DISEASES OF THE LUNGS.

CONGESTION OF THE LUNGS.

SYNONYMS. Pulmonary engorgement; hypostatic congestion.

DEFINITION. An increase in, or abnormal fullness of, the capillaries of the air cells; active congestion when the result of an accelerated circulation; passive congestion when caused by an impeded outflow from the capillaries.

CAUSES. Active.—Increased cardiac action; over-exertion; alcoholic excesses; mental excitement; inhalation of cold or hot air. Passive.—Obstruction to the return circulation. Dilated heart; valvular diseases; low fevers (hypostatic congestion); Bright's disease.

PATHOLOGY. The hyperaemic lung has a bloated, dark red appearance; its vessels are distended to the uttermost, the tissues succulent and relaxed, blood flowing freely over the cut surface; a bloody, frothy liquid is present in the bronchi; and the alveolar walls are so much swollen that the condensed lung shows scarcely any indication of its cellular structure, resembling the tissue of the spleen (splenification).

SYMPTOMS. Active.—Rapidly developing thoracic distress and difficulty of breathing, flushed face, strong, full pulse, throbbing carotids, cardiac palpitation, and congested eyes, with a short, dry cough, followed by scanty, frothy expectoration, slightly streaked with blood. Passive.—Developed slowly, with difficulty of breathing, blueness of the surface, almost continuous hacking cough, followed by scanty, blood-streaked expectoration.

PERCUSSION. The resonance of the lungs slightly diminished, the quality of the sound being somewhat tympanitic.

AUSCULTATION. The vesicular murmur is diminished and accompanied with subcrepitant rales.

DURATION. Active.—Usually from three to five days, terminating either by resolution, hemorrhage, or, rarely, pneumonia. The onset may be so severe and overwhelming that death rapidly supervenes. Passive.—Developed slowly, and is subject to great variations, depending upon the cause.

DIAGNOSIS. Active congestion of the lungs can not be distinguished from the stage of engorgement of a true pneumonia.

PROGNOSIS. An acute congestion of the lungs may prove

fatal within a few hours, but under prompt treatment it generally terminates favorably.

THE TREATMENT.

Freedom of the circulation of the blood in the upper portion of the body should be the first thing to seek. The general treatment is the proper one to institute. Free the neck muscles and intercostal muscles of venous pressure. The clavicles are to be elevated, and the arms drawn strongly upward and backward, the patient directed to inhale all the air the lungs will comfortably hold as each move is made, to free the chest pressure. The venae azigos, major and minor, demand attention, hence treatment down the back from the fifth cervical to the eighth dorsal on both sides of the spine; and pulling of the arms strongly as permissible without producing pain, should be done, endeavoring to remove all the constricture of the chest, to give ample room for lung expansion. The manipulatory vibrations on the chest walls assist in removing the intercostal congestion that is holding the chest muscles and rendering them immobile. Take off the pressure everywhere, and require systematic exercise in breathing at stated periods, several times at a sitting, every two to four hours. In this condition treatment between the shoulders becomes one of the essentials. Stimulate the diaphragm and spine, and effect a general liberation of obstructions all over the body. Treatments repeated every four to six hours, gently, and after relief is obtained, as often as required.

ŒDEMA OF THE LUNGS.

SYNONYM. Pulmonary œdema.

DEFINITION. An exudation of serum into the pulmonary interstitial tissue and the alveoli of the lungs; characterized by dyspnœa, cough, and a frothy, blood-streaked expectoration.

CAUSES. Pulmonary œdema is the result of stasis, occurring when the outflow of venous blood in the lung meets an obstacle that can not be overcome by the right ventricle, as in cardiac diseases, in which the left ventricle fails. Bright's disease; alcoholic excesses, causing cardiac depression. Sequelae to other lung inflammations.

PATHOLOGICAL ANATOMY. The lung tissue is swollen, and does not collapse when the chest is open. The elasticity of the tissue has disappeared, and it pits upon pressure. If following

congestion of the lungs, the color is red; if a symptom of a general dropsy, its color is pale. On cutting into the œdematous spots an enormous quantity of albuminous fluid, sometimes clear, at other times of a red color, mixed more or less with blood, flows over the cut surface. The liquid is filled with bubbles, is frothy, from being copiously mixed with air, providing the air cells have not been entirely filled with serum, thereby excluding the air.

SYMPTOMS. The pre-eminent symptom is dyspnœa, the breathing being hurried, labored and rattling, all the accessory muscles of respiration being called into action. The sense of oppression and anxiety is extreme. There is also a constant, harassing, short cough, and the expectoration is a blood-streaked, frothy mucus. The action of the heart may be tumultuous or feeble. The face is at first flushed, but as the left ventricle fails, or if the effusion into the air cells be sufficient to prevent the entrance of air, symptoms of cyanosis rapidly supervene, the pulse becoming feeble, the surface cold, the breathing shallow and hurried, the cough suppressed, stupor replacing the restlessness, soon deepening into coma.

PERCUSSION. If no other lung disease, the percussion note is but slightly, if at all, impaired.

AUSCULTATION. The vesicular murmur is lost by the diffused subcrepitant and bubbling rales.

DIAGNOSIS. Acute pneumonia in the earlier stages is the only condition likely to be confounded with œdema of the lungs, but as the two diseases progress, the picture of pulmonary œdema is so characteristic that it can not be mistaken.

PROGNOSIS. Grave, and particularly if occurring in pneumonia, cardiac, or Bright's disease. In the majority of instances it is a terminal symptom coming on in all forms of acute and chronic diseases.

THE TREATMENT.

Every indication points to compression of the chest muscles. The lungs are compressed perhaps, from a contraction of the pectoral and all of the intercostales, sub-claviculae, and more than likely the muscles of the neck are drawn tightly over the veins of the neck (the jugulars), hence the indications are to take off the pressure. Placing one hand at the side of the neck, the forefinger free, and three fingers in such a position that, by raising the arm the fingers are made to press the clavicle outward, and raise the chest muscles, taking off the pressure and so relieving the lungs, heart and diaphragm that the patient feels entirely relieved.

After this the general treatment should be given, beginning at the neck. It will be found necessary to equalize every particle of the circulating fluid in the body so as afford opportunity to oxygenate the blood.

To stand by and see a person smother to death in order to observe a phenomenon resulting from pressure is surely reprehensible, when we have the ability to afford immediate relief—surely unworthy the high position that the physician should occupy. There is no indication for medicine. Then, when there is a means always at hand for relief, why let prejudice close the opportunity, and witness imminent dissolution and inexpressible agony while death is encircling its victim? Osteopathy proves its efficacy in these cases most satisfactorily, if properly applied. Repeat general treatment every two to six hours.

CROUPOUS PNEUMONIA.

SYNONYMS. Lobar pneumonia; pneumonitis; fibrinous pneumonia; pleuro-pneumonia; lung fever; winter fever.

DEFINITION. An acute, infectious, croupous inflammation, involving the vesicular structure of the lungs, rendering the alveoli impervious to air; characterized by a severe chill, headache, fever, thoracic pain, dyspnœa, cough, rusty sputum, and great prostration.

CAUSES. Croupous pneumonia is an infective disease caused by the diplococcus pneumoniae of Fraenkel, "which has its seat of election in, and produces its chief effects on, the lung." All ages liable. Males more frequently affected than females. One attack predisposes to another. Debilitating causes render individuals more susceptible. Alcoholism is one of the most frequent predisposing factors. It is most frequent in winter, at times occurring epidemically, the result of atmospheric conditions; exposure to draughts and cold. Gout, rheumatism, diabetes, and Bright's disease.

PATHOLOGICAL ANATOMY. The most frequent seat of croupous pneumonia is the lower right lobe; the next most frequent seat is the lower left lobe; the next, the upper right lobe, although in children and the aged this lobe is affected equally as often as the right lower lobe. The changes are: 1. Hyperaemia (engorgement); 2. Exudation (red hepatization); 3. Resolution (gray hepatization); or it may undergo purulent transformation or the development of abscesses (yellow hepatization).

1. Stage of hyperaemia, or engorgement, consists in the vessels of the alveoli being distended to their utmost, encroaching upon the cavity of the air vesicle; the lung has a reddish brown color, is heavier, sinking somewhat lower in water than a normal lung, and having a slight exudation upon the vesicular surface. The same changes are perceived in the adjacent bronchioles.

2. Stage of exudation, consists in the exudation of a viscid, fibrinous fluid, admixed with white and red corpuscles and blood, which rapidly coagulate, firmly inclosing the corpuscles and completely filling the alveoli. When the exudation and coagulation are completed, the lung is red, sinks at once when placed in water, and its elasticity is destroyed. When cut into, the color, density, and granular appearance so closely resemble the cut surface of a section of the liver, that Laennec termed it red hepatization. A thin section shows under the microscope, as a rule, the lancet-shaped diplococcus of Fraenkel, as well as staphylococci and streptococci.

3. Resolution, or gray hepatization, follows the above condition in the majority of cases, the coagulated albuminous exudation undergoing liquefaction and absorption, the cellular element undergoing a fatty degeneration, the greater part being absorbed, the remainder expelled during acts of expectoration, the alveoli returning to their normal condition, both as to capacity, function and elasticity.

If resolution be retarded and portions of the coagulated exudation undergo purulent transformation, changing from a yellowish to a greenish yellow color (yellow hepatization), pus cells are rapidly formed, the part becoming a granular, fatty mass. The portions of the lung not undergoing this purulent transformation retain the reddish color with intermixed yellowish patches, the lung structure proper remaining intact. The purulent contents may be ejected in part, the remainder undergoing fatty degeneration and finally absorption.

Abscess of the lung may result from the lung structure becoming involved in the purulent disintegration. Abscesses may be solitary or in great numbers, which by disintegration of intervening structure form one or more large abscesses; these abscesses either terminate fatally, or open into the pleural cavity, causing empyema and exhaustion, or open into the bronchi and are expectorated, or an interstitial pneumonia is developed and the abscess encapsulated in a firm cicatricial tissue.

Gangrene of the lungs may result from blocking up of the

bronchial or pulmonary arteries by coagula during any stage of the disease.

The uninflamed portions of the lungs are hyperaemic and their functional activity is increased.

Death sometimes results from a general œdema of the unaffected lung, such cases being often erroneously termed "double pneumonia."

If inflammation of the pleura be associated with a pneumonia the so-called pleuro-pneumonia, the changes in the pulmonary pleura are characteristic. "An uneven, thin, downy-looking layer of plastic exudation covers its surface. This plastic layer may conceal the liver brown color of the pneumonic lung. As the third stage is reached, the opposing surfaces of the pleura may become agglutinated. The pleuritic changes follow very closely those which occur within the lung. The cells in the pleuritic exudation are mainly pus. The pleuritic membrane is opaque, congested, and ecchymotic. It may become so thick as to give a dull note on percussion, after resolution is reached."

Duration of Stages.—Stage of congestion, from one to three days; stage of exudation, from three to seven days; stage of resolution, from one to three weeks. In severe cases or in the very young, the aged, or the depressed, the stage of red hepatization may be fully developed within forty-eight hours.

Symptoms. Begins with a severe and usually protracted chill (in children often convulsions, adults vomiting), followed by a rapid rise of temperature, 103-104 degrees F., a strong, full, but rapid pulse, soon showing evidences of embarrassed cardiac action from obstructed respiratory circulation, either a dull or sharp pain near the nipple, aggravated by pressure, breathing, or coughing, shortness of breath, the inspiration short and superficial, the expiration accompanied with a moan or grunt, the number of respirations increasing to 40, 50 or more per minute, causing interrupted speech, the ratio between pulse and respiration may be 1 to 2 or more; cough, first short, ringing, and harsh, soon followed by a scanty, frothy mucus, soon becoming semi-transparent, viscid, and tenacious, about the second day changing to the familiar rusty sputum, becoming more copious and of a yellow color as the disease advances; rarely cases occur with bloody or blood-streaked sputum during the continuance of the fever. There are present headache, sleeplessness, rarely delirium, save in drunkards, epistaxis, flushed countenance, and especially over the malar bones is a well-defined mahogany blush; gastric

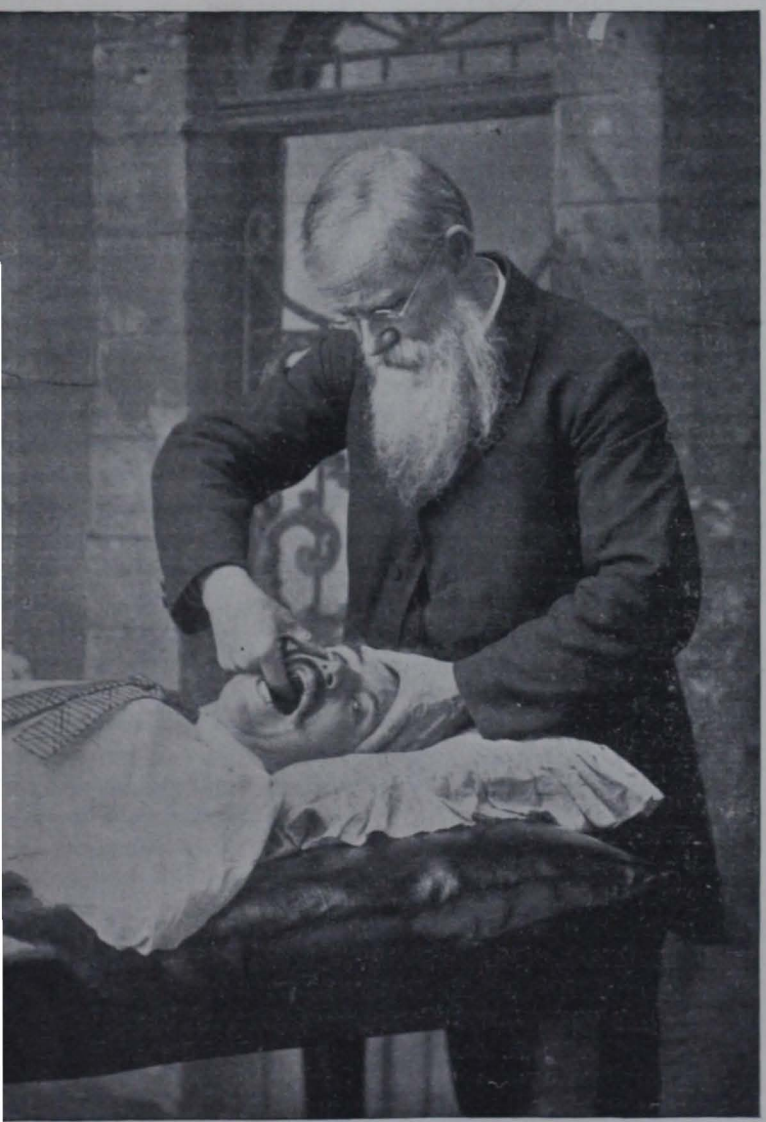
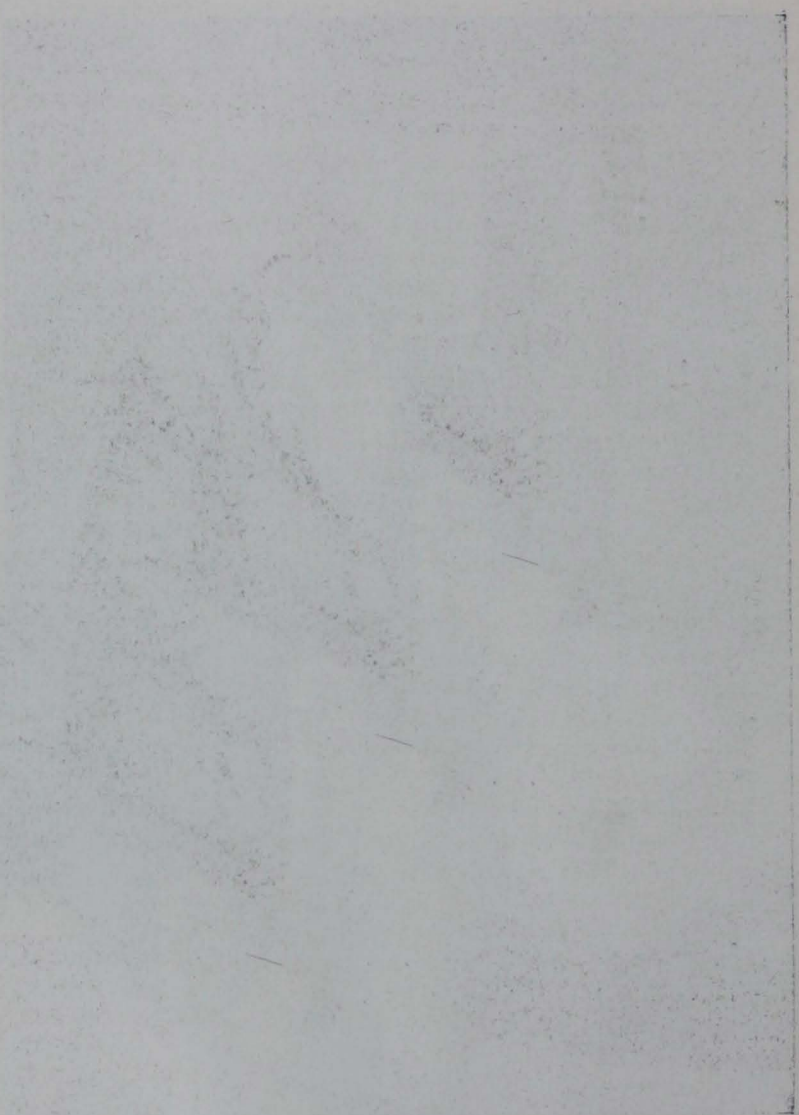


PLATE XXXVII.—Manner of Treating the Soft Palate.



disturbances and scanty, high-colored urine, with diminished chlorides, and often albuminuria. From the very onset of the disease the prostration is of the most serious character. The above symptoms continue more or less marked until either the fifth, seventh, ninth, or eleventh day, when a crisis occurs, and within twenty-four hours convalescence is established, recovery rapidly following.

Typhoid pneumonia is a term applied to those cases which are accompanied by signs of extreme prostration, delirium, tremor, very high temperature, and profuse and prolonged exudation. They may also terminate by a crisis.

Bilious pneumonia occurs in cases accompanied by congestion of the liver, the result of venous stasis from pulmonary obstruction or from an accompanying acute catarrhal jaundice. In malarial districts pneumonia and malaria are often associated, when jaundice more or less pronounced occurs. Such cases are termed malarial or intermittent pneumonia.

Alcoholic, or pneumonia of the intemperate, has one very characteristic symptom, to-wit: early delirium. In pneumonia generally the mind is clear when all the conditions are unfavorable. Pneumonia of the intemperate may begin with symptoms closely resembling an attack of delirium tremens, cough, expectoration, and pain being very slight, or even absent.

If purulent infiltration follow the stage of red hepatization, instead of the crisis, symptoms of exhaustion occur, with profuse purulent expectoration, high temperature, severe sweats, the tongue brown and dry, sordes collecting on the teeth, low delirium, feeble pulse, rapid, rattling breathing, the recovery slow and convalescence tedious.

Pneumonia in the aged or the insane may be latent, coming on without chill or pain, and with only a slight fever; the cough and expectoration are slight, physical signs ill-defined and changeable, and the constitutional symptoms out of all proportion to the amount of lung involved.

INSPECTION. First Stage.—Deficient movement of the affected side, due to pain. Second Stage.—The healthy side rises normally, the affected side lagging behind. If both lower lobes are impervious to air, the diaphragm can not descend and the epigastrium does not project during inspiration, the breathing being conducted by the upper part of the chest (superior costal respiration).

PALPATION. First Stage.—The vocal fremitus more distinct

than normal. Second Stage.—The vocal fremitus is markedly exaggerated, except in those rare instances of occlusion of the bronchi by secretion. The cardiac impulse is felt in the normal position.

PERCUSSION. First Stage.—The percussion note is slightly impaired, indeed, at times having a hollow or tympanitic quality. Second Stage.—Dullness over the affected parts, with an increased sense of resistance.

AUSCULTATION. First Stage.—Over affected part, feeble vesicular murmur, associated with the true vesicular or crepitant (crackling) rale, most distinct during inspiration. Second Stage.—Harsh, high-pitched, bronchial respiration, at times resembling a to-and-fro metallic sound, except in those rare instances in which the bronchi are more or less filled with secretion. Bronchophony, or distinctly transmitted voice, at times pectoriloquy, or distinct transmission of articulated sounds, is present. Third Stage.—Breathing changing from bronchial to vesiculo-bronchial, the crepitant (crepitatio redux) rale returning, and if resolution proceed, the breath sounds are associated with large and small moist and bubbling rales.

"The morbid phenomena, physical signs, and symptoms of the malady correspond usually in this manner" (Da Costa):

I. Stage of engorgement and beginning of exudation.	Crepitant rale; slight percussion dullness.	Cough; beginning dyspnœa and rapidly developed fever heat.
II. Stage of solidification of lung tissue (red hepatization).	Percussion dullness; bronchial respiration; bronchophony.	Rusty-colored sputum, dyspnœa; cough; high fever with marked evening exacerbations and morning remissions.
III. Stage of softening (gray hepatization).	Same physical signs as in second stage unless large abscesses have formed.	Chills; prostration, etc., purulent or brownish sputum; generally high temperature.

TERMINATIONS. Asthenic cases recover within two weeks. When purulent infiltration supervenes, the disease pursues a tedious course of several weeks' duration, with a low exhaustive fever. If death occur during the first or second stages it is usually the result of a collateral œdema of the uninflamed lung, or cardiac failure and impaired nerve force. If abscesses occur, there are exhausting sweats, frequent cough, with a large amount of yellowish-gray, at times blood-streaked, expectoration. Gangrene of the lungs is a rare termination; it is associated with

symptoms of collapse, the expectoration of a blackish, fetid sputum, and the physical signs of a pulmonary cavity.

DIAGNOSIS. Œdema of the lungs may be confounded with the first stage of pneumonia, but the subsequent history, its presence on both sides, and the waterish expectoration and absence of chill and pain and the physical signs of pneumonia soon determine the diagnosis. Pleurisy is oftener confounded with pneumonia than any other disease, the points of distinction between which will be pointed out when discussing that affection.

COMPLICATIONS. Acute pleuritis is a frequent complication of croupous pneumonia, occurring as often as from ten to twenty-five per cent. of cases. The more acute localized pain, the greater embarassment of respiration, and the usual physical signs of effusion are the evidences of a pleuro-pneumonia. Capillary bronchitis is a rare but dangerous complication. Pericarditis, rheumatism, and gout are rare complications.

PROGNOSIS. Depends upon the extent of the inflammation, the dangerous features of croupous pneumonia being cardiac failure, the result of a myocarditis or of embarrassed respiratory circulation, and the rapid tissue waste associated with extreme fever, 105 degrees, resulting in impaired nerve force; double pneumonia has a very grave prognosis, but it is not nearly so frequent as was at one time supposed. The co-existence of pleuritis adds to the gravity of the prognosis, although not as fatal as generally supposed. Pneumonia of drunkards almost invariably terminates fatally. Typhoid pneumonia, pneumonia of the aged and in the insane, the so-called bilious pneumonia, purulent infiltration, abscesses of the lungs and gangrene, all give a grave prognosis.

