everything that will increase respiratory effort. The cough must be kept in check by appropriate remedies aided by mild palliatives if necessary. Inhalation of oxygen will give much relief in severe cases with marked cyanosis.

Remedies.—There are no remedies for emphysema *per se*.

Antimonium arsenite, *Antimonium tartaricum*, *Kali bichromicum* and *Ipecac* are most frequently indicated for the associated chronic bronchitis. Also *Calcarea carbonica* for fleshy females who perspire easily and with copious menstruation. *Calcarea phosphoricum* for old men with atheromatous blood vessels. *Lycopodium* in flatulent dyspeptics with excess of uric acid and gouty symptoms. *Antimonium arsenite*, *Grindelia robusta*, *Ipecac* or *Lobelia inflata* for the frequently accompanying asthma. Gastric symptoms call for *Argentum nitricum*, *Lycopodium*, *Carbo vegetabilis* or *Nux vomica*. In the later stages with cardiac weakness *Arsenicum album* 2x, *Strychnia nitrate* 2x, or *Digitalis θ* may be required.