THE TREATMENT.

When the picture of consequences is observed, in the various stages of this affection, is it not plainly to be seen that venous circulation stands as the only cause of this affection? Watching its various stages, until finally purulent infiltration supervenes, who, with a knowledge of the science of Osteopathy, can but censure the course usually pursued by the medicine dependents for relief—when there is not a single effort made to take off the pressure, that the patient be relieved from this most distressing condition, and arrest consequences that must, with the pressure continued, follow? As the contraction tightens down on all of the nerves that control the circulation, is it any wonder that their functions should cease, and stasis come to every tube controlled

thereby? Every stifled breath and every piercing pain call for help to lift off the pressure. Instead of that the medicine doctor pours his mixtures into the stomach, or hypodermically, expecting favorable results, when his poor victim is suffering the very tortures of the "damned" while trying to live. The stereotyped routinism of the ages has entailed untold misery and slain more than famine, pestilence and sword. The idea of taking off the pressure has not once entered the citadel of thought of the never-so-wise-appearing "assisters," called doctors. It seems like the minds of men have been surreptitiously inveigled so long that freedom is obscured. The apparatus (lungs) for manufacturing pure blood, to build up healthy tissue, is interfered with in the disease under consideration, and the important thing to be done is to reinstate their functions. Capillary congestion, due to pressure, either on the venous system that carries the blood out of the lungs, or on the nervous system that controls the peristalsis of the muscular walls of the blood-vessels, causes the difficulty. The cerebro-spinal nervous system most likely is where the difficulty lies. Cold, compressing the muscular fiber around the terminal filaments in the upper dorsal area, prevents normal reflexes to the brain, thence through the pneumogastric and the sympathetic to the lungs, and the contraction of the chest muscles obstructs the return blood through the veins; hence the carbonic oxide increases, tissue changes ensue, decomposition starts up, poisons accumulate, the normal action is interfered with, blood accumulates in the capillaries and inter-capillary tissue. Œdema is the result. The most forcible indication points to "taking off the pressure." This is done by following the general directions for freedom of the circulation. These are simple, and a general knowledge of the anatomy of the system gives assurance to the operator that accomplishment is certain in all such cases, where too much tissue change has not already occurred. We therefore begin at the vaso-motor area, give thorough general treatment, using the limbs as levers to lift the weights. Gently stretching the spinal nervous system by the hands under the chin and at the occiput accomplishes the object most admirably. All of the neck manipulations should be carefully, profoundly, thoroughly made first; the clavicles and arms gently but strongly raised, the arms extended sidewise and slightly upward as the finger ends are pressed firmly on the side of the upper dorsal spine, and the patient induced to deeply inhale at the same time. This tends to, and does, free the capillary congestion of the lungs, oxygenate

the blood and increase general circulation. The spine is to be stimulated by the vibratory sudden moves along each side of the processes, and lifting upward and outward, the muscular structure clear down the back. The entire general treatment should be given as far as possible each time, and treatment may be given at short intervals until decided amelioration ensues. The manipulations tend to increase the secretions for a short time, and the expectoration shows activity until the accumulation is expelled. Where hepatization results before osteopathic treatment is applied, a continuation or the institution of these manipulations soon removes the difficulty, and air begins to permeate the tissue, regeneration begins, health results. Foreign substances in the way of drugs do nothing but load the blood with impurities. The proper application of the principles of Osteopathy, as indicated and pointed out, satisfies patient and friends that Osteopathy is the *sine qua non*.

CATARRHAL PNEUMONIA.

SYNONYMS. Broncho-pneumonia; lobular pneumonia; capillary bronchitis (?).

DEFINITION. An acute catarrhal inflammation of the bronchioles and alveoli of the lungs, characterized by fever, cough, dyspnœa, copious expectoration, and great depression.

CAUSES. From an extension of a bronchial catarrh downward; following the eruptive fevers, especially measles; complicating whooping cough. Persons of the rickety or scrofulous diathesis, in whom there is a greater irritability of the epithelial elements, are particularly predisposed to this form of pneumonia on slight exposure; emphysema; diseases of the heart; most frequently seen in childhood and old age. Bacteriological investigations seem to indicate that secondary broncho-pneumonia is due to more than one germ.

PATHOLOGICAL ANATOMY. Hyperaemia of the mucous membrane of the bronchi, extending to the connective tissue of the bronchioles and accompanying arterioles and to the alveoli, with swelling and succulence of these tissues, accompanied by an abnormal secretion and an immense production of young cells from the proliferation of the bronchial and alveolar epithelium, admixed with a yellowish, creamy, mucoid material, which blocks up the bronchioles and air cells. The affected parts first have a reddish-gray, soon changing to a yellowish-gray color, due to

the rapid metamorphosis of the newly developed ceils. If the fatty change be completed, absorption takes place, and the consolidation is removed; if it remain incomplete the cells atrophy, the little mass becoming caseous, and the disease passes into a chronic state. The bronchial tubes also participate in the disease, the walls become thickened, from a hyperplasia of the connective tissue (peri-bronchitis), and their caliber is often dilated.

SYMPTOMS. Catarrhal pneumonia begins as a catarrhal bronchitis. It may be either acute, subacute, or chronic in its course.

Acute Variety.—Its onset is announced by a gradual rise of temperature to 102-103 degrees F., the febrile phenomena assuming a typical remittent character, with rapid, laborious, and shallow breathing, as shown by the widely dilated nares and violent action of all the accessory muscles, while the insufficient distention of the lungs is shown by the great recession of the lower part of the chest walls and sinking in of the intercostal spaces. The inspiration is short and imperfect, the expiration noisy and prolonged; the pulse is frequent, 100-120 or more, and somewhat compressible; the cough, which, during the bronchitis, was loose, now becomes short, hacking, dry, and painful, soon followed by more or less copious muco-purulent expectoration; the appetite is impaired, bowels somewhat loose, urine scanty, high-colored, and the surface frequently covered with more or less profuse perspiration.

The subacute and chronic varieties have the same general symptoms, but the duration is longer and the exhaustion greater.

The progress of catarrhal pneumonia is sometimes, although not often, a very acute one. The disease may prove fatal in a few days, especially if it attack feeble children; in such the countenance becomes pale and livid, the lips bluish, the eyes dull, and a restlessness giving place to apathy, and a continually augmented somnolence. Resolution, when it occurs, is by lysis, several weeks elapsing before complete recovery.

PERCUSSION. Dullness, scattered in patches, over both lungs, the intervening healthy lung often giving a more or less hollow or tympanitic note.

AUSCULTATION. Vesiculo-bronchial breathing, changing to moist bronchial breathing, associated with small bubbling (subcrepitant) rales. As the disease progresses toward resolution, the rales become larger (large bubbling) and more copious. If

pneumonic phthisis result, physical signs indicative of that condition are soon evident.

SEQUELÆ. Attacks of catarrhal pneumonia complicated with atelectasis, or collapse of the lobules, when recovery occurs, are followed by emphysema of the lungs. If the catarrhal products which fill the alveoli and bronchioles and intervening connective tissue do not rapidly undergo complete fatty metamorphosis and consequent absorption, pneumonic phthisis results.

DIAGNOSIS. Ordinary bronchial catarrh differs from catarrhal pneumonia by the absence of dyspnœa, fever, and dullness on percussion, and the presence of the large bubbling rales, and also by the subsequent history of the two affections.

Croupous pneumonia is a unilateral disease; catarrhal pneumonia is bilateral and diffused over both lungs; the former a self-limited disease, the latter having no fixed duration.

Acute tuberculosis at its onset is characterized by the presence of a capillary bronchitis, a differentiation being possible only by a study of the clinical history and course of the two maladies and the presence or absence of the tubercular bacilli.

Œdema of the lungs is a bilateral disease associated with a short, dry cough, and dyspnœa, but lacks the previous catarrhal history and high temperature of catarrhal pneumonia.

PROGNOSIS. Fully one-half of the cases of true catarrhal pneumonia terminate fatally. The prognosis must be guarded in scrofulous or rachitic subjects, or those enfeebled by other diseases, for unless prompt resolution can be effected, it will terminate fatally early, or develop pneumonic phthisis. Have seen cases continuing up and down for eight and ten months, and finally make a good recovery.

THE TREATMENT.

When it is considered that fully one-half of the cases of this affection succumb under the ordinary treatment, and ninety per cent. are cured by proper Osteopathic manipulations, does it not stand to reason that merit settles the preference for it? We venture the assertion that not a single case of pneumonia ever occurred without pressure on some nerve or nerves leading to and controlling the lung tissue, or interfering with the return venous circulation. In this affection the same treatment that is recommended for croupous pneumonia should be made. The early institution of the treatment will most frequently abort the disease at once. There is no other means ever used that equals this process of curing these conditions. Every variety of lung

affection should receive the treatment indicated, to take off the pressure and re-establish normal circulation to and from the lungs and heart, and to stimulate the nerve terminals, and to arrest all muscular contracture and compression of the chest contents, including liver, diaphragm and abdominal muscles. The equalization of the circulation must be brought about and maintained to restore and perpetuate life and health. The general treatment, then, should be applied in this pathological condition as far as necessary to accomplish freedom of vessels and nerves and lymphatics. All these are accomplished by the application of the foregoing principles effectually. Treatment should be made every day, or oftener, should emergencies demand it, partial or complete.

PULMONARY TUBERCULOSIS.

SYNONYMS. Phthisis pulmonalis; phthisis; consumption; pneumonic phthisis; tubercular phthisis.

DEFINITION. An infective disease, caused by the bacillus tuberculosis, the lesions of which are characterized by nodular bodies called tubercles or diffused infiltrations of tuberculous tissue which undergo caseation or sclerosis, and may finally ulcerate, or in some situations calcify.—Osler.

CAUSE. It is now generally accepted that all varieties of pulmonary consumption are due to the active presence of the bacillus tuberculosis, discovered by Koch in 1881. The lung tissue must be in a receptive state, as the bacilli may be present in the respiratory tract without the development of the disease. Any condition that lowers the tone of the general system renders the tissues susceptible to the changes produced by the tubercle bacilli. These will be enumerated in speaking of the clinical varieties of the disease.

CLINICAL VARIETIES. 1. Acute miliary tuberculosis; 2. Pneumonic phthisis; 3. Tubercular phthisis; 4. Fibroid phthisis.

I. ACUTE MILIARY TUBERCULOSIS.

SYNONYMS. Acute phthisis; galloping consumption.

DEFINITION. An acute infective febrile affection, due to the rapid eruption in various parts of the body, but especially in the lungs, of miliary tubercles; characterized by high fever, rapid

pulse, hurried respiration, pains in chest, cough, profuse expectoration, and rapid prostration.

CAUSES. In the majority of cases it is the result of an auto-infection, arising from either an active or latent tuberculosis focus. Cases develop in which no cause can be assigned. Often follows measles, whooping-cough, variola, and influenza. Most common between puberty and middle life.

"That the gray granulation is deposited throughout the body under the influence of certain conditions of irritation, it is necessary that a peculiar vulnerability of the constitution exist, in other words, that it be of the scrofulous type.

CLINICAL FORMS. General or typhoid, pulmonary and cerebral. The cerebral will be described in the section on nervous diseases.

PATHOLOGICAL ANATOMY. Pulmonary Form.—"The gray granulation or miliary tubercle consists of a fine reticulation of fibers, with a mass of epithelioid cells and granules, and often having a giant cell for its center." The deposit is generally over both lungs and the bronchial tubes, and is followed by hyperaemia, increase of secretion, having a viscid and adhesive character, and the destruction of all the tissue with which it comes in contact. Deposits also take the place in the brain, pleurae, intestines, peritoneum, and kidneys.

GENERAL OR TYPHOID. Symptoms.—Gradual progressive weakness, with loss of appetite, dry, clean tongue, costive bowels, flushed cheeks, fever, irregular in type, and rapid, feeble pulse. Rarely the temperature reaches 103 degrees F. to 104 degrees F., associated with mild delirium. The respirations are increased with slight or no cough, and little or no expectoration. As the symptoms continue the prostration increases, cyanosis develops, the patient growing stupid, gradually deepening into coma and death.

Diagnosis.—There are none or so slight local conditions, the symptoms pointing to an acute general infection, that the disease is apt to be mistaken for typhoid fever. The points of difference are, absence of the typical typhoid temperature record, the characteristic eruption, and the diarrhea.

Prognosis.—Recovery is the rarest termination.

PULMONARY FORM. Symptoms.—The onset is usually sudden, with a chill or chilliness, followed by fever, 102 degrees to 104 degrees F., rapid, dicrotic pulse, 120-140, cough, with scanty, glairy sputum, increased respiration, 30-50 per minute, pain in

the chest, hot skin, dry tongue, deranged digestion, and great prostration, the severity of the symptoms rapidly increasing, with evidences of cyanosis, the sputum becoming more abundant and often rusty in color, with more or less frequent attacks of haemoptysis, soon followed by headache, vertigo, sleeplessness, often delirium, coma, and death. If deposits have occurred in the meninges, or the intestines, symptoms of these affections are superadded.

PERCUSSION. The percussion resonance is normal until considerable deposits have occurred, when it is either slightly impaired or even slightly tympanitic. With the development of cavities the amphoric percussion note is present.

AUSCULTATION. Vesiculo-bronchial breathing, associated with large and small, moist or bubbling rales, soon followed by bronchial and broncho-cavernous breathing, with large and small, moist and circumscribed gurgling rales.

DURATION. Acute phthisis usually terminates fatally in from four to twelve weeks. Rarely of several months' duration.

DIAGNOSIS. Commonly mistaken for typhoid fever with lung complications, an error that is readily made unless a close study of the history, symptoms, physical signs, and sputum be made.

THE TREATMENT.

In the early stages of this affection much may be done by applying the principles of Osteopathy to the contingencies as they arise, lifting off the weights that obstruct the breathing and blood circulation to and from the lungs and throughout the general system. Treat patient according to indications, and as often as the circumstances permit. Aim to relieve pressure.

II. PNEUMONIC PHTHISIS.

SYNONYMS. Chronic catarrhal pneumonia; catarrhal phthisis; caseous pneumonia; caseous phthisis.

DEFINITION. A form of pulmonary consumption characterized by the destruction of the pulmonary tissue resulting from the action of the bacilli, causing the caseation or cheesy degeneration of inflammatory products in the lungs, and the subsequent softening and destruction of the caseous matter, with greater or less destruction of the pulmonary tissue; characterized by hectic fever, cough, shortness of breath, purulent expectoration, and more or less rapid prostration.

CAUSES. The predisposing factor in the etiology of pneumonic phthisis is a strumous or scrofulous diathesis, or a condition of lowered health, the result of various unfavorable hygienic influences. The exciting causes are: the irritation produced by the presence of the bacillus tuberculosis and a catarrhal pneumonia in any portion of the lung, but especially at the apex; inflammation occurring about a blood clot; inhalation of irritant particles occurring in certain occupations, to-wit: weavers, grinders, miners, hatters, millers, cigar-makers, and the like. Many cases of pneumonic phthisis can be traced to an attack of influenza a year or so before.

PATHOLOGICAL ANATOMY. When a pneumonia terminates in resolution the inflammatory products are absorbed by first undergoing a fatty metamorphosis. If the fatty metamorphosis be incomplete the cells are atrophied and undergo the caseous degeneration, which consists in the absorption of the watery parts, the fatty degeneration of the cellular elements, and the granular disintegration of the fibrinous material, so that ultimately a soft, solid mass is produced, yellowish in color, having the appearance of cheese. The destructive changes are thus described by Niemeyer: "Cells, the products of inflammation, accumulate in the alveoli, and minute bronchi crowd upon each other, becoming densely packed, and thus, by their mutual pressure, they bring about their own decay, as well as that of the lung textures, by interfering with their nutrition, the alveolar walls being also themselves damaged by the inflammatory process."

The position of the catarrhal pneumonia resulting in the above changes is usually at the apex, but it may occur at any portion of the lungs, or a whole lung becomes infiltrated, and undergoes the cheesy degeneration (phthisis florida).

SYMPTOMS. Pneumonic phthisis occurs in three forms, the chronic, the subacute, and the acute.

Chronic Form.—The origin is rather insidious, the individual being susceptible to "colds," or "catarrhs," on the slightest exposure; gradually a persistent cough, with the expectoration of mucopus, is established, each severe cold being accompanied with chill, fever, pain in the chest, and either slight hemorrhage or blood-streaked sputa. Finally, the catarrhal symptoms become persistent, with morning chills, evening fevers, and rather profuse night sweats, distressing cough, profuse muco-purulent sputa, containing the bacilli, great weakness and exhaustion, loss of appe-

tite and feeble digestion, the symptoms growing persistently worse, death occurring from exhaustion after one or two years' duration.

Subacute Variety.—History of an acute attack of pneumonia of one or two weeks' duration, followed by a decided improvement, but not complete recovery. After a lapse of some weeks or months, symptoms of pulmonary softening begin, destroying the lung structure and forming cavities, accompanied by chills, fever, night sweats, emaciation, cough, muco-purulent and blood-streaked expectoration containing the bacilli, the patient dying from exhaustion within a year.

Acute Variety.—The so-called phthisis florida, runs a rapid course, beginning either as a croupous or catarrhal pneumonia, involving the whole of one or part of both lungs, associated with rapid loss of flesh and strength, high but variable temperature, 103 degrees to 105 degrees F., with remissions, profuse night sweats, shortness of breath, severe cough, profuse, purulent, and blood-streaked sputa containing the bacilli, loss of appetite, and feeble digestion, the patient succumbing in a few weeks or months from exhaustion.

A decided remission in the local and general symptoms of the acute variety may occur, the disease afterward pursuing a more chronic course.

Inspection. Shows deficient respiratory movements of the diseased portion of the lungs.

Palpation. Increased vocal fremitus over the consolidated lung tissue and cavities.

Percussion. The percussion note varies from a slight impairment of the normal note to dullness, and when cavities are formed, associated with scattered points of the tympanitic or hollow note. If the cavities communicate with a bronchial tube the cracked-pot or cracked-metal sound is elicited. If the cavities are filled with pus the percussion note is dull. If the pus be expelled, the tympanitic or cracked-pot sound returns.

Auscultation. The vesicular murmur is unimpaired in those parts free from disease; it is feeble or indistinct if many bronchioles are obstructed; and is harsh or blowing if the bronchioles are narrowed. The inspiratory sound will be jerking, and the expiratory sound prolonged and blowing when the lung has lost its elasticity. Associated with the impaired vesicular murmur is a fine, dry, crackling sound (crepitation), appearing at the end of inspiration. If bronchitis be associated, large and small

moist or bubbling rales are also heard during respiration. When cavities form, either bronchial or broncho-cavernous respiration is heard, associated with more or less distinct gurgling rales. If the cavity be free from pus and have rather firm walls, the breathing is more amphoric in character.

DIAGNOSIS. Catarrhal bronchitis has many points of resemblance to pneumonic phthisis. The subsequent course of the latter, with the high temperature, prostration, emaciation, sputa containing bacilli, and physical signs will prevent error.

Acute fibrinous and catarrhal pneumonia, often after a course of two or three weeks, show the bacilli and yet are not recognized as tuberculosis. It is a safe rule of practice to suspect tuberculosis and examine daily for the bacilli in all cases of pneumonia that show the least tendency to linger, and particularly where there are chills and a remittent temperature record.

PROGNOSIS. Acute variety, the phthisis florida, usually terminates fatally within a few months.

The subacute and chronic varieties may, under judicious treatment and favorable hygienic conditions, be arrested, the caseous matter partly expectorated and partly absorbed, leaving more or less loss of structure, cicatricial tissue supplying its place, which after a time contracts, causing more or less retraction of the chest walls.

Cases not properly treated, either from carelessness or poverty, succumb after a year or two.

THE TREATMENT.

The early and persistent application of these principles cures many cases, mitigates suffering, and lifts the clouds of despondency from many a sad heart. Living witnesses are numerous, whose lives are veritable evidences of the efficiency of applied Osteopathy. Its possibilities are beyond computation, wonderful, always a source of relief, often effectual in curing. Treatment applied every other day, persistently and intelligently, works wonders in the end. Take courage.

III. TUBERCULAR PHTHISIS.

SYNONYMS. Tuberculosis; consumption; incipient phthisis; chronic phthisis; chronic ulcerative phthisis.

DEFINITION. A chronic pulmonary disease caused by the bacillus tuberculosis, resulting in the deposition of tubercle in the

lung structure, which in turn undergoes ulceration and softening, which results in a septic infection, characterized by progressive failure of health, fever, cough, dyspnœa, emaciation, and exhaustion.

CAUSES. Hereditary and acquired susceptibility to the influence of the bacillus tuberculosis. It is questionable if an individual is born with pulmonary tuberculosis, or makes his advent with tissues that are a congenial soil for the growth and ravages of this wide-spread germ. Amongst the acquired causes are syphilis, alcoholism, chronic nephritis, certain occupations, and living in damp, overcrowded, dark, and illy ventilated locations. Debility following an attack of influenza predisposes to the deposition of tubercle.

PATHOLOGICAL ANATOMY. Tubercle is a grayish-white, translucent, and semi-solid granulation, about the size of a millet seed, most commonly deposited in the walls of the bronchioles, exciting a low form of inflammation, the result of its own death. The masses of tubercle soon undergo softening (cheesy transformation); the lung structure is secondarily affected, undergoes softening, which results in more or less destruction of the tissue, whence cavities are formed. The inflammation may extend to the small arteries, causing hemorrhage. The deposit of tubercle is generally at one of the apices, and "once present in an apex, the disease usually extends in time to the opposite upper lobe; but not, as a rule, until the apex of the lower lobe of the lung first affected has been attacked. Lesions of the base may be primary, though this is rare." Depositions may also occur in the brain, intestines, and liver. The pleura is usually the seat of a chronic inflammation (dry pleurisy, tubercular), resulting in the obliteration of the pleural cavity.

SYMPTOMS. The symptoms correspond closely to the stages of deposition, of softening, septic infection, and of the formation of cavities. The development is insidious, with increasing dyspepsia and anaemia, the loss of appetite, distress after meals, and feeling of weakness, often misleading the patient and physician for some time until the occurrence of an irritable heart, a slight, dry hacking cough, referred to the throat or stomach, scanty, glairy expectoration, gradual loss of weight, impaired muscular strength, pallid appearance, and a more or less copious haemoptysis. Pain, sharp in character, below the clavicles, is often present. These symptoms are characteristic of the development of the disease.

The beginning of softening is announced by increased cough, freer expectoration, showing under the microscope the bacilli, dyspnœa increased on exertion, morning chills, evening fever and night sweats—the so-called hectic fever, diarrhea, increased emaciation and weakness, the patient, however, continuing very hopeful.

With the formation of the cavities, the cough is more aggravated, with profuse and purulent expectoration, at times containing yellow striae, the amount depending upon the number and size of the cavities; haemoptysis is not common at this stage; the pulse rapid and weak, increased hectic, burning of the soles and palms, copious night sweats, greater debility and emaciation, with œdema of the feet and ankles, denoting failure of the circulation, death soon following from asthenia, the mind clear and hopeful to the end.

INSPECTION. First Stage.—Often shows slight depressions in the supra-clavicular, and at times in the infra-clavicular regions.

PALPATION. Second Stage.—The vocal fremitus is slightly increased.

PERCUSSION. First Stage.—Slight impairment of the normal percussion resonance can sometimes be elicited. Second Stage.—The resonance is impaired, and may be even dull. Third Stage.—Dullness, with circumscribed spots of the amphoric, or tympanitic or cracked-pot sound.

AUSCULTATION. First Stage.—Inspiration jerky, expiration prolonged, the pitch higher than normal, the inspiration associated with crackling rales. Second Stage.—Vesiculo-bronchial breathing, associated with subcrepitant and large and moist or bubbling rales. Third Stage.—Bronchial, broncho-cavernous, and cavernous respiration, associated with large and small moist or bubbling and localized gurgling rales.

Bronchophony in its various degrees is associated with the second and third stages of tuberculosis.

COMPLICATIONS. Tubercular diseases of the brain, larynx, pleura, intestines, and peritoneum; perineal abscess leading to fistula, endocarditis, and myocarditis.

DIAGNOSIS. The early diagnosis of tubercular phthisis rests mainly on the history, together with the symptoms and physical signs. In the first stage it is often mistaken for dyspepsia, anaemia, malarial fever, or disease of the heart; if the bacilli can be found in the sputum the diagnosis is settled.

PROGNOSIS. In the main unfavorable, although under proper treatment, change of climate and like favorable conditions, life may be prolonged for years.

TREATMENT. See Fibroid Phthisis.

IV. FIBROID PHTHISIS.

SYNONYMS. Chronic intestinal pneumonia; cirrhosis of the lungs; Corrigan's disease.

DEFINITION. A hyperplasia (thickening) of the pulmonary connective tissue, resulting in atrophy and degeneration of the vesicular structure, associated with bronchial inflammation; characterized by cough, profuse expectoration containing the bacillus tuberculosis, fever, emaciation, and ultimately death by asthenia.

CAUSES. Hereditary predisposition; inhalation of irritants and associated with certain occupations, such as stone cutters, grinders, etc. Following lobar pneumonia; chronic bronchitis; alcoholism; syphilis; chronic nephritis.

PATHOLOGICAL ANATOMY. Thickening of the bronchial mucous membrane and dilatation of the air tubes; hyperplasia of the pulmonary connective tissue, resulting in the compression and consequent destruction of the vesicular structure, which is assisted by the contraction of the newly formed tissues. Sooner or later catarrhal pneumonia results, the product undergoing the cheesy degeneration, cavities being formed, and as a result of the long continued suppuration, tubercular depositions occur, hastening the destruction of the lung tissue.

Professor Da Costa has reported a number of cases of "grinders' phthisis," in whose sputum was found the "bacillus tuberculosis," and in whose family history there were no traces of consumption.

SYMPTOMS. The course is chronic, beginning as a bronchial catarrh, worse in winter, better in summer, when, after several years, the cough becomes more continuous, the expectoration freer and muco-purulent, containing the bacillus tuberculosis in large numbers, hectic fever develops, night sweats, dyspnœa, and rapid emaciation, soon followed by œdema of the feet and ankles, the result of failing circulation, death occurring by asthenia.

INSPECTION. Depression of the chest walls.

PERCUSSION. Impaired resonance, followed by dullness,

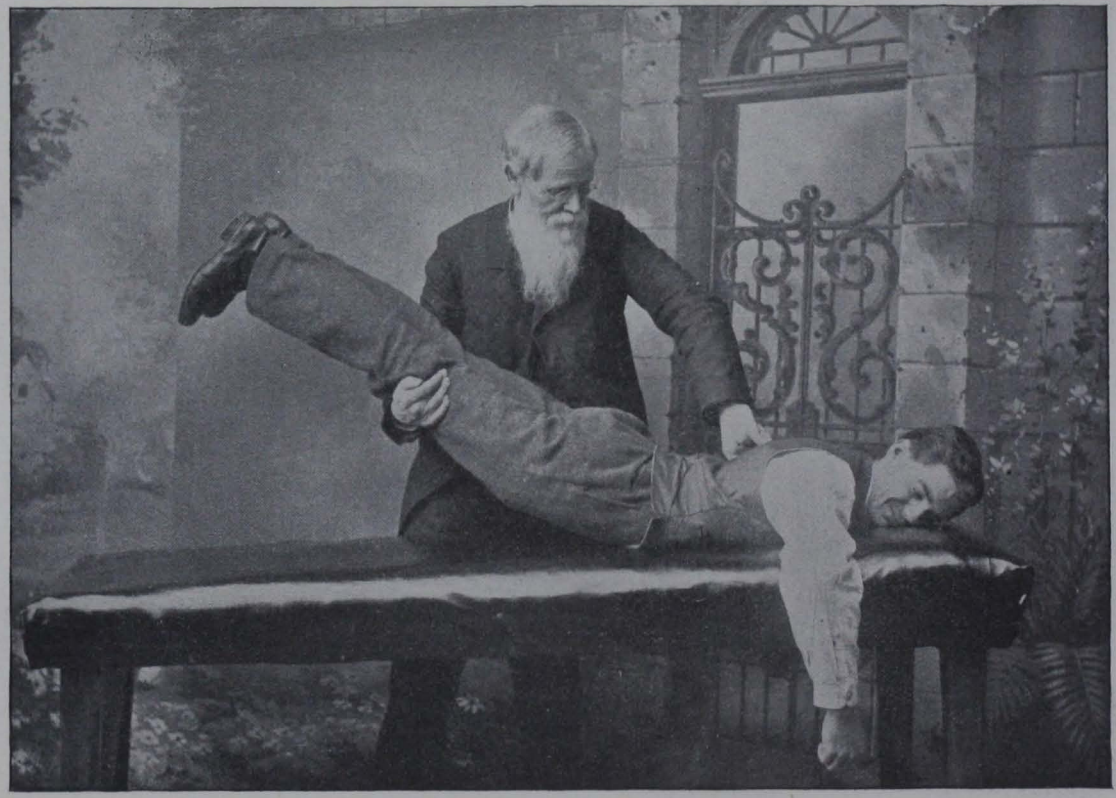


PLATE XXXIX.—Dorsum Treatment of Kidneys.

with irregular spots of amphoric or tympanitic percussion note over the points of depression.

AUSCULTATION. First Stage.—Vesiculo-bronchial, or harsh respiration associated with large and small, moist or bubbling rales, followed by bronchial, broncho-cavernous, and cavernous respiration, with circumscribed gurgling rales.

DIAGNOSIS. Beginning as a bronchial catarrh, slowly progressing, with the remission of the symptoms during the summer months, finally becoming progressively worse, the discovery of the bacilli in the sputum, with the formation of cavities, and symptoms of asthenia, are the chief points in the diagnosis.

PROGNOSIS. The duration of fibroid phthisis is most protracted, six to twelve years being the average duration; death, however, is the inevitable termination.

Professor Da Costa has records from one hundred deaths from "grinders' consumption" whose average life was twelve years.

THE TREATMENT.

To presume to assert that Osteopathy in any way favorably affects tuberculosis at once brings down upon the head of the manipulator supreme contempt. The habit of taking medicine (although without a cure) is so deeply rooted in the mind, that to pretend to benefit anybody otherwise seems the height of supreme ridiculousness to nearly everybody! When it is recognized that blood "is the life of man," and that it must be kept up to a normal and perfect standard, and that this can only be done by uninterrupted circulation and contact with oxygen, and that for the most part in the lungs, there will not be so much wonder at the idea of adjustment of the system so to itself as to promote it; and there will be less opposition manifest along these lines. To keep up a normal circulation means health, and however prone by heredity to take on this affection, from undue exposure or otherwise, a constant regard to the circulation and to the respirations will be had, and early promotion of these two essentials will ward off the disease; and when disease is noticed, especially in its early stages, many cases can be permanently cured, and others greatly relieved. The whole system should be adjusted to itself as often as twice or three times a week, and the patient instructed how to breathe correctly, to live long on earth. The various manipulations recommended to promote and keep up a normal circulation should be repeatedly and scientifically applied, ad libitum.

DISEASES OF THE PLEURA.

PLEURISY.

SYNONYMS. Pleuritis; "stitch in the side."

DEFINITION. A fibrinous inflammation of the pleura, either acute, subacute, or chronic in character, occurring either idiopathically or secondarily; characterized by a sharp pain in the side, a dry cough, dyspnœa, and fever. It may be limited to a part, or may involve the whole of one or both pleural membranes.

CAUSES. Idiopathic pleuritis is said to be due to cold and exposure, to injuries of the chest walls, or the result of muscular exertion. Tuberculosis is the cause of a few acute pleurisies. Secondary pleuritis occurs during an attack of pneumonia, pericarditis, rheumatism, variola, scarlatina, measles, Bright's disease, or puerperal fever. Chronic pleurisy follows an acute attack or is the result of tuberculosis, Bright's disease, or alcoholism.

PATHOLOGICAL ANATOMY. The course pursued by an inflammation of a serous membrane is hyperaemia followed by exudation of lymph, the effusion of fluid, its absorption, and the adhesion of the membranes. The first or dry stage is a hyperaemia or diffused irregular redness of the membrane, with little specks of exudation. The second stage is characterized by the copious exudation of lymph, more or less completely covering the membrane, giving it a dull, cloudy, or shaggy appearance. If the inflammation ceases at this point, it is termed dry pleurisy. The third, or stage of effusion, is characterized by the pouring out of a semi-fibrinous liquid, more or less completely filling and distending the pleural cavity, and floating in the fluid are fibrinous flocculi, blood, and epithelial cells. Absorption of the fluid and more or less of the exudative lymph soon occurs, the unabsorbed portion becoming organized, forming adhesions which obliterate the pleural cavity. The effusion, if on the right side, pushes the heart further to the left; if on the left side, the heart is displaced to the right, the impulse often being seen to the right of the sternum. The lungs are also compressed and displaced upward and against the spinal column, and, on removal of the fluid, expand again, except in cases of chronic pleurisy, when the functional activity of the pulmonary structure is more or less permanently impaired.

Chronic pleurisy results when the fluid is not absorbed or

when it is effused into the cavity in a slow and insidious manner. The membrane is irregularly thickened, with firm adhesions, fluid being found in the meshes; depressions of the thoracic walls also occur. The fluid may be serum, pus (empyema), or pus and blood. Openings may form, through which there is a permanent discharge, either externally (fiistulous empyema) or into the bronchi, or, rarely, into the bowels.

Symptoms. Acute Variety.—Begins with a chill, followed by a sharp lancinating pain (stitch) near the nipple or in the axilla, aggravated by coughing and breathing, associated with slight tenderness on pressure. The respirations are rapid and shallow, 30-35 per minute, a short, dry, hacking cough, moderate fever, compressible pulse, 90-120. With the effusion of liquid the dyspnœa becomes aggravated, the cough more distressing, the cardiac action embarrassed, the countenance wearing an anxious expression, the patient usually lying on the affected side. With the absorption of the fluid the symptoms gradually ameliorate, convalescence being more or less rapid.

Subacute Variety.—Begins insidiously after cold, exposure, and fatigue in those enfeebled. Patients usually complain of a sense of weariness, shortness of breath, aggravated on exertion, evening fever, followed by night sweats, short, harassing cough, none or very scanty sputum; the pulse is small, feeble, but frequent, 100-120 beats per minute. The characteristic pain in the side is usually wanting.

Chronic Variety.—Irregular chills, fever, night sweats, dyspnœa, palpitation, embarrassed circulation, with more or less prostration.

Inspection. First Stage.—Deficient movement of the affected side, on account of the pain induced by full breathing. Second Stage.—Bulging or fullness of the affected side, with obliteration of the intercostal spaces and displacement of the cardiac impulse.

Palpation. Second Stage.—Vocal fremitus feeble or absent over the site of the effusion, exaggerated above the site of the fluid. Rarely, fluctuation may be obtained.

Percussion. First Stage.—May be slightly impaired. Second Stage.—Dullness or even flatness over the site of the effusion; tympanitic percussion note above the fluid.

Auscultation. First Stage.—Feeble vesicular murmur over the affected side, the patient breathing superficially, to prevent the pain; a friction sound, slight and grating or creaking,

becoming louder as the exudation of lymph increases, limited usually to the angle of the scapula of the affected side, rarely heard over the entire side, accompanies the respiratory movements. Second Stage.—Feeble or absent vesicular murmur on the affected side, depending upon partial or complete compression of the lungs by the fluid. Above the fluid puerile breathing, and just at the upper margin of the fluid, a friction sound may be heard. The vocal resonance is diminished or absent over the site of the fluid and markedly increased above, aegophony being present at the upper margin of the fluid. With the absorption of the fluid the vesicular murmur gradually returns, associated with a moist friction sound.

DIAGNOSIS. Acute pneumonia is often mistaken for the effusion stage of pleurisy. The points of distinction are, in pneumonia there are the pronounced chill, high fever, characteristic sputa, bronchial breathing, exaggerated vocal fremitus and resonance, and no displacement of the heart, the reverse occurring in pleurisy.

Enlargement of the liver may be mistaken for pleurisy with effusion, the chief point of distinction being that, in enlargement of the liver, the superior line of dullness is depressed upon full inspiration, while in pleurisy with effusion inspiration does not modify the location of the dullness.

PROGNOSIS. Idiopathic pleurisy usually terminates in recovery within three weeks. Pleurisy the result of constitutional causes has its prognosis modified by the condition with which it is associated. Empyema, unless the result of a diathesis, terminates favorably. Double pleurisy is unfavorable. The etiological factor of tuberculosis must always be borne in mind in making a prognosis in pleurisy, whether acute or chronic.

THE TREATMENT.

The same as for Asthma, the object being to take off the pressure. Daily manipulations, or even twice a day in acute forms, will relieve all the difficulty at once, within a day or two, and disperse the effusion so as that all further trouble is removed —arrested. The usual six weeks' suffering is aborted. The pleura acting as an assistant to expiration, it can be easily understood why breathing is limited to short and quick exhalations, attended with pain, in infiltrations of this organ. The closure of the lymph tubes causes accumulation, lessens action of the muscles of respiration, crowds other organs, fills the chest cavity, holds taut the intercostal muscles, and as the accumulation

increases the pain keeps intensifying, until partial paralysis of sensory and sympathetic nerves ensues. Relaxation allows transfusion and exudation and gradual absorption of fluid. The remedy with the Osteopath is, take off the pressure and open up the outlets (the channels that are closed, causing the accumulation), and the effects at once subside—the relief comes at once. Any objection to this? Why didn't this occur to the physician a long time ago? Why doesn't it occur to him now? The raising of the arm on the side of the pain, as STRONGLY as the patient can well bear, whilst a long and as deep inspiration as possible for the patient to bear is made, and a strong pressure is made with the fingers at the side of the spine about the seventh and eighth dorsal vertebrae, held there a moment, and then quickly returned to the side, clears out the intercostal spaces of venous blood, opens the tubes in the pleura itself, relaxes the muscular fibers of the two walls of the pleura, and disengorgement takes place as if by magic—the condition is changed, a cure is effected. One or two treatments usually suffice to clear out the whole of the obstruction. The general treatment of the upper portion of the body is well enough to make, for equalization of all the forces is an essentiality to right the malcondition of the blood and other fluids involved. In chronic pleurisy a repetition of the same treatment soon restores the parts to a normal state. These treatments should be mild, slowly performed, and thoroughly done, and the results are invariably satisfactory. Every other day is often enough to treat the patient for chronic pleurisy. The head, neck, chest and spine should receive particular and special attention each time. Do not fail to use vibratory manipulations over the painful parts. Rapid circular vibratory movements are most effectual.

HYDROTHORAX.

SYNONYM. Dropsy of the pleura.

DEFINITION. The effusion of the fluid into the pleural cavities (bilateral), the result of a general dropsy from renal or cardiac disease.

PATHOLOGICAL ANATOMY. More or less clear serous fluid in both pleural sacs, compressing the lung. No signs of inflammation are present.

SYMPTOMS. Following dropsy of the abdomen occurs

dyspnœa, with signs of deficient blood aeration, both lungs being compressed.

PALPATION. Absent vocal fremitus over the site of the fluid.

PERCUSSION. Dullness over the site of the fluid.

AUSCULTATION. Absent vesicular murmur over the site of the fluid.

DIAGNOSIS. Easily determined by association of the symptoms with a general dropsy.

PROGNOSIS. Controlled by the cause producing the general dropsy.

THE TREATMENT.

The treatment for the affections causing it is the rational course to pursue. If the heart is affected, direct attention to it. If the kidneys are diseased, use the means provided therefor. As in all cases of dropsy, the effusion comes from obstructed venous return circulation. That may result from nerve pressure, arresting capillary circulation, and the watery portions (70 per cent.) of the blood exuding through the walls of the capillaries; or, if from engorgement of veins, the broken-down tissue can not escape through the lymph canals, and the fluid increases, hence the dropsy of the pleura. The relief may be afforded in two ways: One by paracentesis thoracis, and the other by general treatment, to open the outlets through which the fluids normally escape. The pressure is due to contraction of the chest walls, and preventing escape from the intercostal veins into the azygos veins, thus preventing chest expansion, hence closure of outlets—tubes. The treatment must be directed especially to the respiratory muscles, to the ribs, clavicles, neck and spinal and abdominal muscles. Also see to the veins of the lower limbs (the saphenous) and lumbar and sacral nerves, and especially relieve the kidneys, at the renal splanchnic—the lower splanchnic area, twelfth dorsal. Institute stated acts of respiration and see to it that all the large veins are properly relieved of their accumulated contents. The liver should not be slighted. Frequent bathing of the whole body with tepid water, to keep the skin healthy, is essential.

The tissue elements should be supplied also. The potass. chloride is one of the elements needed to aerate the blood. The sodium sulph. becomes the remedy when the areolar tissue is involved, and the sodium chloride is indicated where the subcutaneous tissue is involved; calcium phos., if from non-assimilation of food; ferric phos., if from loss of blood; calcium fluoricum, if

from heart disease, dilatation of cavities; and potass. sulph. after scarlatina and where there is deficiency of perspiration.

These belong to no system of medication, are not medicines, but simply elements of the system, which, when deficient, should be supplied.

It will be found of great importance to know when to supply these elements, and to be informed on this subject the reader should study Schussler's "Biochemic Treatment," which may be obtained at almost any Homeopathic pharmacy in the country, by M. Docetti Walker, M.D.; compiled by Drs. Boericke and Dewey, San Francisco, California. Luyties' Hom. Phar. Co., 306 N. Broadway, St. Louis, Mo., sell it. $1.50.

Our success depends largely on what we know. The physician's quiver should be full of arrows. This life is one of warfare with the enemy that has great power, and persistently claims his victim, and at the last "mows him down." All are his subjects. Our time is set. He is the executioner. Be ready.

PNEUMOTHORAX.

SYNONYMS. Air in the pleural cavity; hydropneumothorax.

DEFINITION. The accumulation of air in the pleural cavities, with the consequent development of inflammation of the membranes; characterized by sharp pain, followed by rapidly developing dyspnœa and cough.

CAUSES. Generally the result of tubercular phthisis, causing perforation of the pleura. Perforation may take place from the pleura into the lung, in connection with empyema or abscess of the chest wall. Direct perforation from without, by laceration of a fractured rib or severe contusion.

PATHOLOGICAL ANATOMY. The gas in the pleural cavity consists of oxygen, carbon anhydride, and nitrogen in variable proportions. It may fill the pleural sac completely, compressing the lung, or is sometimes limited by adhesions. The gas tends to excite inflammation, the resulting effusion being either serous or purulent.

SYMPTOMS. Symptoms of pneumothorax, the result of perforation, are sudden or sharp pain in the side, intense dyspnœa, attended with symptoms of collapse, coldness of the surface, and cold sweats. These symptoms, in many instances, follow a severe or violent paroxysm of coughing. In severe cases there is never

a moment's cessation of the acute pain and distressing dyspnœa, causing orthopnœa from the onset until death.

INSPECTION. Enlargement of the affected side, the intercostal spaces being widened and effaced or even bulged out so that the surface of the chest is smooth. Respiratory movements of the affected side are diminished or absent.

PERCUSSION. Immediately after the rupture the percussion note is hyper-resonant, or even tympanitic or amphoric in quality. If the amount of air in the pleural cavity becomes extreme, there is dullness on percussion, associated with a feeling of great resistance or density. When effusion of blood occurs, dullness is observed over the lower part of the chest, hyper-resonant or tympanitic percussion note over the upper portions of the chest, these sounds changing as the patient changes position.

AUSCULTATION. The normal vesicular murmur may be diminished or absent. The typical amphoric respiratory sound is heard when the fistula is open, usually associated with a metallic echo. Metallic tinkling, or the bell sound, is sometimes distinctly produced by breathing, coughing, or speaking, after the development of inflammation of the pleura. The vocal resonance may be diminished or absent, or, rarely, it may be exaggerated, with a distinct metallic echo. After the development of inflammation in the pleura, suddenly shaking the patient gives rise to a splashing sensation, the succussion sound, if both air and fluid are present in the pleural cavity.

PROGNOSIS. When occurring as the result of tuberculosis, the prognosis is extremely unfavorable; rarely, the fistulous opening being inclosed by inflammatory action; the case then becomes one of chronic pleurisy.

THE TREATMENT.

The first thing to be done is to use the aspirator, to relieve the pressure. Close up the orifice in which air enters, expand the chest walls on both sides and use the arm movements, inhalations and dorsal manipulations to induce absorption of the air. If the pleura is inflamed, treat the patient as directed for it. The application of the principles of Osteopathy is to be studied in all cases, and skill is required in this, as in all other sciences, to succeed. The desideratum to achieve lies in removing the pressure, as every one should understand. Raising the clavicles, arms, ribs, diaphragm, and the vibratory manipulations over the part affected result most satisfactorily, generally. The cases are rare, but when met with, must be met understandingly or censure is sure to be heaped upon you.

PLATE XL.—Scapula, Dorsum and Shoulder Treatment.

DISEASES OF THE CIRCULATORY SYSTEM.

The methods employed in making a physical examination of the heart are: 1. Inspection. 2. Palpation. 3. Percussion. 4. Auscultation.

INSPECTION.

INSPECTION indicates the exact point of the cardiac impulse, and the presence or absence of any abnormal pulsations or any change in the form of the praecordium. Normally, the impulse is visible only in the fifth interspace, midway between the left nipple and the left border of the sternum, its area covering about one square inch, most distinct in the thin, while often barely seen in the very fleshy; often displaced downward by full inspiration and elevated by complete expiration. Disease may alter the position and area of the impulse.

The position of the impulse is moved to the right by left pleuritic effusions; downward by cardiac hypertrophy or pulmonary emphysema; upward by a pericardial effusion.

The area of the impulse is changed and enlarged by pericardial adhesions, cardiac dilatation, or hypertrophy.

PALPATION.

PALPATION confirms the observations of inspection, and also determines the force, frequency, and regularity of the cardiac impulse. The force of the impulse is diminished by cardiac dilatation, fatty and fibroid degenerations of the heart, emphysema, pericardial effusion, and adynamic diseases. The impulse is increased by cardiac hypertrophy, during the first stage of endocarditis and pericarditis, functional cardiac disturbances and sthenic inflammations.

PERCUSSION.

PERCUSSION will determine the boundaries of the superficial and deep cardiac space, the so-called praecordium. It is essential that the upper, lower, and two lateral boundaries of the pericardial region be memorized, to-wit: superior boundary, the upper edge of the third rib; the lower boundary is a horizontal line passing through the fifth intercostal space; the left lateral boun-

dary is about or a little within a vertical line passing through the nipple, the linea mammalis; and the right lateral boundary is an imaginary vertical line situated one-half an inch to the right of the sternum. These boundaries vary somewhat in health, but are sufficiently accurate for all practical purposes.

The superficial cardiac space represents that portion of the heart uncovered with lung; it is triangular in form, its apex being the junction of the lower border of the left third rib with the sternum, its area not exceeding two inches in any direction. The superficial space is increased by cardiac hypertrophy, dilatation, or pericardial effusion. Diminished at the end of full inspiration or by emphysema.

The deep cardiac space represents that portion of the heart covered by lung, and extends from the upper border of the third rib to the lower edge of the fifth interspace, and from half an inch to the right of the sternum to near the left nipple. It is increased by hypertrophy or dilatation of the heart, left pleuritic effusion, and apparently increased by consolidation of the anterior border of the investing lung.

AUSCULTATION.

AUSCULTATION indicates the character of the normal cardiac sounds, and the point at which they are heard with greatest intensity, and should be thoroughly familiarized if abnormal sounds are to be fully appreciated.

The ear or stethoscope applied to the praecordium distinguishes in health two sounds, separated by a momentary silence —the short pause, and the second sound followed by an interval of silence—the long pause.

The first sound, corresponding to the contraction of the heart—the systole—is louder, longer, and of a lower pitch and a more booming quality than the second sound, and has its point of greatest intensity at the cardiac apex or a little to the left. It corresponds closely in time to the pulsations as felt in the carotid or radial arteries.

The second sound is shorter, weaker, and higher in pitch than the first sound, and has a clicking or valvular quality, having its point of greatest intensity at the second right costal cartilage and a little above, and corresponds to the closure of the aortic and pulmonary valves. The sound made by the closure of the tricuspid valves is best isolated at the ensiform cartilage. The sound made by the closure of the pulmonary valves at the third left costal cartilage.

The extent of surface over which the cardiac sounds are heard varies, according to the size of the heart and the condition of the adjacent organs for transmitting sounds. These sounds may be altered in intensity, quality, pitch, seat, and rhythm, or they may be accompanied, preceded, or followed by adventitious or new sounds, the so-called endocardial or cardiac murmurs. The intensity is increased by cardiac hypertrophy, irritability of the heart, or consolidation of adjacent lung structure. The intensity is diminished by cardiac dilatation or degeneration, during the course of adynamic fevers, emphysematous lung overlapping the heart, or pericardial effusion.

The quality and pitch of the first sound may be sharp or short and of higher pitch when the ventricular walls are thin, or have undergone beginning fibroid change, the valves being normal; its pitch and quality are also raised during the course of low fevers. The second sound becomes duller and lower in pitch when the elasticity of the aorta is diminished or the aortic valves thickened. Either or both sounds have a more or less metallic quality in irritable heart and during gaseous distention of the stomach.

The seat of greatest intensity of the cardiac sounds is changed by displacement of the heart, pleuritic effusion, pericardial effusion, and abdominal tympanites.

The rhythm is often interrupted by a sudden pause or silence, the heart missing a beat, or the sounds are irregular, confused and tumultuous, the result of organic changes in the cardiac muscle, valves, or orifices; or a reduplication of one or both sounds of the heart may occur.

The adventitious cardiac sounds or murmurs are of two kinds, those made external to the heart, as pericardial, exocardial, or frictional murmurs, and those made within the cardiac cavity, endocardial murmurs.

Pericardial murmurs, or friction sounds, are made by the rubbing upon one another of the roughened surfaces of the pericardial membrane during the early stages of inflammation. The sounds have a rubbing, creaking, or grating character, and are differentiated from a pleural friction sound by their being limited to the praecordium, synchronous with every sound of the heart, and not influenced by respiration. They are distinguished from an endocardial murmur by their superficial rubbing, creaking, or grating character, and by not being transmitted beyond the limits

of the heart, either along the course of the vessels, or to the left axilla, or back.

Endocardial murmurs are of two kinds, to-wit: organic and functional.

Functional endocardial or blood murmurs are the result of changes in the natural constituents of the blood. Their character is soft, they are heard most distinctly at the base to the left of the sternum, during the systole, are not transmitted beyond the limits of the heart, either to the left axilla or the back, and are associated with general anaemia.

Organic endocardial murmurs are produced by blood currents pursuing either a normal or an abnormal direction.

In health there are two direct blood currents upon each side of the heart, to-wit: the current from the left auricle to the left ventricle, the mitral direct current; the current from the left ventricle to the aorta, the aortic direct current; the current from the right auricle to the right ventricle, the tricuspid direct current, and the current from the right ventricle to the pulmonary artery, the pulmonic direct current.

When, from disease, the valves are not properly closed, the blood is allowed to flow back against the direct current, producing abnormal blood currents, to-wit: when the mitral valve is incompetent, the blood flows from the left ventricle back into the left auricle during the cardiac systole, producing the mitral regurgitant or indirect current; when the aortic valves are incompetent, the blood is permitted to flow from the aorta into the left ventricle during the cardiac diastole, producing the aortic regurgitant or indirect current; when the tricuspid valves are incompetent, the blood flows from the right ventricle back into the right auricle during the systole, producing the tricuspid regurgitant or indirect current; when the pulmonary valves are incompetent, the blood flows from the pulmonary artery into the right ventricle, producing the pulmonic regurgitant or indirect current.

The mitral direct current occurs during the contraction of the left auricle, or just before the first sound of the heart and immediately after its second sound. The aortic direct current is produced by the contraction of the left ventricle, and occurs with the first sound of the heart. The tricuspid direct current occurs during the contraction of the right auricle, or just before the first or immediately after the second sound. The pulmonic direct current is produced by the contraction of the right ventricle, occurring during its first sound.

The mitral direct or presystolic murmur occurs before the first sound of the heart and immediately after the second sound. It is caused by a narrowing of the mitral orifice, has a blubbering quality, well imitated by throwing the lips into vibration by the breath, of a low pitch, and it has its seat of greatest intensity at the cardiac apex, and is not transmitted to the left axilla or to the base of the heart.

The mitral regurgitant or systolic murmur occurs with the first sound of the heart, resulting from the failure of the mitral valves to close the mitral orifice during the systole, in consequence of which the blood flows back, or regurgitates into the left auricle. It is usually of a blowing or churning character, and has its seat of greatest intensity at the cardiac apex, being well transmitted to the left axilla and inferior angle of the left scapula.

The aortic direct murmur occurs with the first sound of the heart. It is caused by a narrowing of the aortic orifice, has a rough or creaking character, is of high pitch, having its seat of greatest intensity in the second intercostal space, to the right of the sternum, and is well transmitted over the carotid artery.

The aortic regurgitant murmur occurs with the second sound of the heart, and is caused by the failure of the aortic valves to close the aortic orifice during the diastole, permitting the blood to flow back or regurgitate into the left ventricle. It is usually of a blowing or churning character and of low pitch, having its seat of greatest intensity over the base of the heart, and is well transmitted downward toward or below the cardiac apex. It is the only organic murmur produced in the left side of the heart which occurs with the second sound of the heart.

The tricuspid direct murmur occurs before the first sound of the heart, the result of the failure of the tricuspid valves to close by a narrowing of the tricuspid orifice, has a blubbering quality, and is low in pitch, having its seat of greatest intensity near the ensiform cartilage. This murmur is exceedingly rare.

The tricuspid regurgitant murmur occurs with the first sound of the heart, the result of the failure of the tricuspid valves to close the tricuspid orifice during the systole, thus allowing the blood to flow back or regurgitate into the right auricle. It is usually of a blowing or soft, churning character, having its seat of greatest intensify at the ensiform cartilage. This murmur is also very infrequent, and occurs mostly when the right ventricle is considerably dilated, and without the existence of any valvular disease.

The pulmonic direct murmur occurs with the first sound of the heart. It is generally connected with congenital lesions. It occurs at the same instant that the aortic direct murmur occurs, and is distinguished from the latter by its not being transmitted into the carotid artery, whereas the aortic direct murmur is always thus transmitted.

The pulmonary regurgitant murmur occurs, like the aortic regurgitant murmur, with the second sound of the heart. This murmur is exceedingly rare, and its presence is only positively differentiated from the aortic regurgitant murmur by the absence of aortic lesions and symptoms.

ACUTE PERICARDITIS.

DEFINITION. An acute fibrinous inflammation of the pericardium; characterized by slight fever, pain, praecordial distress, and disturbed cardiac action and circulation. If the inflammation be limited to the parietal or visceral layer, or to a part of either, it is termed partial or circumscribed pericarditis; if it involve the whole of both surfaces it is termed general or diffused pericarditis. The inflammation may be primary or secondary.

CAUSES. Primary pericarditis is rare, resulting directly from cold and exposure or injuries. Secondary pericarditis follows, or is associated with, rheumatism, influenza, scarlatina, variola, puerperal fever, tuberculosis, septicaemia, Bright's disease, gout, scurvy, and diabetes. It is frequently associated with pneumonia and pleuro-pneumonia, particularly in alcoholics.

PATHOLOGICAL ANATOMY. The same as of serous membranes in other situations. The morbid changes may be seen, as (1), acute plastic, or dry pericarditis (frequently tubercular), (2), pericarditis with effusion, sero-fibrinous, hemorrhage, or purulent.

Hyperaemia of the membrane, most marked on the visceral layer, followed by the exudation of lymph scattered in irregular patches, giving it a rough and shaggy appearance (dry pericarditis), followed by the effusion of a sero-fibrinous fluid, with flocculi floating in it, and at times mixed with blood. Rarely, the fluid is purulent.

The fluid and lymph undergo absorption with resulting adhesions identical with those described under pleurisy.

SYMPTOMS. Acute pericarditis may be well marked and still

present none of the characteristic subjective symptoms. It usually begins with rigors, fever of the remittent type, frequently nausea and vomiting, praecordial distress, and tenderness, acute shooting pains, increased by breathing and coughing, dry, suppressed cough, increased cardiac action, and sometimes violent palpitation. An attack of pericarditis secondary to an existing disease presents no marked symptoms other than those mentioned to indicate its onset. Attacks of nausea and vomiting occurring during the course of rheumatism, pneumonia, pleurisy, and nephritis, should call attention to the heart. Duration of this early stage from a few hours to a day or two.

Effusion Stage.—The symptoms of this stage are in keeping with the amount and rapidity of the effusion: praecordial oppression, tendency to syncope, dyspnœa, sometimes amounting to orthopnœa, dysphagia, hiccough, nausea and vomiting, feeble, irregular pulse, sometimes either melancholia, delirium, or acute maniacal excitement.

Absorption is generally rapid, the heart remaining "irritable" for a long time after. If, instead of absorption, the fluid accumulates, and life is not destroyed, the pericardial sac becomes dilated, chronic pericarditis resulting.

Inspection. Early Stage.—Excited cardiac action is evidenced by the impulse. Effusion Stage.—Feeble, undulatory or absent impulse, its position displaced upward, or, rarely, downward, bulging of the praecordium and protruding abdomen.

Palpation. Early Stage.—Excited or tumultuous impulse; pericardial friction fremitus rare. Effusion Stage.—Feeble or absent impulse, and if present its position is changed.

Percussion. Early Stage.—Normal. Effusion Stage.—Cardiac dullness enlarged vertically and laterally, and if considerable fluid, of a triangular shape, with the base of the triangle on a line with the sixth or seventh rib, extending from the right of the sternum to the left of the left nipple, narrowing as it proceeds upward to the second rib, or above, which represents the apex of the triangle. The shape of the dullness is sometimes altered by changing the position of the patient.

Auscultation. Early Stage.—Excited cardiac action, and usually a friction sound (exocardial murmur) synchronous with cardiac sounds and uninfluenced by respiration, but often increased by pressure with the stethoscope. Effusion Stage.—Cardiac sounds feeble and deep-seated at the cardiac apex, becoming louder and distinct toward the cardiac base. The friction

sound is sometimes heard at the cardiac base. If absorption occur, the above signs gradually give place to the normal, the friction sound returning, of a churning, or clicking, or grating character, gradually disappearing.

DIAGNOSIS. Endocarditis is often confounded with pericarditis, the points of distinction between which will be pointed out when discussing that affection. Cardiac hypertrophy or dilatation is sometimes confounded with pericardial effusion, the differences between which will be pointed out when discussing those affections. Hydro-pericardium may be mistaken for pericardial effusion; see that affection.

PROGNOSIS. Controlled by the severity of the inflammation, causes, and coexisting affections. If slight effusion, favorable. Death has quickly occurred when a large quantity of fluid has been rapidly effused, the patient being really drowned in his own fluid. Adherent pericardium is a frequent sequela.

THE TREATMENT.

The results of venous obstruction are seen here as in all other parts of the system. The obstruction is usually due to compression of the upper chest walls, due to contraction of the pectoral and intercostal muscular fibers, causing contraction of space for the action of the heart, hence venous obstruction and engorgement of the covering of the heart—pericardium. The most wonderful results attend the lifting off of the pressure, as indicated above—the only thing to do in the case. Such measures as seem indicated should be instituted, and gently, persistently applied, and relief is at once obtained. The medication used to promote circulation and to reduce inflammation needs proof of its efficacy before further trifling with life, waiting on a supposed probability of its curative powers. What else is needed than to open the channels closed by pressure, and let the pent-up foreign fluids be carried out, healthy arterial blood come in and supply the normal elements to build up the waste and repair the damages sustained? Think of it! Many a "heart failure" is recorded that need not have been, had Osteopathy been known and applied. Lift off the pressure. Let circulation proceed.

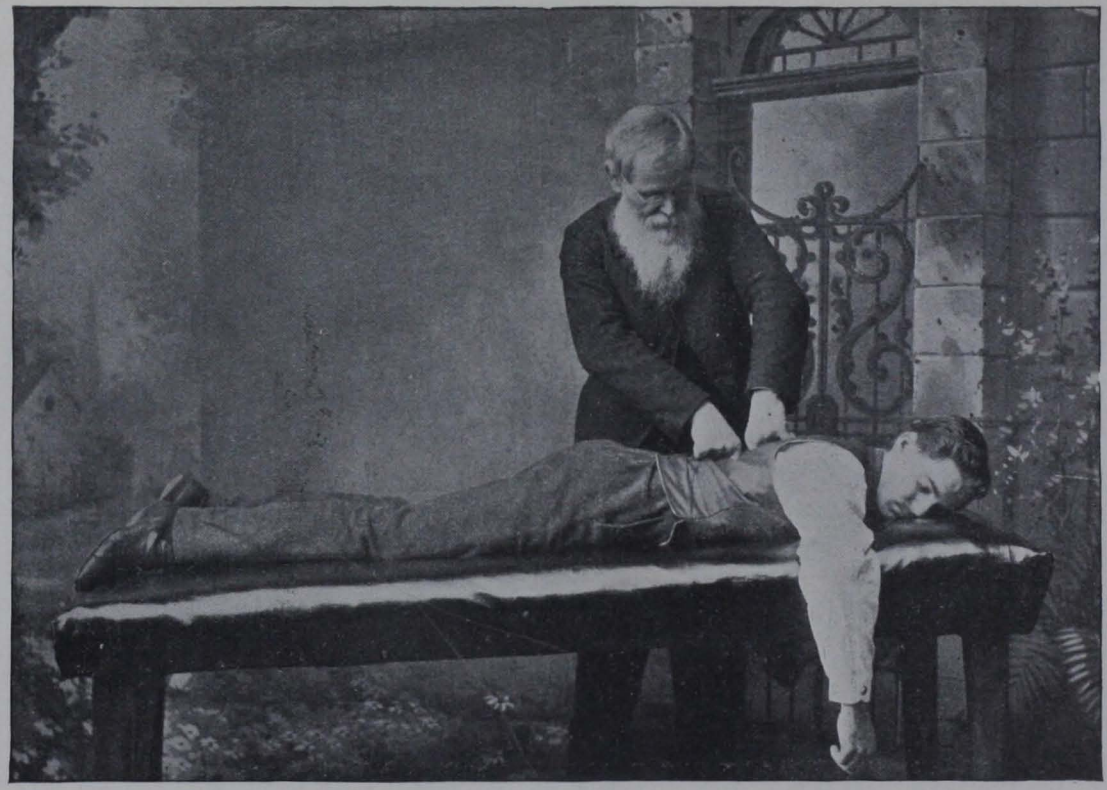


PLATE XLI.—Spinal Concussion, Knuckle Treatment.

CHRONIC PERICARDITIS.

DEFINITION. A chronic inflammation of the pericardium, with either distention of the sac by fluid or adhesions of the pericardium (adherent pericardium); characterized by impaired cardiac action and disturbances of the circulation.

CAUSES. Almost always the result of an acute attack.

PATHOLOGICAL ANATOMY. If the effusion be absorbed, the pericardial surfaces are agglutinated by several layers of lymph, which increase the thickness of the membranes half an inch or more, and the outer surface of the pericardium becomes adherent to the chest walls. If the fluid be not absorbed, it may progressively accumulate, distending the sac in all directions, displacing the diaphragm and interfering with the functions of the surrounding viscera, or a low grade of inflammation supervenes, the fluid becoming purulent, the disease terminating fatally after a variable period. As much as eight to ten pints of fluid have accumulated in the sac.

SYMPTOMS. Praecordial pain and distress, irregular, feeble cardiac action, dyspnœa aggravated by movement, and disturbed circulation. An agglutinated pericardium seriously increases the danger from an attack of any pulmonary inflammation.

INSPECTION. If the effusion be present, bulging of the praecordium and displacement of the impulse. If adhesions are formed between the pericardial surfaces as well as with the chest walls, inspection reveals depression of the praecordium, narrowing of the spaces, increased extent but displaced impulse, uninfluenced by deep inspiration, and recession of the intercostal spaces (systolic dimpling) and epigastrium with every systole of the heart, the result of the adhesions.

PALPATION. If effusion, displaced, feeble or absent impulse; if adhesions, displaced and tumultuous impulse; occasionally a pericardial fremitus is distinguished.

PERCUSSION. If effusion, the dullness has more or less the character described for acute pericarditis. If adhesions, the cardiac dullness is but slightly modified.

AUSCULTATION. If effusion, cardiac sounds feeble and deep-seated at the apex, louder and more distinct at the cardiac base. If adhesions, cardiac sounds are heard with equal distinctness in their several positions, associated with a rough friction sound (exocardial murmur).

TREATMENT. Same as for Acute Pericarditis.

HYDRO-PERICARDIUM.

SYNONYM. Pericardial dropsy.

DEFINITION. The accumulation of water in the pericardial sac, minus inflammation; characterized by praecordial distress, disturbed cardiac action, dyspnœa, and dysphagia.

CAUSES. Usually a part of a general dropsy; Bright's disease; sudden pneumothorax; pressure of an aneurism or other mediastina tumor; disease or thrombosis of the cardiac veins.

PATHOLOGICAL ANATOMY. The fluid may range in quantity from an ounce to one or two pints, and is of a clear, yellowish or straw-colored serum, at times turbid or bloody, and of an alkaline reaction. If the amount of fluid be large the sac is dilated, its walls thinned by the pressure, and has a sodden appearance.

SYMPTOMS. Dropsy of the pericardium is so generally associated with hydrothorax that the symptoms are but an aggregation of those attending upon that condition, to-wit: disturbed cardiac action, dyspnœa, dysphagia, dry cough, and feeble circulation. The physical signs are exactly those of the stage of effusion of pericarditis, minus a friction sound.

DIAGNOSIS. Pericarditis with effusion and hydro-pericardium present nearly the same signs and symptoms, a differentiation being possible only by a history of the case and the symptoms of the attack.

TREATMENT. Same as for Acute Pericarditis.

ACUTE ENDOCARDITIS.

SYNONYMS. Valvulitis; exudative endocarditis.

DEFINITION. An acute fibrinous inflammation of the serous membrane lining the cavity of the heart and forming its valves, in severe cases the chordae tendineae being involved, resulting in changes in the valves or orifices of the heart, or both; characterized by cough, dyspnœa, disturbed cardiac action, nausea, vomiting, and more or less marked febrile reaction. Acute endocarditis occurs in two distinct forms: plastic or simple exudative endocarditis; ulcerous or diphtheritic endocarditis.

CAUSES. Usually secondary to acute articular rheumatism, pleuritis, pneumonia, pericarditis, Bright's disease, scarlatina, influenza, and diphtheria. The association of acute endocarditis and chorea is frequent. While as yet no specific micro-organism

has been discovered, the view is gaining ground that it is a microbic affection.

PATHOLOGICAL ANATOMY. Inflammation of the endocardium is usually limited to the left side of the heart after birth, during foetal life the reverse being the case. The inflammation is limited or especially marked at the valvular portions of the endocardium, owing probably to the presence of fibrous tissue beneath the membrane in these situations, and to the strain which falls upon the valves during the performance of their functions.

Hyperaemia from congestion of the vessels beneath the membrane, with considerable swelling of the valves, the result of an exudation of lymph and serum beneath and on the free surface of the membrane covering the valves and chordae tendineae, resulting in the roughening of the surfaces and the agglutination of the mitral valves to each other, and of the aortic segments to the walls of the aorta, or the proliferation of the endocardial connective tissue, forming the nuclei of the so-called warty excrescences or vegetations, their size being increased by the deposition of fibrin from the blood within the cavities of the heart. These vegetations may be detached by friction, giving rise to emboli which may be washed by the blood current to the left side of the brain, or into the kidneys and the spleen.

In the ulcerative variety a process of softening takes place in the fibrinous deposits, leading to ulcerations and perforations.

SYMPTOMS. The affection is usually masked by the course of another disease until disturbances of the circulation direct attention to the heart. The onset is often by increase of temperature, praecordial distress, short cough, slight dyspnœa, more or less persistent vomiting, increased cardiac action, often rapid and tumultuous, with throbbing carotids and noises in the ear. As the inflammation progresses, the cardiac action and pulse decline in rapidity, with more or less congestion of the lungs and venous stasis.

AUSCULTATION. Shows a change in the character of the sounds or the development of murmurs at the various orifices, the character and points of distinction between which will be pointed out when discussing valvular diseases of the heart.

DURATION. Between one and three weeks.

DIAGNOSIS. Unless it is a rule of practice to always auscult the heart, many cases will pass unobserved or undetected. Pericarditis is distinguished from endocarditis by the character of the physical signs. In pericarditis the murmur or friction sound is

heard with either sound, is near to the ear and influenced by the pressure of the stethoscope, besides being associated with more or less alteration in the size and shape of the cardiac dullness, and is not transmitted, while in endocarditis the murmur takes the place of, or is associated with, the cardiac sounds, and is transmitted, with the absence of change or increased dullness on percussion. If embolism occur, a new set of symptoms develop; embolism of the kidneys causes sudden, deep-seated lumbar pain, with albuminuria and even haematuria; embolism of the brain, sudden palsies and sudden disturbance of consciousness; of the spleen, sharp pain and tenderness in the splenic region; of the skin, petechial or purpuric spots.

PROGNOSIS. Acute endocarditis is not very dangerous to life, hence a favorable prognosis may be given; regarding the ultimate results of valvular lesions, however, the prognosis is grave.

TREATMENT. The same as for Acute Pericarditis. Use the treatment that lifts off the pressure, which is the only thing to be done.

MALIGNANT ENDOCARDITIS.

SYNONYMS. Ulcerative endocarditis; septic, mycotic, and diphtheritic endocarditis.

DEFINITION. An acute septic inflammation of the lining membrane of the heart, with a strong tendency to ulceration; characterized by depression of the vital forces with more or less cardiac distress.

CAUSES. The specific micro-organism has not yet been determined. Frequently complicates pneumonia. Associated with acute rheumatism. Cases have been reported associated with or following influenza.

PATHOLOGICAL ANATOMY. The changes are those of acute endocarditis up to the development of the thickening of the endocardium lining the valves, and the development of the vegetations. Instead of the poison spending its force and the chronic condition obtaining, a process of softening, ulceration, development of abscess and perforation of leaflets follows, resulting in loss of structure, general septic infection, and the development of emboli, which lead to infarctions, with their results in either brain, kidney, spleen, eye, or skin.

SYMPTOMS. Vary greatly, but always associated with con-

stitutional signs of sepsis—a typhoid state, such as headache, restlessness, varying delirium, coated, dry tongue, sordes on teeth and lips, nausea, vomiting, loose or disordered stools, enlarged spleen, albumen in urine, and an irregular temperature record, varying from 100 degrees F. to 104 degrees F., or higher, associated with rigors and heavy sweating. The cardiac action is rapid, irregular, and weak—a compressible pulse. In the notes of twelve cases observed in the Philadelphia Hospital are the following symptoms: attacks of prolonged dyspnœa with paroxysms of intensity, or a slightly quickened respiration with paroxysms of dyspnœa occurring every few days. In four cases the paroxysms occurred three times daily, with respirations under twenty-five between the paroxysms, for three weeks preceding death. Usually the respirations are so oppressed that the recumbent position is impossible for long periods. Another frequent symptom is marked cyanosis, either transient or lasting for days before the end. A frequent symptom of ulcerative endocarditis is a peculiar facies, indicative of a sense of impending danger, great anxiety, or terror. If embolism occur, there are superadded symptoms varying with the organ affected. If the brain, rapidly developing palsies with disorder of consciousness; if the kidneys, deep-seated lumbar pains with haematuria or disordered urinary flow; if the spleen, pain and tenderness of the splenic region, with increase of temperature record.

Auscultation. The booming, muscular, first sound is superseded by a feeble, irregular cardiac pulsation. Generally, a murmur may be detected.

Diagnosis. One of the most difficult. Remembering the diseases with which malignant endocarditis may occur, and particularly pneumonia or sepsis, and the dyspnœa, the cyanosis, the facies, and the temperature record, it may be possible to diagnose the disease much more frequently than is done.

Prognosis. Unfavorable. Recovery the rarest termination.

Treatment. Same as for Acute Pericarditis.

CHRONIC ENDOCARDITIS.

Synonyms. Sclerotic endocarditis; interstitial endocarditis; chronic valvular disease.

Definition. Alterations in the cardiac valves or orifices, rendering the former incapable of properly closing the orifices, or

causing the narrowed orifice to interrupt the blood current in its normal movement. The lesions are of two kinds: obstructive and regurgitant.

A regurgitant lesion, termed also insufficiency, is such change in the valves as to permit a portion of the blood to flow backward instead of onward, the true direction of the blood current. An obstructive lesion, termed also stenosis, is a narrowing of the orifice, thereby obstructing the onward passage of the blood.

VARIETIES. 1. Mitral regurgitation. 2. Aortic regurgitation. 3. Tricuspid regurgitation. 4. Pulmonary regurgitation. 5. Mitral obstruction. 6. Aortic obstruction. 7. Tricuspid obstruction. 8. Pulmonic obstruction.

CAUSES. The great majority of cases are the result of acute endocarditis following rheumatism, chorea, or the infectious diseases. Chronic endocarditis from the onset is caused by alcoholism, syphilis, gout, and excessive muscular labor. Chronic Bright's diseases are also exciting causes. Professor Da Costa has clearly established the development of aortic disease in early life by overwork and strain of the heart. In the elderly, chronic endocarditis is the result of atheromatous or fibroid changes.

MITRAL REGURGITATION.

This form, also termed insufficiency, is the most frequent of all the varieties.

PATHOLOGICAL ANATOMY. The most common conditions observed are more or less contraction and narrowing of the tongues of the valves, with irregular thickening and rigidity; atheroma or calcification of the segments; laceration of one or more segments; adhesion of one or more segments to the inner surface of the ventricle; thickened and stiffened or rupture of the chordae tendineae, and also contraction and hardening of the musculi papillaries. As a result of the regurgitation of the blood into the left auricle, there is dilatation of the auricle, followed by slight hypertrophy.

SYMPTOMS. Insufficiency of the mitral valves soon leads to cardiac hypertrophy, to compensate for the diminished amount of blood sent onward by the ventricular systole. This condition causes quickened and strong pulse with some shortness of breath on severe exertion. When the "compensation ruptures" there occur praecordial distress, cough, dyspnœa, feeble, soft, rapid, irregular pulse; finally pulmonary congestion, œdematous limbs and general cyanosis, the abdominal cavity filled, liver congested,

urine scanty and albuminous, the patient dying "drowned in his own fluid."

INSPECTION. Cardiac impulse (apex-beat) displaced to the left and downward. In children and youths, bulging of the praecordia and increased cardiac impulse.

PALPATION. Displaced cardiac impulse, early stage being forcible and diffused; as compensation fails, impulse feeble or absent.

PERCUSSION. Transverse and vertical cardiac dullness increased.

AUSCULTATION. Systolic blowing or churning murmur, audible in the mitral area, propagated to the apex, left axilla, and under the angle of the scapula, either occurring with or taking the place of the first sound of the heart; the second sound being markedly accentuated.

PROGNOSIS. So long as the compensating hypertrophy can be maintained the prognosis is not unfavorable; when dilatation supervenes, however, the patient soon perishes, either from congestion of the lungs or dropsy and exhaustion.

AORTIC REGURGITATION,

Termed also, aortic insufficiency, is next in frequency to mitral insufficiency.

PATHOLOGICAL ANATOMY. The valves or segments adhere to the walls of the aorta, or a segment is lacerated or may be perforated, or, more commonly, the segments are shrunken, deformed, and rigid, permitting the regurgitation of the blood. These deficiencies in the valves are usually associated with more or less narrowing of the orifices. The inability of the aortic valves to close the aortic orifice at the proper moment allows the blood that should go onward to flow back into the left ventricle, and the normal flow of blood from the left auricle continuing, causes overfilling of the ventricle, which results in a dilatation of its cavity, and the extra effort of the ventricle to empty itself results in hypertrophy of the walls. In no other condition does the dilatation and hypertrophy of the cardiac walls reach such a degree. The older writers named this enormous enlargement of the heart *cor bovinum*.

SYMPTOMS. Those of marked hypertrophy, to-wit: forcible cardiac action, headache, tinnitus aurium, congestion of the face and eyes, with pulsating vessels, even small ones pulsating that before were not visible to the eye; pulsations of the retinal vessels can be recognized with the ophthalmoscope; the receding pulse,

which is particularly characteristic—forcible impulse but rapidly declining, called "water-hammer" pulse; also, the "Corrigan pulse." When "compensation ruptures," dyspnœa, cough, cyanosis, hepatic enlargement, congestion of the kidneys, with scanty, albuminous urine, ascites, and dropsy. If mitral insufficiency is now superadded, general venous stasis and death rapidly occur. Praecordial pain is usually present in aortic disease. It may be a sensation of constriction in the cardiac region, or sharp, shooting pains extending to the arms—anginoidal attacks.

INSPECTION. Forcible cardiac impulse.

PALPATION. Strong, full cardiac impulse.

PERCUSSION. Cardiac dullness increasing transversely and vertically.

AUSCULTATION. First Sound.—Forcible. Second Sound.—Replaced or associated with a churning, rushing, or blowing murmur of low pitch, distinct at the second right costal cartilage, but most distinct at the junction of the sternum and the fourth left costal cartilage, transmitted downward toward and below the apex.

PROGNOSIS. The one valvular disease most likely to occasion sudden death; still, so long as the compensating hypertrophy remains intact, compatible with quite an active life.

TRICUSPID REGURGITATION.

PATHOLOGICAL ANATOMY. This form of valvular insufficiency is either associated with right-sided cardiac dilatation from pulmonary obstruction, or is the result of mitral disease. The tricuspid orifice is dilated in the majority of cases; occasionally the segments of the valves are contracted or adherent to the ventricle.

SYMPTOMS. Venous stasis with its various consequences, and especially pulsation of the jugulars, synchronous with the cardiac movement, and, finally, general venous pulsation, especially of the liver, pulmonary congestion, engorgement of the kidneys, and dropsy. These symptoms are superadded to those of the affections with which tricuspid insufficiency is always associated.

INSPECTION. Diffused, wavy, cardiac impulse; jugular pulsation synchronous with the cardiac movement, uninfluenced by respiration, also more or less prominent hepatic pulsation.

PALPATION. The cardiac impulse extended, but feeble.

PERCUSSION. Dullness on percussion, extending to the right and below the sternum.

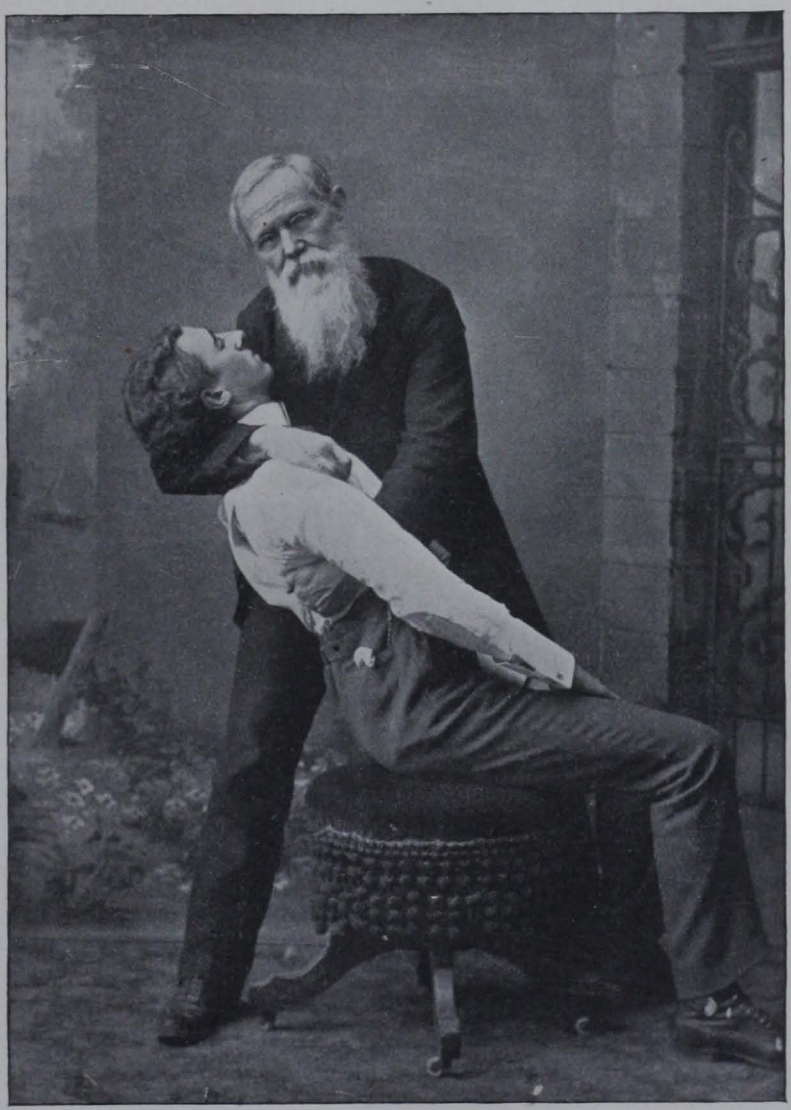


PLATE XLII.—Liver, Chest and Side Treatment.

AUSCULTATION. The first sound is accompanied by a blowing murmur most intense at the junction of the fourth and fifth ribs with the sternum, distinct over the xiphoid appendix, becoming feeble or lost in the left axillary region; often associated, however, with a mitral systolic murmur.

PULMONIC REGURGITATION.

PATHOLOGICAL ANATOMY. Insufficiency of the pulmonary valves is of rare occurrence, but when present the changes correspond more or less with those described for aortic regurgitation.

SYMPTOMS. Those of dilatation of the right side of the heart and consequent pulmonary congestion, to-wit: dyspnœa, deficient aeration of the blood and cyanosis, distention of the superficial vessels, palpitation of the heart, praecordial distress, sudden suffocative attacks, and dropsy.

PERCUSSION. The cardiac dullness extending to the right of the sternum.

AUSCULTATION. A loud, blowing murmur associated with the second sound of the heart, most distinct at the junction of the third left costal cartilage and the sternum.

PROGNOSIS. Death results, sooner or later, from dropsy and exhaustion.

MITRAL OBSTRUCTION.

Mitral obstruction or stenosis is not as frequent as regurgitation, and is very often associated with the latter.

PATHOLOGICAL ANATOMY. Mitral stenosis is caused by deposits around the orifice, the result of endocarditis, or else the segments of the valves are "glued together by their margins," leaving but a funnel-shaped opening, the so-called "buttonhole" mitral valve. Vegetations on the valves lead to more or less obstruction to the blood-current.

SYMPTOMS. Hypertrophy of the left auricle results from obstruction at the mitral orifice, followed in time by dilatation, the symptoms of stenosis being unobservable until the "compensation ruptures," or until dilatation becomes excessive, when occur irregular, small, and feeble pulse, dyspnœa, cough, bronchorrhoea the result of bronchial congestion; dilatation of the right side of the heart, soon leading to general venous stasis, dropsy, and death.

INSPECTION. Normal until auricular hypertrophy, when an undulatory impulse is observed over the left auricle.

PALPATION. When cardiac dilatation occurs, a diffused,

feeble, and irregular cardiac impulse is felt near the xiphoid appendix.

AUSCULTATION. First sound normal in character, but often irregular in rhythm. The second sound normal. A blowing, sometimes rasping, sound is heard, immediately after the second sound of the heart ceases, and immediately before the first sound begins—a presystolic murmur, heard most distinctly in the mitral area, lessening in intensity toward the cardiac base. The cardiac sounds are all more or less enfeebled if cardiac dilatation occur.

PROGNOSIS. The prognosis is controlled by the duration of the hypertrophy. Under favorable circumstances mitral stenosis is compatible with a long and rather active life.

AORTIC OBSTRUCTION.

PATHOLOGICAL ANATOMY. Stenosis of the aortic orifice is caused by the projection of the valves inward, and their becoming rigid and thickened, or atheromatous or calcareous, so that they can not be pressed back by the blood, but remain constantly in the current of the circulation. Occasionally the valves are covered with fibrinous masses, the opening into the artery being thus more or less completely closed, or the segments may be adherent by their lateral surfaces, leaving a central opening, which may be so contracted as to permit the passage of only the smallest probe.

SYMPTOMS. Hypertrophy of the left ventricle rapidly supervenes upon aortic stenosis. The pulse is small, slow, and hard. The supply of blood to the brain is insufficient in many cases, and hence attacks of vertigo, syncope, or slight epileptiform seizures occur; finally, dilatation of the left ventricle and incompetence of the mitral valve result, with subsequent pulmonary congestion, dyspnœa, and general venous stasis, the pulse soft and feeble.

PALPATION. Lowered cardiac impulse, strong in the early stage, feeble when dilatation occurs.

PERCUSSION. The cardiac dullness is increased vertically, the transverse dullness being slightly affected.

AUSCULTATION. The first sound replaced or associated with a harsh, rasping sound, whistling at times, having its greatest intensity at the junction of the second right costal cartilage with the sternum, transmitted along the vessels; the murmur may sometimes be heard a short distance from the patient. Usually aortic stenosis is associated with more or less aortic regurgitation, whence a double murmur occurs, having its greatest intensity at the base of the heart, the so-called to-and-fro, or see-saw murmur.

PROGNOSIS. So long as compensation is maintained the condition of the patient is comfortable, if a quiet life be followed. When the compensation is ruptured, the usual symptoms of dilatation, venous stasis, and dropsy soon ensue.

TRICUSPID OBSTRUCTION.

This condition is one of the rarest affections of the heart, and if it ever does occur with or following an attack of endocarditis, the anatomical changes are similar to those of mitral obstruction. This condition soon leads to auricular dilatation; venous stasis rapidly supervenes, associated with venous pulsations similar to those described when speaking of tricuspid regurgitation.

PULMONIC OBSTRUCTION.

PATHOLOGICAL ANATOMY. Always a congenital malady, the changes consisting in "constriction of the pulmonary artery, unclosed foramen ovale, unclosed ductus Botalli, stricture at the ductus Botalli, with hypertrophy of the right cavity and frequent association with tuberculosis of the lungs." Hypertrophy of the right ventricle may ensue, the walls becoming almost as thick as those upon the left side. Those in whom these congenital defects in the cardiac structure occur are otherwise weak. Develop slowly, have flabby tissues, soft bones, and seem poorly nourished.

SYMPTOMS. The hypertrophy whch often ensues may keep life apparently comfortable for some time, but sooner or later "compensation ruptures," when cough, dyspnœa, cyanosis, and death occur.

PROGNOSIS. The duration of these congenital affections is short, usually from a few days to a few months; although several well authenticated cases record a much longer duration.

DIAGNOSIS OF THE VALVULAR DISEASES.

In making a differential diagnosis between the various forms of valvular disease of the heart, strict attention must be paid to the points of greatest intensity at which the several murmurs are heard.

A murmur occurring with or taking the place of the first sound of the heart—the ventricular systole—heard most distinctly at the apex, transmitted to the left axilla, and to the inferior angle of the scapula, signifies mitral regurgitation—a mitral systolic murmur. A murmur occurring with or taking the place of the first sound of the heart, with its point of greatest intensity at the xiphoid appendix, signifies regurgitation at the tricuspid orifice—tricuspid systolic murmur. A murmur heard with the first sound

of the heart, high-pitched, rasping or grating in character, with its point of intensity greatest at the second right costal cartilage, signifies obstruction at the aortic orifice—an aortic systolic murmur. A murmur heard with the first sound of the heart, soft in character, with its point of intensity most distinct at the junction of the third left costal cartilage with the sternum, signifies obstruction at the pulmonic orifice—a pulmonic systolic murmur. A murmur occurring immediately after the second sound of the heart, and immediately before the beginning of the first sound of the heart, signifies obstruction at the mitral orifice—a presystolic mitral murmur. A murmur heard with or taking the place of the second sound of the heart, most distinct at the second costal cartilage, to the right of the sternum, and well transmitted toward the apex or below, signifies insufficiency or regurgitation at the aortic orifice—an aortic regurgitant or diastolic murmur.

Although eight distinct valvular murmurs have been described as occurring in the heart, those on the right side are of rare occurrence, and hence of little clinical importance.

If a murmur be heard with the first sound of the heart, it is almost certainly aortic obstructive or mitral regurgitant; and if heard with the second sound, it is probably aortic regurgitant. A presystolic mitral murmur is also of comparatively rare occurrence, the force with which the blood passes from the left auricle into the left ventricle being, under ordinary circumstances, insufficient to excite sonorous vibrations.

Functional or anaemic murmurs may be confounded with the various forms of valvular disease of the heart. The chief points of distinction between them are, that an anaemic murmur, which is always heard at the base of the heart, is always systolic in time, not transmitted away from the heart, and is soft in character, low in pitch, and of variable intensity, now being heard, now entirely absent.

CARDIAC HYPERTROPHY.

DEFINITION. An overgrowth or increase in the muscular tissue which forms the walls of the heart; characterized by forcible pulse, over-fullness of the arteries, diminished blood in the veins, and accelerated circulation.

CAUSES. Obstruction to the outflow of blood, resulting from valvular disease of the heart; emphysema; Bright's disease;

arterio-fibrosis; functional over-action; excessive use of tobacco, tea, coffee, or excessive muscular action.

VARIETIES. 1. Simple hypertrophy, or a simple increase in the thickness of the cardiac walls. 2. Eccentric hypertrophy, increase in the cardiac walls and dilatation of the cavities, to-wit: Dilated hypertrophy. 3. Concentric hypertrophy, increase in the cardiac walls, with decrease of the cavities, a very rare form.

PATHOLOGICAL ANATOMY. Hypertrophy of the heart is usually limited to the left side, the ventricles more commonly than the auricles, the latter dilating. The shape of the heart is altered by hypertrophy; if the right ventricle, the heart is widened transversely and the apex blunted; if the left ventricle, the heart is elongated and, as a rule, the cavity is dilated; if both ventricles are hypertrophied, the heart has a globular shape. From increase in weight the heart may sink lower during the recumbent position, thereby lessening the area of cardiac dullness, but during the sitting or upright posture it sinks lower in the chest and to the left, causing more or less prominence of the abdomen. The increase in the size of the organ is a true increase or hypertrophy of the muscular tissue, and not a hyperplasia. The tissue is firmer and the color brighter and fresher than when the size of the organ is normal. The *cor bovinum* of the old writers is an enormous hypertrophy of the heart with dilatation of its cavities.

SYMPTOMS. Depend upon the amount of hypertrophy. The most common are increased and forcible cardiac action, the arteries becoming fuller, the veins less full, and the circulation accelerated, pulsating carotids and aorta, headache, often vertigo, frequent epistaxis, congestion of the face and eyes, tinnitus aurium, dyspnœa on exertion, dry cough, restless nights, with more or less jerking of the limbs, occasional praecordial pains shooting toward the left axilla, full, firm, bounding pulse, and pulsations in the superficial arteries.

A sphygmographic tracing shows the line of ascent vertical and abrupt, but the apex is rounded, and the line of descent is oblique, unless there is more or less insufficiency of the valves.

INSPECTION. Often fullness or prominence of the praecordium, with distinct impulse.

PALPATION. The impulse is felt one or two intercostal spaces lower down and to the left, and is stronger and more or less diffused—the heaving impulse.

PERCUSSION. The area of cardiac dullness is increased vertically and transversely upon the left side of the sternum, unless

the right ventricle is also hypertrophied, when the cardiac dullness is increased to the right of the sternum.

AUSCULTATION. If simple hypertrophy without any coexisting changes in the valves or orifices, the first sound has a loud and somewhat metallic quality, the second sound being strongly accentuated.

SEQUELÆ. Cerebral hemorrhage; miliary cerebral aneurisms; dilatation of the heart; fatty changes of the cardiac tissue.

DIAGNOSIS. Hypertrophy of the heart can scarcely be mistaken for any other disease if a careful study of the physical signs be made.

PROGNOSIS. When the result of valvular disease, the hypertrophy is said to be compensatory. If the result of Bright's disease, emphysema of lung, or if occurring late in life, or associated with atheromatous degeneration of the vessels, the prognosis is unfavorable; when the result of functional over-action in the strong and robust, a further enlargement can often be prevented by active and persistent treatment.

DILATATION OF THE HEART.

DEFINITION. An increase in the size of one or more of the cavities of the heart, without any increase or thickening of the cardiac walls; in fact, the walls are frequently thinner—stretched; characterized by feebleness of the circulation, terminating in venous stasis, cyanosis, œdema, and exhaustion.

CAUSES. Over-exertion in those of feeble resisting powers, as youths or soldiers, as first pointed out by Professor Da Costa; chronic valvular disease; emphysema; chronic bronchitis; gout; Bright's disease; alcoholism; syphilis.

VARIETIES. 1. Simple dilatation, the cavities being enlarged, the walls normal. 2. Active dilatation, corresponding to eccentric hypertrophy; the cavities being enlarged and the walls increased in thickness, the so-called "dilated hypertrophy." 3. Passive dilatation, the cavities being enlarged and the walls thinned or stretched.

PATHOLOGICAL ANATOMY. The right side of the heart is far more frequently involved than the left side. The shape of the organ is altered, depending on the part affected. The weight of the organ is, as a rule, increased, as hypertrophy almost always accompanies or precedes dilatation.

The muscular tissue is generally pale, mottled, and softened, and under the microscope presents evidences of degeneration. The orifices also participate, and especially the auriculo-ventricular, resulting in the valves becoming incompetent to close the orifices, and this latter effect is added to by the removal of the basis of the papillary muscles a greater distance from the orifice, in consequence of the extension of the wall. When the auricles dilate, the large venous trunks opening into them unprotected by valves commonly participate in the dilatation, and may become greatly enlarged. The passive congestion of the organs that follows the feeble circulation produces changes in their structure.

SYMPTOMS. Those associated with enfeebled circulation, to-wit: feeble pulse, veins distended, arteries emptied; headache, aggravated by the upright position; attacks of syncope, cough, with any of the following phenomena of venous congestion: of the lungs, dyspnœa; liver, jaundice; stomach, dyspepsia; intestines, constipation; kidneys, scanty, often albuminous urine; brain, dullness of the mind and vertigo, often relieved by a copious epistaxis; and, finally, dropsy, beginning in the lower extremities, the patient dying from exhaustion.

Great relief often temporarily follows the above symptoms under treatment; sooner or later, however, the venous stasis produces the final symptoms noted.

INSPECTION. Veins of the surface distended and enlarged; indistinct cardiac impulse, often diffused and wavy; if associated with tricuspid insufficiency, there is pulsation of the jugular.

PALPATION. Feeble and irregular fluttering but heaving impulse.

PERCUSSION. Cardiac dullness extended transversely, and especially increased on the right side.

AUSCULTATION. If no valvular lesion accompany the dilatation, the cardiac sounds are weaker than normal, the first sounds having a sharper quality than normal; if accompanied by valvular lesions, cardiac murmurs are present.

DIAGNOSIS. Hypertrophy of the heart shows increased cardiac dullness, and is a disease of powerful cardiac action, while dilatation is an affection of feeble action associated with dropsy.

Pericardial effusion has many points of resemblance to cardiac dilatation, but it begins suddenly, associated with some acute malady; and while the heart sounds are indistinct or feeble at the apex, they both have their normal qualities at the cardiac base, while dilatation of the heart has a chronic history, results in

general venous stasis, the cardiac sounds being of the same intensity over the entire praecordia.

PROGNOSIS. Unfavorable, death resulting from gradual exhaustion, or suddenly by cardiac paralysis if there be some undue excitement.

ACUTE MYOCARDITIS.

SYNONYMS. Carditis; abscess of the heart.

DEFINITION. An inflammation of the muscular tissue of the heart, by extension from an inflamed pericardium or endocardium, or secondary to pyaemia; characterized by pain, feeble circulation, symptoms of blood poisoning, and collapse.

CAUSES. The result of endocarditis or pericarditis; pyaemia; typhoid fever; emboli of the coronary arteries.

PATHOLOGICAL ANATOMY. Discoloration and softening of the cardiac substance and the infiltration of a sero-sanguineous fluid, fibrinous exudation and pus, leading to the formation of abscesses in the muscular structure of the heart.

The disease leads to the formation of either a cardiac aneurism or to rupture of the walls of the heart. If recovery occur, cicatrices or depressed scars may mark the site of a former abscess.

SYMPTOMS. The clinical evidences of inflammation of the cardiac muscles are very obscure. If, during the course of one of the maladies mentioned, there are developed praecordial pain, irregular and feeble cardiac action, cardiac dyspnœa, pyrexia of a low type, with symptoms of blood poisoning, and a tendency to collapse, or the symptoms of the so-called typhoid state, myocarditis may be suspected.

DIAGNOSIS. The existence of myocarditis can scarcely ever be anything but a presumption, the signs being all negative rather than positive. If during the course of rheumatism, pyaemia, puerperal fever, typhoid fever, pericarditis, or endocarditis, symptoms of cardiac failure appear suddenly, associated with signs of blood poisoning and collapse, inflammation of the cardiac muscle may be suspected.

PROGNOSIS. The course of acute myocarditis is vary rapid, death being the usual termination, in from three to five days. Chronic myocarditis pursues a very latent course.

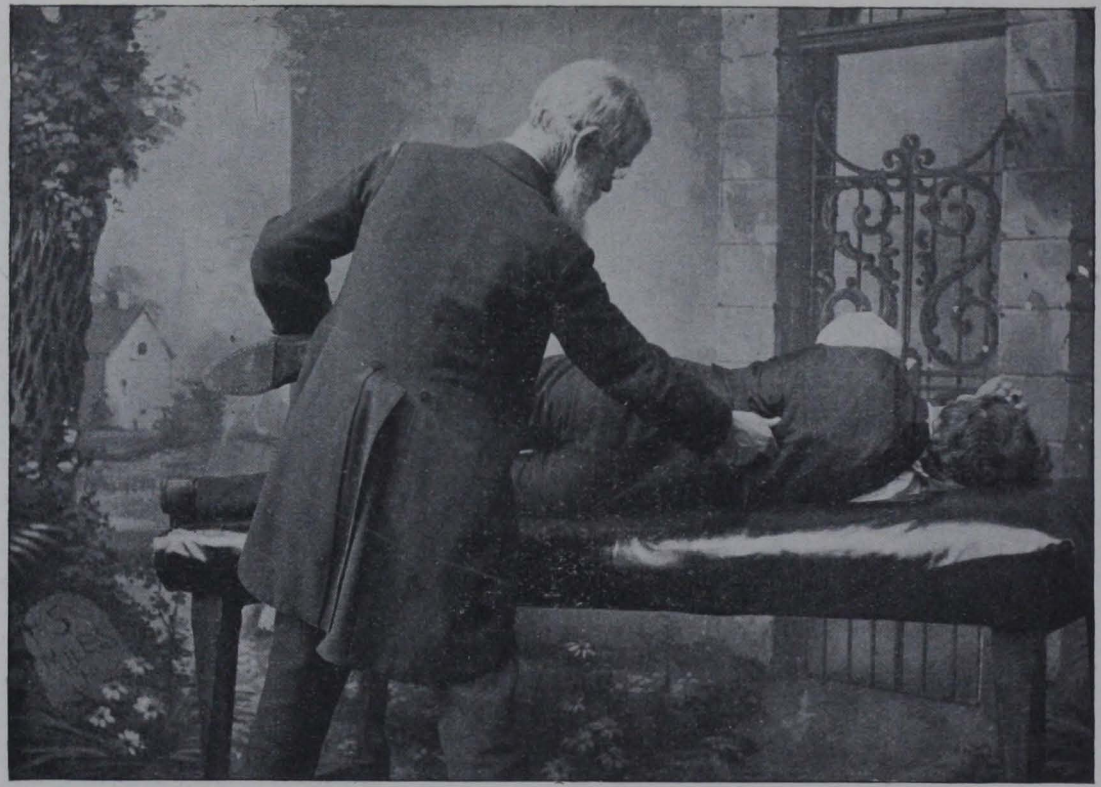


PLATE XLIII.—Manipulation of Spine, Dorsal Treatment.

CHRONIC MYOCARDITIS.

SYNONYMS. Fibroid heart; chronic interstitial myocarditis; fibrous myocarditis; chronic carditis; cardio-sclerosis.

DEFINITION. A slowly developing hyperplasia of the interstitial connective tissue of the heart, leading to induration of its substance; characterized by shortness of breath on slight exertion, attacks of tachycardia, praecordial pain, disordered circulation, and vertigo. It is proper to state that many cases present no symptoms whatever.

CAUSES. The most frequent cause is sclerosis of the coronary arteries, leading to imperfect blood supply to the cardiac muscles. Amongst other frequent causes are diseases of the kidneys, alcoholism, excessive use of tobacco, syphilis, secondary to pericarditis, endocarditis, and acute myocarditis. There is, undoubtedly, often an inherited predisposition to fibroid changes in the vessels, in which cases the causes named would act as exciting causes. It is a disease of the aged, save in those instances resulting from excesses. The old saying, "A man is as old as his arteries," is applicable to this disease.

PATHOLOGICAL ANATOMY. The heart is enlarged and dilated. The morbid changes may be diffused, or limited to the walls of the left ventricle, the papillary muscles, and the septum. There is always more or less atheromatous deposit or changes in the aorta. All cases show atheroma in one, more, or all of the coronary arteries. Complete closure of one coronary artery, if produced suddenly, is usually fatal. On section the cardiac wall cuts with a distinct resistance. The changes in the heart wall are an "overgrowth of the interfibrillar connective tissue, with development of fibrous tissue. These changes may be uniformly distributed through the substance of the heart when some intoxication, as by alcohol, or some general disturbance of the cardiac nutrition, has led to the myocardial disease; or they may be seen in circumscribed areas when embolic or thrombotic occlusion of branches in the coronary arteries has occasioned anaemic infarcation and subsequent sclerosis. In either case the microscope reveals masses of wavy fibrous tissue between the muscular bundles, and often slow degeneration or atrophy of the fibers themselves." (Pepper.) The terminal branches of the coronary arteries are narrowed and sclerotic to the point of obliteration, particularly in cases resulting from syphilis.

"Aneurism of the heart is commonly due to localized cardio-sclerosis. The inelastic fibrous tissue gradually gives way before

the intracardial pressure, and saccular dilatation results." (Pepper.)

Atheromatous changes are often found in other than the coronary vessels, particularly the aorta. Various degenerative changes occur in other organs, the result of disturbed circulatory action.

SYMPTOMS. The great majority of patients having chronic myocarditis present no symptoms until an extra cardiac effort is called for. An early symptom is breathlessness on slight exertion, with either cardiac palpitation or a feeble, irregular pulse. Anginal attacks (cardiac pain) or a sensation of constriction or pressure over the praecordia are frequent, often following some exertion or an attack of indigestion. The pulse-rate is decreased in number in cases which present no other symptom. A frequent symptom is syncope, coming without warning or after sudden exertion, the result of sudden failure of the cerebral circulation. Amongst other periodical symptoms are cardiac asthma, pseudo-apoplectic attacks, hepatic, gastric, and nephritic disorders.

As the fibroid changes progress, there develops progressive weakness, dyspnœa, insomnia, disordered digestion, and cerebral weakness, often showing itself as mania, delusional attacks, or dementia.

PERCUSSION. Increased praecordial dullness is usually present, due to the dilated hypertrophy.

AUSCULTATION. The first sound of the heart is valvular in character, the booming or muscular quality having disappeared. Murmurs are very frequent, the result of valvular disease. A very characteristic point is the irregularity in rhythm and in force, one contraction being fairly forcible, another weak or feeble, and so on.

DIAGNOSIS. A proper appreciation of chronic myocarditis is one of the most important questions in clinical medicine. The term Heart Failure is the opprobium of the profession, and yet chronic myocarditis is one of the great causes of cardiac failure during the prevalence of some over-exertion, in acute pneumonia, typhoid fever, and other like diseases. The points of value in arriving at a diagnosis are: a careful study of the first sound of the heart at the apex; the character of murmurs if present, the condition of the arteries, the dyspnœa, the feeble, irregular pulse in patients past fifty years, and the occurrence of anginal attacks after exertion or mental worry.

PROGNOSIS. Ths is controlled by the habits of the patient.

The disease is incurable, but life may be fairly comfortable for many years if care be exercised.

FATTY HEART.

SYNONYMS. Fatty degeneration of the heart; chronic myocarditis.

DEFINITION. A change in the muscular fibers of the heart, in which the transverse striae are replaced by granules and globules of fat; characterized by feeble cardiac action, venous stasis, and dyspnœa.

CAUSES. Impaired nutrition in the elderly; prolonged anaemia; chronic gout; alcoholism; phosphorus poisoning; cancer; tuberculosis and scrofula; diseases of the coronary arteries.

PATHOLOGICAL ANATOMY. The distinction must be made between a deposit of fatty tissue upon or around the heart, and the degeneration of its muscular tissue.

The fatty metamorphosis may affect the whole organ, or the entire ventricular walls, or be limited to portions of them. If the degeneration be marked, the color is yellowish, the tissues soft and easily torn, and to the touch have a greasy feeling, oil being yielded on pressure.

The microscopic changes are characteristic. The striae of the muscle are easily rendered indistinct by fat and oil globules, gradually becoming more and more obscured, and finally disappearing altogether, the fibers being replaced by fat granules.

SYMPTOMS. Those of weak heart, anaemia of organs, and venous stasis, to-wit: feeble, irregular, but slow cardiac action, compressible pulse, praecordial distress, often aggravated by attacks of angina pectoris; dyspnœa, aggravated on exertion, with anaemia of the various organs from the feeble propulsive power; if of brain, vertigo, swooning, or pseudo-epileptic attacks, especially marked on suddenly rising from a recumbent position; if of lungs, dry, hacking cough; if of gastro-intestinal tract, dyspepsia and constipation; if of kidneys, scanty urine, at times albuminous; and finally dropsy, beginning in the lower extremities.

A formidable symptom, causing much inconvenience as well as alarm to the patient, is what he will term his constant "sighing," the Cheyne-Stokes breathing—"A pause in the breathing, a complete suspension of the respiratory acts for a period of time

(during which breathing might occur several times in the normal manner), then the resumption of respiration very feebly and slowly, and a gradual and progressive increase in the number and depth of respirations until the maximum is reached, and then again a gradual and progressive diminution, in the same order, in the number and depth of the respirations, until another pause occurs"—the "oscillating respiration."

Concomitant symptoms are atheromatous changes in the vessels, and the arcus senilis.

PALPATION. Weak cardiac impulse.

PERCUSSION. Not markedly changed unless preceded by enlargement of the heart

AUSCULTATION. First sound feeble, toneless, almost inaudible, the second sound being normal, unless changes in the valves are present.

DIAGNOSIS. Feeble cardiac sounds, with slow pulse, attacks of cardiac asthma or Cheyne-Stokes breathing, with evidences of arcus senilis, make the diagnosis very certain. The question of fibroid heart must always be considered.

PROGNOSIS. Incurable, the affections pursuing a more or less chronic course. Life may be prolonged at times by treatment, but death finally results from exhaustion, or suddenly, from cardiac paralysis or rupture of the heart.

PALPITATION OF THE HEART.

SYNONYM. Irritable heart.

DEFINITION. A functional disturbance of the heart, characterized by increasing frequency of its movements, and more or less irregularity of the rhythm, with a strong tendency toward hypertrophy.

CAUSES. Over-exertion, "the heart strain" of Da Costa; dyspepsia; uterine diseases; excesses in tea, coffee, tobacco, alcohol, or venery; moral and emotional causes, grief, anxiety, and fear.

SYMPTOMS. Usually palpitation of the heart has a sudden onset after some one of the causes mentioned, praecordial oppression or pain, rapid, tumultuous beating, the impulse being visible through the patient's clothing, dyspnœa, anxiety, and a sense of choking or fullness in the throat, the recumbent position impossible, vertigo, faintness, flashes of light, the pulse full and strong

or feeble, the face flushed or pale, the patient having a feeling of anxiety with a sense of impending danger and fear of sudden death. These attacks are paroxysmal, lasting from a few moments to several hours, or a day, the patient often voiding a large quantity of limpid urine after the paroxysm has subsided, when there is a strong tendency to sleep.

DIAGNOSIS. Irritability of the heart is differentiated from the various forms of cardiac disease by the absence of all the physical signs mentioned as occurring in those conditions.

PROGNOSIS. If early and properly treated, favorable.

TACHYCARDIA.

SYNONYMS. Rapid heart; quick heart; paroxysmal rapid heart.

DEFINITION. Paroxysmal rapid cardiac action minus or with subjective symptoms, the result of excessive cardiac rapidity.

CAUSES. Tachycardia is one of the "crises" of cerebral or spinal diseases. Menopause. Neuritis of the pneumogastric nerve; chronic myocarditis; neurasthenia; chronic gastritis; excessive use of tobacco.

PATHOLOGICAL ANATOMY. No characteristic lesions. There may be paralysis of the inhibitory fibers of the vagus, an irritation of the accelerators of the sympathetic, or reflex action from some lesion in the cardiac wall or elsewhere.

SYMPTOMS. The paroxysm is sudden in its onset, with or without "warnings"—if these latter, they are in the shape of vertigo, ringing in the ears, and a sense of impending danger. The cardiac action is increased to 150, 175, 200, rarely 250 beats per minute. The pulse is small, weak, easily compressible, and often irregular. The respiration is slightly increased; rarely there is dyspnœa. The surface is at first pale, but soon becomes flushed. The expression is anxious and denotes suffering. There is a feeling of praecordial constriction, with more or less smothering. Rarely, there are no subjective symptoms. The duration is from a few minutes, to hours, or days.

AUSCULTATION. The first sound is clear and ringing, but not strong and booming. The second sound is weak and lacks the valvular quality of the normal. A murmur is often heard at the apex.

DIAGNOSIS. The differentiation between tachycardia and

palpitation is to be made, as also the rapid heart of valvular disease and of irritable heart. The chief point is that in tachycardia the attack is paroxysmal, and the number of pulsations exceeds the rapid heart of other conditions.

PROGNOSIS. As a rule, it is an unfavorable symptom of some central lesion. If it develops in patients suffering from chronic myocarditis or atheroma of vessels, the fatal result may be sudden.

BRADYCARDIA.

SYNONYM. Brachycardia.

DEFINITION. A paroxysmal or permanent slowness in the cardiac action.

CAUSES. Often associated with organic nervous diseases. It is a symptom of such cardiac diseases as fibroid and fatty heart and atheroma of the coronary arteries. It frequently occurs during convalescence from infectious diseases, such as diphtheria, pneumonia, typhoid fever, erysipelas, and rheumatism; uraemia, lead poisoning, anaemia, and chronic alcoholism are often causes.

SYMPTOMS. Slow action of the heart is the chief symptom, varying from 50, 40, 30, 20, to 10 or 8 beats per minute. The pulse is weak, small and slow. As results of the slow cardiac action are vertigo, noises in the ears, syncopal attacks, and rarely convulsions. The onset may be either sudden or follow "warnings."

AUSCULTATION. The first sound is soft and feeble, and often the second sound is not heard. As a rule, with reduction in the number of contractions is an increase in their force; this not obtaining in bradycardia determines its central origin.

DIAGNOSIS. A feeble cardiac contraction, with less than fifty beats per minute, determines the diagnosis.

PROGNOSIS. Sudden death a very frequent termination. The cause controls the prognosis.

ARRHYTHMIA.

SYNONYMS. Arrhythmia cordis; irregularity of the pulse.

DEFINITION. A lack of cardiac rhythm, or irregularity in the cardiac pulsations. It is a symptom rather than a disease.

CAUSES. Valvular diseases; myocardial diseases; cardiac

dilated hypertrophy; atheroma of coronary arteries and aorta; excessive use of tobacco, tea, coffee; flatulent dyspepsia. Neurasthenia, hysteria, and melancholia.

SYMPTOMS. An irregularity in cardiac action, either in the rhythm or the regularity in the force of the beats, or an intermission in the cardiac contractions. The sphygmograph gives the exact condition of the cardiac pulsations and should always be used in cardiac diseases. Other symptoms that may be present are due to the condition producing the arrhythmia.

DIAGNOSIS. An examination of the pulse, auscultation of the heart, and the use of the sphygmograph determine the arrhythmia.

PROGNOSIS. Depends upon the cause. In functional cases favorable, in organic cases unfavorable.

ANGINA PECTORIS.

SYNONYM. Neuralgia of the heart.

DEFINITION. Paroxysms in which there occur sharp cardiac pains, extending usually into the left shoulder and down the left arm, accompanied by a feeling of constriction of the thorax and a strong fear of impending death.

CAUSES. Depending upon the variety, whether of nervous or organic origin. Often hereditary; associated with chronic cardiac changes, as diseases of the coronary arteries or calcification of the valves; the excessive use of tobacco; syphilis; according to Trousseau, it is a form of masked epilepsy, and may alternate with true epileptic attacks; often associated with hysteria.

PATHOLOGICAL ANATOMY. A disease of the arteries, ossification and occasionally obliteration of the cardiac arteries, producing ischaemia.

"The pathological changes which stand in a causative relation to the attacks are those of the cardiac plexus of the phrenic and of the pneumogastric nerves. Pressure of enlarged lymphatics, inflammation of parts of the cardiac plexus, with changes in the coronary arteries, seem to be most constant."

SYMPTOMS. A paroxysmal affection, the attacks occurring irregularly; in the interval entire absence of symptoms, or the symptoms of the organic disease causing the paroxysms.

"The patient suddenly sits up in his bed; with a cry of horror

indicates the sense of pain at the praecordium. The pain is of great intensity, but is of a cold and sickening character; the chest is fixed, the breathing quickened, and the hand placed over the praecordia finds that the heart's action is slight and enfeebled. The face wears a look of horror, pale and slightly leadened; a cold sweat breaks out upon the forehead; worse than the pain is the feeling of fearful sickness and depression. The poor patient gasps, 'I shall die! I shall die!' and sometimes his short but concentrated sufferings in a few moments end in death. The attack ends suddenly with vomiting, or great flow of urine."

The unpleasant sensations of these patients during an attack and the nervous disorder associated with it, slowly bring about a mental change. They are depressed and gloomy, sometimes suicidal, and often developing epilepsy.

Attacks of angina in nervous women and children, the hysterical or pseudo-anginal attacks, come on gradually with distention of the abdomen, eructations of gas, excessive restlessness, flushed face, irritable pulse, diffused praecordial pain, and general hysterical phenomena.

Diagnosis. The points to be remembered are that the attacks are always paroxysmal, the patient having a sense of coldness, and frequently a cold sweat, the heart's action not increased, the chest fixed, and the breathing slow.

Prognosis. True angina pectoris is unfavorable, the patient, sooner or later, either succumbing during the paroxysm or from exhaustion, the result of the cardiac changes.

Pseudo-angina is always favorable.

ARTERIO-SCLEROSIS.

Synonyms. Atheroma; arterio-capillary fibrosis (Gull and Sutton); endocarditis chronica deformans (Virchow).

Definition. An overgrowth of the connective tissue of the arteries followed with calcareous deposits. The changes may extend to the capillaries and veins. As a result of the impairment of the arterial circulation occur fibroid degenerations in other organs, resulting in loss of elasticity in the walls of the vessels, increase of arterial tension, narrowing of the caliber of smaller arteries, and impairment of the nutrition of the organs supplied.

Causes. Old age, alcoholism, syphilis, lead-poisoning, diabetes, malaria, rheumatism. Heredity is a predisposing factor in

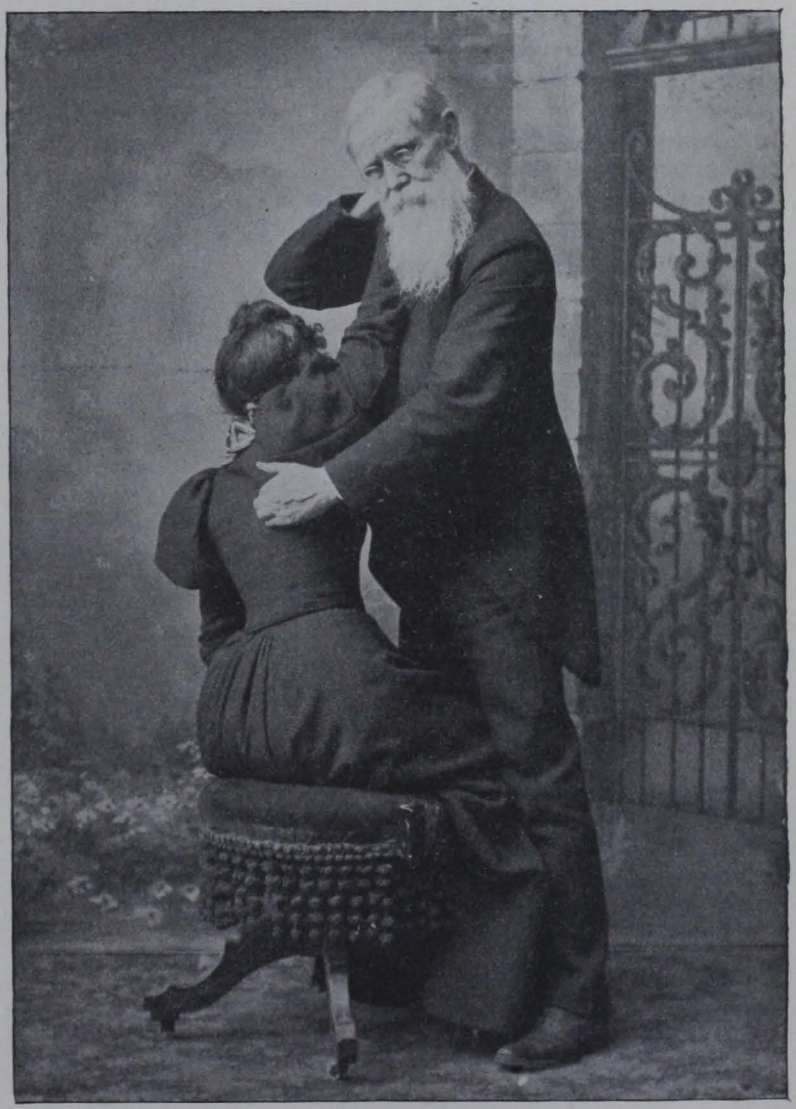


PLATE XLIV.—Arm Extension, Upright Treatment.

some cases. Chronic nephritis. More common in men than in women.

PATHOLOGICAL ANATOMY. The atheromatous changes are most frequent in the aorta. Other arteries affected are the coronary, the radial, ulnar, brachial, iliac, femoral, and the arteries of the brain.

The internal surface of the affected vessels is irregularly thickened with either gelatinous and translucent, or dense and fibrous, or calcareous deposits or products. If the calcification is extensive, the vessel is changed into a hard, stiff tube. Often the surface of the thickening or deposit is destroyed, presenting the so-called "Atheromatous ulcers," which may be covered with masses of thrombus.

The above changes are the result of inflammatory change in the intima of the affected vessel. This appears three or four times as thick as normal, due to the swelling of its elements, the new growth of connective tissue, and the deposit of round cells. Fatty degeneration of the inflammatory products results.

The result of the changes in the arteries is a loss of their elasticity, thus hindering the propulsion of the blood current and raising the arterial tension, leading to hypertrophy of the left ventricle. The changes finally affecting the coronary arteries lead to changes in the myocardium. If the intima of the smaller vessels be involved the blood supply to the organs supplied is lessened, resulting in disturbance of their nutrition.

SYMPTOMS. Not always apparent. The symptoms vary with the arteries involved, and the organs whose blood supply is lessened or cut off. Cardiac hypertrophy from the increased resistance to the arterial circulation. The peripheral arteries involved in the atheromatous changes can be determined by palpation, they having a hard, bony feeling, much like whip-cord. Attacks of vertigo, pseudo-apoplectic attacks, or spells of unconsciousness in the aged or those having superficial hardened arteries are generally due to changes in the cerebral vessels. Evidences of myocarditis and angina pectoris point to atheroma of the aorta and coronary arteries. Gangrene of the extremities in the old—senile gangrene—point to atheroma or thrombi, the result of the fibrosis.

PALPATION. Hard, superficial arteries, those at the wrist feeling like a string of beads, pulsating. The cardiac impulse is forcible in the early stages.

PERCUSSION. Increased praecordial dullness, particularly over left ventricle.

AUSCULTATION. In the early stages the first sound of the heart is prolonged, the second sound accentuated over the aortic cartilage. As the heart dilates and the walls become diseased, the sound becomes feeble and often irregular and intermittent.

DIAGNOSIS. Only determined by a close study of the various symptoms and sequelae.

PROGNOSIS. Incurable.

ANEURISM OF THE AORTA.

VARIETIES. 1. Aneurism of the arch of the aorta. 2. Aneurism of the thoracic aorta. 3. Aneurism of the abdominal aorta.

The arch of the aorta is divided by Gray into three parts, the ascending, the transverse, and the descending. The ascending portion is two inches in length, arising from the left ventricle, on a level with the lower border of the left third costal cartilage, behind the left edge of the sternum. It ascends obliquely upward to the right to the upper border of the right second costo-sternal articulation. The transverse portion commences at the upper border of the right second sternal articulation, and, arching to the left and forward, passes in front of the trachea and œsophagus to the left of the third dorsal vertebra. The descending portion extends downward to the left side of the fourth dorsal vertebra.

The thoracic aorta extends from the left lower border of the fourth dorsal vertebra, and ends in front of the body of the twelfth dorsal vertebra, at the aortic opening in the diaphragm.

The abdominal aorta begins at the aortic opening in the diaphragm, descends a little to the left side of the vertebral column, and terminates over the body of the fourth lumbar vertebra, where it divides into the two common iliac arteries.

DEFINITION. A circumscribed dilatation of some portion of the aorta, the result of disease of the vessel wall weakening its resistance to the blood pressure.

CAUSES. Those causing arterio-sclerosis are the chief causes. Exertion is an exciting cause. Aneurisms occur in early middle life rather than in old age, when the force of the heart has decreased. More common in men than in women.

PATHOLOGICAL ANATOMY. All aneurisms may be divided into two classes, dissecting and circumscribed.

Dissecting Aneurism—false aneurism—is the result of fatty changes in the internal and middle coats of the artery. The shape may be sacculated, fusiform, or cylindrical. A disease of the aged. Circumscribed Aneurism may be true or false, depending on the rupture of the walls or not. It is a disease of middle life or under. Most frequent in men, usually a true dilatation. Syphilis is a most frequent cause.

I. ANEURISM OF THE ARCH.

SYMPTOMS. The onset is usually gradual, with evidences of arterio-sclerosis and failing health.

Pain, either paroxysmal or constant, is a constant symptom, with increasing dyspnœa. The difficulty in breathing may be constant with exacerbations, or it may be remittent. Rarely dysphagia occurs. A slight cough from pressure on the laryngeal nerve with more or less alterations in the voice may be present. The pupils are dilated or contracted or are irregular, in some cases due to pressure on the sympathetic nerve. There is a gradual loss of flesh, disorders of the circulation, and a careworn expression of the face.

INSPECTION. Negative until the appearance of a pulsating tumor.

PALPATION. A pulsation over the tumor expansive in character (Corrigan's sign). If the aneurism is situated at the transverse portion of the arch, the left pulse and the left carotid are smaller and weaker than those on the right side. Tracheal tugging is a diagnostic sign (Page). "Place the patient in the erect position with his mouth closed and chin elevated to the fullest extent. Then, on grasping the cricoid cartilage between the fingers and thumb and making gentle traction upward, the pulsations of dilated aorta or aneurism, if any exist, will be distinctly felt, in most cases transmitted through the trachea to the hand."

PERCUSSION. Dullness, the extent depending on the size of the tumor. Dullness, other than cardiac, across the sternum is diagnostic of a mediastinal tumor.

AUSCULTATION. Over the tumor a murmur or bruit is usually heard, synchronous with the first sound of the heart. It is louder than the systole, lower in pitch, and of a blowing character.

DIAGNOSIS. If the tumor can be seen or felt, the diagnosis is made, its location being determined by a study of the physical signs.

II. ANEURISM OF THE THORACIC AORTA.

SYMPTOMS. The most constant symptom is deep-seated thoracic pain, constant or paroxysmal. Dysphagia is a frequent condition. There is seldom dyspnœa, and alterations of voice and pupils do not occur. Physical signs are usually wanting, and the diagnosis is rarely made during life.

III. ANEURISM OF THE ABDOMINAL AORTA.

SYMPTOMS. The chief and most constant symptom is pain at a circumscribed spot in the abdomen, or diffused. Other symptoms depend upon the location of the aneurism, as they are the result of pressure. There is a gradual loss of health.

INSPECTION. Usually negative unless the aneurism reach an enormous size.

PALPATION. A pulsating tumor in the abdomen to the left of the median line. The pulsation is synchronous with the first sound of the heart, and is expansile (Corrigan's sign) in character.

PERCUSSION. Dullness may be elicited if the tumor is large and the abdomen emaciated.

AUSCULTATION. Rarely a murmur or bruit is heard, systolic in time.

DIAGNOSIS. Abdominal aneurism and pulsating abdominal aorta may be mistaken for each other. The point of difference is, in the aneurism, the presence of the tumor with an expansile pulsation, while in pulsating abdominal aorta the beating is like a pulsating cord, an up-and-down movement, not expansile. The condition of the patient is also important; aneurism in males, at middle life, with changes in the vessels; abdominal pulsation occurring in nervous women or effeminate men.

Tumors located over the abdominal aorta may give rise to an apparent pulsation, causing them to be mistaken for an aneurism. The rule is in all cases of abdominal pulsation to place the patient in the knee-chest position; if the tumor is aneurismal, the expansile pulsation continues; if not an aneurism but a cancer, impacted faeces, or other tumor, the pulsation at once ceases.

PROGNOSIS OF AORTIC ANEURISMS. Unfavorable. The duration of life after the development of the aneurism is from one to four years.

TREATMENT FOR ALL HEART AFFECTIONS.

The equalization of the circulation throughout the body should be promoted as much as possible.

These affections demonstrate the variableness of the symptoms according as different nerve filaments are involved, in the different structures, and yet the removal of the venous obstruction leaves the channels free to carry off the waste, and makes room for capillary circulation; that brings in arterial blood to rebuild the waste tissue, caused by degenerative tissue metamorphosis. Keep lifting off the pressure. The treatments should be applied according to conditions, effects, etc.

The condition recognized as Palpitation (the most common of all complained of) is relieved almost instantaneously by steadily and strongly raising the left arm, and lifting the clavicle in the usual way, and pressing the knee between the scapulae, while the arms are drawn upward and backward a time or two.

The other affections of the heart require a very special attention, recognizing the fact that too much or too severe treatment should be avoided, and always to endeavor to do all that is possible to regulate and equalize the circulation as much as can be under the circumstances, persistently, repeatedly.

DISEASES OF THE NERVOUS SYSTEM.

The diseases of the nervous system will be described under the following named headings:

1. Diseases of the cerebral membranes. 2. Diseases of the cerebrum. 3. Diseases of the spinal cord. 4. Diseases of the nerves. 5. General or nutritional diseases. 6. Mental diseases.

DISEASES OF THE CEREBRAL MEMBRANES.

PACHYMENINGITIS.

SYNONYMS. Meningitis; haematoma of the dura mater.

DEFINITION. Inflammation of the dura mater; when the external layer is primarily involved it is termed pachymeningitis externa; when the internal layer is primarily involved it is termed pachymeningitis interna.

CAUSES. Pachymeningitis externa is a surgical malady, excited by fractures, penetrating wounds, and other injuries of the skull.

Pachymeningitis interna is due to blows upon the head without injury to the skull. A predisposition may be created by chronic alcoholism, scurvy, Bright's disease and syphilis. Chronic internal otitis and suppurative inflammation of the orbit may cause it, also inflammation in the venous sinuses the result of a thrombus undergoing suppurative changes.

PATHOLOGICAL ANATOMY. Pachymeningitis Interna.—Hyperaemia of the membrane, followed by an exudation which develops into a membranous new formation, containing a great number of vessels of considerable size, but having very thin walls.

Hemorrhages from these new vessels are of frequent occurrence, which increase the size and thickness of the neo-membrane.

The usual position of the neo-membrane or new formation is on the upper surface of the hemispheres, extending downward toward the occipital lobe. The changes in the adjacent portion of the brain are dependent on the size and thickness of the neo-membrane. Bartholow observed a case in which the "cyst" was half an inch in thickness at its thickest part, and it depressed the hemisphere correspondingly, the convolutions being flattened, the sulci almost obliterated, and the ventricle lessened one-half in size.

In Pachymeningitis syphilitica, the pathological lesion is in the form of gummatous tumors or masses which may degenerate and become either cheesy masses or be converted into a purulent looking fluid. In old age the dura mater becomes thick, cartilaginous, and of a dull, white color. The sheaths of the arteries are also thickened.

SYMPTOMS. Very obscure; principally those of cerebral pressure. Cases of persistent headache, vertigo, photophobia, anorexia, insomnia, gradual impairment of intellect and locomotion, followed by delirium, and convulsions and coma, or by apoplectic attacks and paralysis; in the aged, or those in whom some one of the causes of the affection are present, an inflammation of the dura mater may be suspected. Circumscribed painful œdema behind the ear and less fullness of the jugular of the corresponding side, the phlegmasia alba dolens en miniature of Griesinger, are indicative of thrombosis in the transverse sinus, as was first shown by Virchow.

DIAGNOSIS. Always problematical, as the symptoms are masked and so obscure that a positive diagnosis is impossible.

PROGNOSIS. Most unfavorable for either forms, although the course of the malady is usually slow. Surgical treatment in traumatic cases offers some hope.

TREATMENT. See General Treatment for Brain Troubles.

ACUTE MENINGITIS.

SYNONYMS. Acute leptomeningitis; cerebral fever; arachnitis.

DEFINITION. An acute exudative inflammation of the cerebral pia mater and arachnoid membranes, usually limited to the convexity of the cerebrum; characterized by fever, vomiting,

headache, delirium, and followed by symptoms of general collapse.

CAUSES. During the course of the acute infectious diseases; erysipelas; associated with or a sequela of influenza. Cerebral overwork; prolonged wakefulness; acute alcoholism; exposure to the sun; disease of the internal ear; secondary to diseases of serous membranes. Most frequently in early adult life and in young children, and in males rather than females. "The micro-organisms found in meningitis are the pneumococcus, streptococcus pyogenes, intracellular diplococcus, the pneumo-bacillus, and a bacillus resembling that of typhoid fever." (Dana.)

PATHOLOGICAL ANATOMY. The inflammatory changes may be limited either to the convexity or to the base of the brain, but more frequently both portions are involved.

Intense hyperaemia of both membranes, followed by a purulent and fibrinous exudation. The ventricles may be filled with fluid, compressing and flattening the convolutions.

In twenty-five post-mortem examinations at the Philadelphia Hospital a meningo-encephalitis was present in fourteen.

SYMPTOMS. Vary according to the stages:—

Prodromes—Headache, vertigo, cerebral vomiting, more or less feverishness, continuing from a few hours to one or two days, when occurs the

Stage of Invasion.—Onset sudden, with chill, high fever, 103 degrees to 104 degrees, pulse 100-120, face flushed, with congested eyes, headache, most intense and continuous, ringing in the ears, photophobia, vertigo, the nausea aggravated, projectile vomiting, with delirium.

Stage of Excitation.—General sensibility of the body increased, sensitiveness to light, and acuteness of hearing, delirium furious, often resembling mania, continual jerking of the limbs, oscillations of the eyeballs—nystagmus—twitching of the muscles, even to the extent of opisthotonos, and in children convulsions. Duration, from one day to a week or two. The finger drawn across the surface leaves a red line—the *tache cerebrale*.

Stage of Depression or Collapse.—The patient gradually becomes more quiet, the delirium subsiding, as well as the muscular agitation; somnolence develops, passing into coma, at times temporary consciousness, coma soon following again; pulse irregular and slow, fever less; various palsies, to-wit: strabismus, ptosis, pupils uninfluenced by light, mouth drawn to one side,

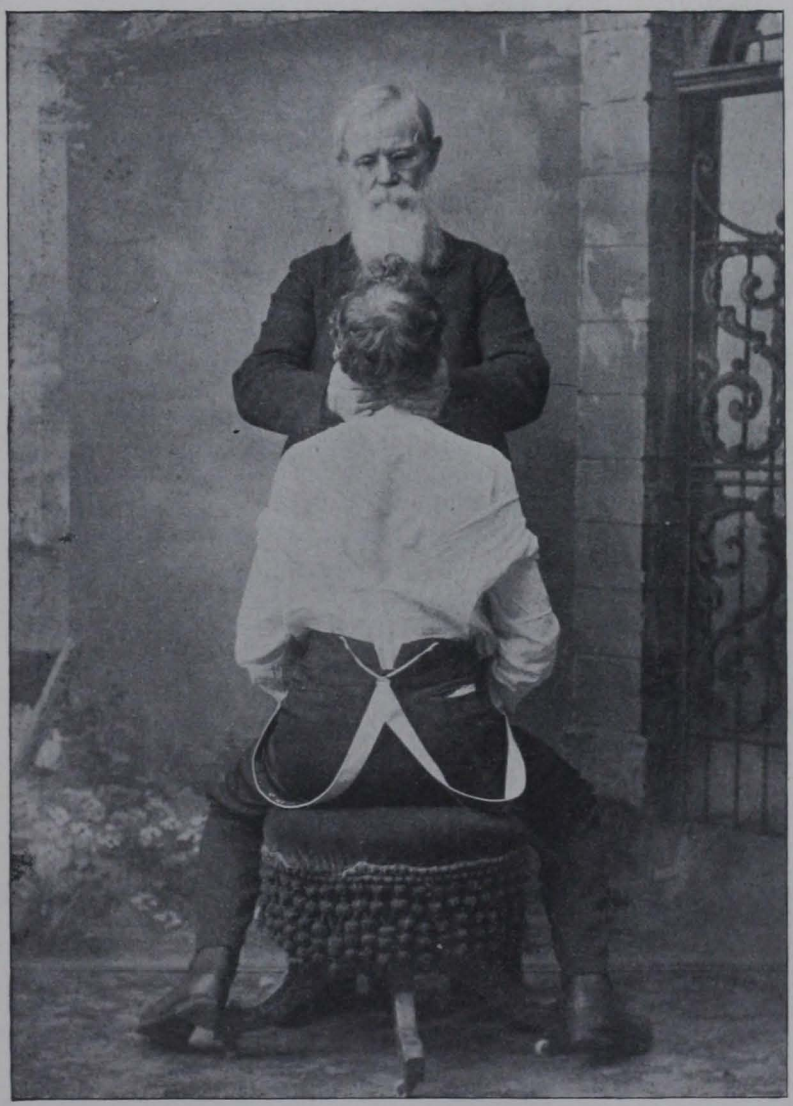


PLATE XLV.—Method of Stimulating Vaso-motor Nerves.

urine and faeces involuntarily discharged. Death following, either by convulsions or by deepening coma with cyanosis.

DIAGNOSIS. The characteristic symptoms indicating the existence of acute meningitis are headache, vomiting, fever and delirium, all developing rather rapidly. The headache is more persistent, the vomiting not due to gastric trouble. The absence of any one of the four characteristic symptoms named above does not prove the absence of meningitis, nor does the combination of delirium and fever alone determine the presence of meningeal disease.

Cerebro-spinal fever closely resembles acute meningitis, the points of distinction between which are the first named occurring epidemically, associated with marked spinal symptoms and an eruption. Meningitis and abscess of the brain are apt to be mistaken for each other, the differential diagnosis being pointed out in that disease.

The cerebral symptoms of rheumatism are differentiated from idiopathic meningitis by the association of the joint trouble. Cerebral symptoms of typhoid and typhus fever have a close resemblance to idiopathic meningitis, and are only determined by a study of the clinical history.

In acute uraemia the face is turgid, œdematous, with puffiness of the eyelids; in meningitis the face is pale and no œdema; uraemia has decided albuminuria; it is slight or absent in meningitis; meningitis has chills followed by fever; uraemia has irregular temperature record rapidly rising to 104 degrees F. to 106 degrees F., and dropping to 99 degrees F., to as rapidly rise again, and usually associated with convulsions.

In delirium tremens the delirium is a busy one, the patient imagining persons and animals around him, and is wild in his gestures and utterances; the temperature is normal or subnormal, the skin wet and clammy. In meningitis the delirium is mild but incoherent, the surface is hot and dry, and there are severe vomiting and headache.

PROGNOSIS. Not very favorable. If recognized early and treated, a fair number of recoveries occur, but it usually leaves the patient subject to attacks of epilepsy or with a persistent headache, and more or less mental impairment.

TREATMENT. See General Treatment for Brain Troubles.

TUBERCULAR MENINGITIS.

SYNONYMS. Basilar meningitis; acute hydrocephalus.

DEFINITION. An inflammation of the leptomeninges (soft membranes), most particularly the basal pia mater, attended with or due to the deposit of gray miliary tubercle; characterized by gradual decline of the bodily and mental powers.

CAUSES. Usually a secondary affection, a sequel to tubercular disease of some other organ. Most frequently occurs in children between two and six years of age, although numerous cases are reported occurring between twenty and thirty years; scrofulous diathesis; inherited diathesis. The "gelatinous children of albuminous parents," as the phrase goes, possess a special susceptibility to tubercular meningitis.

PATHOLOGICAL ANATOMY. The deposition of tubercle usually occurs at the base of the brain.

Depositions of grayish-white granules, of a translucent, somewhat gelatinous appearance—miliary tubercle, are distributed along the vessels of the pia mater, resulting in inflammation and the exudation of lymph, with the consequent thickening and opacity of the membranes. The cerebral tissue is not usually involved, although on section the lines indicative of blood vessels are very much increased in number. The ventricles are distended by a clear, or milky, or even bloody serum. Tubercular deposits occur in the lungs, intestines, and, at times, in other organs. The presence of the tubercles alone may give rise to no symptoms until the exudative products of the resultant inflammation develop.

SYMPTOMS. The advent is either gradual and insidious, or with convulsions, in which cases the after progress is rapid.

Prodromes.—The child grows irritable, with loss of appetite, loss of flesh, swollen abdomen, constipation alternating with diarrhea, irregular attacks of feverishness, with attacks of grinding its teeth during sleep, or sleeplessness. Headache occurs, as shown by the child, even when at play, suddenly stopping and resting its head on its hand or on the floor. Duration of this stage is from one week to a month or two.

Stage of Excitation.—The onset is rather sudden, with obstinate vomiting, severe headache, convulsions, fever, 102 degrees to 103 degrees in the evening, falling to 99 degrees in the morning, pulse soft and compressible, with irregular rhythm. On drawing the finger nail lightly over the surface a red line results, "the cerebral stain" of Trousseau. The symptoms grow progressively

worse with exaltation of the special general senses; the least pinch or even touch causing exquisite pain; spasmodic movements of the muscles, with contraction and rigidity, at times opisthotonos. Duration of this stage is about two weeks.

Stage of Depression.—The result of the pressure of the exudation; the pulse slow and compressible, with irregular rhythm; temperature depressed; tendency to somnolence alternating with quiet delirium, mental stupor, continual movement of the fingers, as in picking up objects; convulsions from time to time, strabismus, oscillation of the eyeballs, followed by intervals of wakefulness, when the headache is excruciating, causing the peculiar, unearthly shrill cry or shriek, "the hydrocephalic cry," associated with contraction of the muscles of the face, as if suffering were experienced; finally collapse, occurring with the "Cheyne-Stokes" respiration, the coma deepening, followed by death, convulsions ending the scene. Duration, from a day or two to two weeks.

Diagnosis. Acute meningitis and tubercular meningitis have closely analogous symptoms during the stage of excitation, but the history and clinical course of the two maladies determine the diagnosis.

Prognosis. Unfavorable. Usual duration, three or four weeks after fully developed prodromes. If ushered in by convulsions, the duration is shorter.

Treatment. See General Treatment for Brain Troubles.

Diseases of the Cerebrum.

CONGESTION OF THE BRAIN.

Synonyms. Cerebral hyperaemia; cerebral congestion.

Definition. An abnormal fullness of the vessels (capillaries) of the brain; active, when arterial fullness; passive, when venous fullness; characterized by headache, vertigo, disorders of the special senses, and, if hyperaemia be decided, convulsions.

Causes. Active.—Increased cardiac action, the result of hypertrophy of the left ventricle; general plethora; excesses in eating and drinking; acute alcoholism; sunstroke; prolonged mental labor; diminished amount of arterial blood in other parts, the result of the compression of the abdominal aorta; ligation of

a large artery, and the suppression of an habitual bleeding hemorrhoid are examples. Passive.—Dilatation of the right heart; pressure upon the veins returning the cerebral blood.

While congestion of the brain is not so common as was once supposed, the view that it can not occur is disproven by the results following the inhalation of a full dose of amyl nitris. The relief of head symptoms after a free epistaxis and the distress resulting if it does not occur is another instance.

PATHOLOGICAL ANATOMY. The post-mortem appearances are, overloading of the venous sinuses and of the meningeal vessels, including the finer branches; the pia mater appears vascular and opaque; the gray matter of the convolutions unduly red; the convolutions may be compressed and the ventricles contracted, with the displacement of a corresponding amount of cerebro-spinal fluid. Long continued or repeated congestions lead to enlargement and tortuosity of all the vessels, a moist and slimy condition (œdema) of the cerebral substance, and an increase in the sub-arachnoid fluid.

SYMPTOMS. "Rush of blood to the head" may be gradual or sudden in its onset, the symptoms aggravated by the recumbent position. Headache, with paroxysmal neuralgic darts, disorders of vision and hearing, buzzing in the ears and sparks before the eyes, contracted pupils, vertigo, blunted intellect, inability to concentrate the mind, irritable temper, and curious hallucinations. The face is red, the eyes congested, and the carotids pulsating. The sleep is disturbed by dreams and jerkings of the limbs. If the attack be sudden (apoplectiform), sudden unconsciousness with muscular relaxation occur. Cerebral hyperaemia in children often presents alarming symptoms, such as great restlessness, insomnia, night terrors, gnashing of the teeth during sleep, vomiting, contraction of pupils, followed by general convulsions. Any or all of these symptoms may continue more or less marked from an hour or two to a day, the child enjoying its usual health, after a sound sleep, save some fatigue.

PROGNOSIS. Mild cases terminate favorably in a few hours to a day or two, but show a strong tendency to recur. Severe cases (apoplectiform) may terminate in health, but usually foretell cerebral hemorrhage.

TREATMENT. See General Treatment for Brain Troubles.

CEREBRAL ANÆMIA.

DEFINITION. An abnormal decrease in the quantity of blood in the cerebral vessels; general, when the diminished supply includes all the vessels; partial, when the diminished supply is limited in area; characterized by pallor, headache, vertigo, some loss of power, and, rarely, convulsions.

CAUSES. Partial cerebral anaemia results from obstruction of a vessel, from embolism or thrombosis. General cerebral anaemia results from hemorrhages, wasting diseases, during convalescence from severe attacks of fevers, sudden shock, feeble cardiac action, and general anaemia.

PATHOLOGICAL ANATOMY. The blood in the brain is contained in the arteries, capillaries, and veins. The functional condition of the brain depends on the quantity and quality of the blood circulating in the cerebral capillaries. Any decrease in the normal quantity or impairment in the quality produces the symptoms of cerebral anaemia. The brain is pale and milky in color, and on transverse section there are no bloody points; the ventricles and perivascular lymph spaces are well filled with fluid. In partial anaemia the local conditions differ somewhat from the above.

SYMPTOMS. General.—Headache, relieved by the recumbent position; vertigo, aggravated by exertion; general pallor, and anaemia, with attacks of fainting; when the general cerebral anaemia is sudden and decided, convulsions occur. In partial anaemia, sudden loss of power, of limited muscular area, gradually returning to the normal condition.

PROGNOSIS. Favorable in all cases save those the result of severe and repeated hemorrhages.

TREATMENT. See General Treatment for Brain Troubles.

CEREBRAL HEMORRHAGE.

SYNONYMS. Apoplexy; "a stroke."

DEFINITION. The sudden rupture of a cerebral vessel and escape of blood into the cerebral tissue, causing pressure and more or less destruction of the brain substance; characterized by sudden unconsciousness, irregular, noisy respiration, and complete muscular relaxation.

CAUSES. Rare under forty years of age. The principal cause is disease of the vessels—the development of miliary aneu-

risms, or a chronic endarteritis with an associated cardiac hypertrophy; hereditary tendency; Bright's disease; syphilis; alcoholic and dietary excesses; gout. More frequent in the spring and autumn.

PATHOLOGICAL ANATOMY. The most common locations of cerebral hemorrhage are the internal capsule, corpus striatum, and thalamus opticus; less common the anterior and middle cerebral lobes and the cerebellum; next in frequency the pons and medulla oblongata; and rarely on the convexity of the brain, termed meningeal hemorrhage. When the hemorrhage is large, the blood may break into the ventricles and pass by the iter from the third to the fourth ventricle. A recent clot is dark in color, and in consistency a soft, grumous mass, composed of coagulated blood and brain substance in varying proportions, at whose center is the opening into the ruptured vessel. The clot excites inflammation around it, resulting in its being encysted, by the development of new connective tissue from the neuroglia, and then gradually absorbed, leaving a cicatrix; or the brain tissue around the clot softens and degenerates—localized softening.

SYMPTOMS. The attack may occur suddenly as an apoplectic shock or stroke, or slowly with prodromes or "warnings."

Prodromes.—Headache, vertigo, transient deafness or blindness, sensation of numbness of the extremities, with local palsies, together with the constant dread of an attack.

The attack begins with vomiting, followed by either partial or complete insensibility; respiration slow, irregular, and noisy; during the inspiration the paralyzed cheek is drawn in, and puffed out in expiration; pulse slow and full; pupils uninfluenced by light, the face flushed, the eyes congested and the carotids throbbing; the temperature declines below the normal, a degree or two, but rises within twenty-four hours to 100-101 degrees F. In fatal cases it may rapidly rise to 107-108 F.

The muscular system is profoundly relaxed, and the reflex movements are abolished. The head and eye deviate, in many cases, toward the affected side in the brain, or from the paralyzed side. Rarely convulsions occur.

Ingravescent apoplexy begins as a mild stroke with a rapid return to consciousness and power, except, perhaps, of speech. Headache is present with some one or more local symptoms, and in a few hours to a few days consciousness gradually becomes impaired, the loss of power again occurs, the coma deepens, the patient dying comatose. If the unconsciousness continues longer

than twenty-four hours, death is the usual termination, preceded by pale face, irregular and rapid pulse and respiration, and rise of temperature.

Reaction obtains in from a half to three hours, consciousness returning, reflex excitability reviving, associated with headache, confusion of mind, and more or less paralysis of motion, and sensibility of one side of the body, termed hemiplegia.

The electro-excitability of the paralyzed parts is preserved.

Recovery may be delayed by inflammatory symptoms, the temperature rising to 101-104 degrees F., with tonic contractions (early rigidity) of the paralyzed muscles and severe neuralgic pains.

Localization of the lesion of a cerebral hemorrhage is of great practical importance.

Capsular hemorrhage, the most frequent, causes loss of consciousness, of sudden or rapid onset, hemiplegia, involving face, arm, and leg, with motor aphasia if the hemiplegia be on the right side. There is also a unilateral loss of reflex action, conjugate deviation of the eyes from the paralyzed side, and unilateral defective movement with flaccidity of the limbs.

Cortical hemorrhage, localized unilateral paralysis of the face, the arm, or the leg, with local convulsions or convulsions that have a local beginning, or profound unconsciousness.

Centrum ovale hemorrhages resemble the cortical with the local convulsions.

Crus-cerebri hemorrhage, loss of consciousness with hemiplegia involving the lower half of the face and the limbs, with paralysis of the third nerve on the opposite side, or the side of the lesion. The unilateral third nerve symptoms are ptosis, external strabismus, dilatation of the pupil, and loss of accommodation for near objects. The paralysis is termed "crossed" or "alternate" hemiplegia.

Pons hemorrhage causes either general convulsions or irregular convulsions in the legs, bilateral motor paralysis, bilateral anaesthesia, either contracted or dilated pupils, embarrassed respiration, repeated non-gastric vomiting, and high temperature. If the hemorrhage is large, death is sudden or within a few hours, and even if small the prognosis is unfavorable.

Ventricular hemorrhages are generally of the ingravescent variety, and are characterized by a second apoplectic seizure soon after the first, with extension of the hemiplegic symptoms or a relaxation of the muscles from one side to both sides of the body.

Cerebellar hemorrhage varies so greatly in the symptoms that a positive diagnosis can seldom be made.

Meningeal or dural hemorrhage, usually due to a trauma. Two varieties: 1. Infantile meningeal hemorrhage, occurring during labor. 2. Extra-dural hemorrhage, the result of direct injury to the head. The infantile variety presents symptoms of irritation and compression of the cortex, such as convulsions, general or unilateral, rigidity, opisthonos, and either hemiplegia or diplegia. The extra dural variety is almost always the result of fracture or trauma of the skull, resulting in an extravasation of blood between the dura and the skull from the middle meningeal artery; the hemorrhage may be on one or both sides. The symptoms may develop at once or after some days, and are those of pressure, hemiplegia, partial or complete, convulsions, impaired or absent reflexes, dilatation with loss of reaction of pupil of opposite side, stupor gradually deepening into coma and death.

SEQUELÆ. Paralysis of the muscles of the face, tongue, body and extremities of one side, opposite to the location of the hemorrhage, termed unilateral paralysis or right or left hemiplegia. Paralysis of both sides of the body, due to simultaneous hemorrhage on both sides, termed bilateral hemiplegia, or diplegia.

Paralysis of one side of the face and the extremities of the opposite side, due to hemorrhage into the pons Varolii, termed alternating or crossed paralysis. Occasionally tonic contractions occur in muscles long paralyzed, termed late rigidity, and is evidence of a secondary degeneration of the nerve fibers. Chronic movements in paralyzed muscles are termed post-hemiplegic chorea, due, according to Charcot, to changes in the motor centers. The mental powers are always more or less permanently impaired, the patient irritable and emotional, and the same holds good concerning the memory.

DIAGNOSIS. The diagnosis of the apoplectic seizure is often one of the most difficult questions in medicine, and yet of the greatest importance, as the treatment hinges on it. The diagnosis of the sequelae is comparatively easy.

Insensibility from drink differs from apoplexy in the following points, to-wit: insensibility is not so complete, no drawing in and puffing out of one cheek with respiration, the pulse frequent instead of slow, the pupils influenced by light; upon raising both legs no difference is apparent on allowing them to drop; the eyes and head are not turned to one side, and lastly, the condition is ameliorated on the inhalation of ammonia. I have satisfactor-

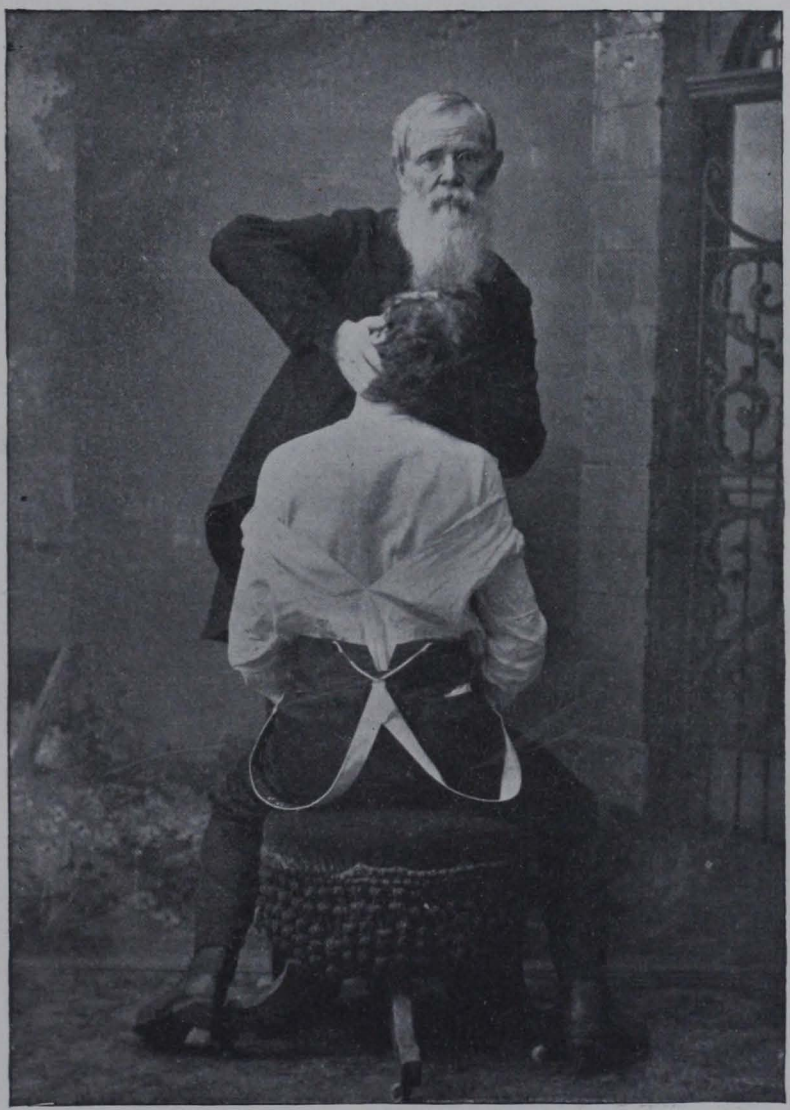


PLATE XLVI.—Extenso-rotary Neck Treatment.

ily used Dr. Von Wedekind's test for temulence, to-wit: "By simply pressing on the supraorbital notches with a steadily increasing force you may, with certainty of success, bring an unconscious alcoholic to his senses, and thus differentiate between alcoholic and other comas."

Opium poisoning differs from apoplexy by the gradual approach of the coma, and that the patient can be momentarily aroused, and also by the absence of the heavy stertor of apoplexy.

Uraemia causes a coma that closely resembles apoplexy. A history of Bright's disease at once clears up the case; again, uraemic coma is generally preceded by convulsions, a rapid rise of temperature as shown by the thermometer, often 104 to 106 degrees F., while to the hand the surface appears but little, if at all, above the normal; the pulse is usually weak with irregular force, the respirations averaging twenty-five to thirty per minute, the face having a glossy appearance.

Cerebral embolism can not always be differentiated from apoplexy. We may suspect cerebral plugging, if the patient be young; if he be laboring under acute or chronic cardiac valvular trouble; if, within brief periods, several incomplete attacks have occurred before a complete comatose condition obtains; or, if hemiplegia results with passing or slight unconsciousness; or, if the phenomena are sooner of later followed by cerebral softening, as embolism and thrombosis are the most common causes of softening.

Syncope or a fainting fit is of sudden onset, but being due to a failure of the circulation, the pulse is feeble, the face pale, the respiration quiet, and the duration of unconsciousness short, all the very opposite of an apoplectic attack.

PROGNOSIS. If the patient survive the immediate effects of a cerebral hemorrhage, he is always in danger of a new attack, since the causes of the original attack still remain. Another attack or two is the usual course, a fatal termination ultimately occurring.

The hemiplegia is uncertain; a partial recovery may occur within a few months, or it may continue for years.

TREATMENT. See General Treatment for Brain Troubles.

CEREBRAL THROMBOSIS AND EMBOLISM.

SYNONYMS. Partial cerebral anaemia; occlusion of cerebral vessels; cerebral apoplexy (?).

DEFINITION. The occlusion of a cerebral vessel, from the formation of a thrombus, or the presence of an embolus, thus causing anaemia of some portion of the brain; characterized by the gradual—when the result of thrombosis, and the sudden, when due to embolism—development of headache, vertigo, disorders of intelligence, with more or less complete insensibility and paralysis.

CAUSES. Thrombosis, or the formation of a clot in the vessel—an ante-mortem coagulation—is almost always the result of chronic endarteritis, as seen in the aged, together with a showing and weakening of the blood current. Chronic alcoholism and syphilis are the usual causes when occurring in young adults. Emboli, in the great majority of instances, result from an endocarditis—cardiac emboli; small particles of the exudation being carried into the circulation and deposited in the brain. Emboli may also be derived from aortic aneurism, or syphiloma of the great vessels.

PATHOLOGICAL ANATOMY. The cerebral arteries may be obstructed by emboli or thrombi; the cerebral veins and sinuses by thrombi only. The changes in the cerebral tissue are those of anaemia of the part or parts supplied by the occluded vessels. The subsequent changes depend upon the anatomy of the vessels. If the obstructed artery has anastomoses, the collateral circulation is soon established and the brain tissue assumes its normal condition. If, on the other hand, the occluded vessel be one of "Cohnheim's terminal arteries"—arteries without anastomoses—the blood in the whole extent of the occluded vessel coagulates, thus preventing the backward flow of blood from the surrounding capillaries, and so obstructing collateral circulation, whence the anaemic tissue dies or undergoes necrobiosis, followed by yellowish-white softening; or, if the vessel beyond the seat of the occlusion remains pervious, blood flows back through the capillaries from the nearest artery or vein; the parts that a short time before were bloodless now become deeply engorged, the succeeding changes in the vessels permitting diapedesis of the red blood globules; the tissues which are undergoing disintegration are colored by the red globules, causing the appearance entitled "red softening," which after some weeks becomes "yellow softening," finally changing to "white softening," when there is a milky, or

rather creamy, fluid mixed with masses or particles of broken-down nerve elements.

The vessel most commonly occluded is the left middle cerebral artery, which sends branches to the second and third frontal convolutions, the anterior and superior portions of the three temporal convolutions, the island of Reil, the parietal convolutions, part of the external and all of the internal capsule, the lenticular nucleus, and most of the corpus striatum—the motor centers.

SYMPTOMS. Two distinct modes of onset: gradual, when the result of thrombosis; sudden or apoplectic, when due to embolism.

Cerebral Thrombosis.—Most common in the aged. Persistent headache and vertigo, at one time severe and at another mild. Next, alterations in patient's character; irritable, morose and despondent, with periods of absent-mindedness, disorders of vision, and impairment of memory, speech becoming hesitating and mumbling. Impaired locomotion, the result of the vertigo, and of muscular weakness and trembling, followed sooner or later by hemiplegia, which may be preceded by sudden insensibility or occur gradually, the symptoms slowly proceeding to senile dementia and death from exhaustion; or, rarely, the symptoms are not so grave, and partial or complete recovery occurs after the hemiplegia, from establishment of the "collateral circulation."

Cerebral Embolism.—The symptoms are sudden, but either mild or grave in character.

Mild Variety.—Sudden and severe vertigo, confusion of mind, muscular twitchings, usually one-sided, and vomiting, followed by hemiplegia, most frequently of the right side, the intellect clear but hesitating. After some weeks or months the paralysis usually disappears and recovery is complete.

Grave or Apoplectic Variety.—Sudden headache, vertigo, flushing or pallor of the face, or the patient may utter a sharp cry, fall to the ground with sudden unconsciousness and complete muscular relaxation, followed by death, or a gradual return of consciousness with hemiplegia, which is generally right-sided, with aphasia, remaining for several weeks or months, or is persistent, the mind remaining normal or enfeebled and the emotional nature highly excitable and the reason and judgment clouded, continuing thus for years, or gradually developing into dementia, exhaustion and death.

The following are some of the symptoms of "localization" if particular vessels are blocked:

Vertebral artery, the left most frequently, which results in

acute bulbar paralysis from involvement of the nuclei in the medulla, associated or not with hemiplegia.

Basilar artery, which causes diplegia with bulbar symptoms. There is rapid rise of temperature. Death follows within a day or two, or suddenly, if respiratory centers are involved.

Middle cerebral artery is the most frequent seat of embolic or thrombotic occlusions. The symptoms depend upon the exact branch involved: if plugged before the central arteries are given off, the internal capsule is deprived of its blood supply and permanent hemiplegia may follow; if the blocking is in the central branches the hemiplegia involves the arm and face, and if left side, aphasia occurs. The individual branches passing to the third frontal (aphasia), the ascending parietal (hemiplegia, particularly hand), supra-marginal and angular gyri (word blindness), and the temporal gyri (word deafness), may be plugged.

DURATION. Thrombosis, essentially an affection of the elderly, has a chronic course. Months and years may be occupied with the various symptoms until the phenomena of senile dementia develop. Embolism is of sudden onset, and may be followed by a rapid recovery.

DIAGNOSIS. Thrombosis is associated with changes in the vessels, the arcus senilis, and other evidences of senile degeneration.

Embolism may be mistaken for cerebral apoplexy, and while a positive differentiation can not always be made, the chief point to be considered is the presence of cardiac murmurs.

PROGNOSIS. Thrombosis is a permanent and progressive condition in the majority of instances. Recovery is a rare termination. Embolism may be followed by a perfect recovery. Usually, however, some evidences of the plugging remain permanently. Death may be the result within a day or two, from the plugging of a large vessel, the patient never emerging from the coma. In other cases the patient arouses from the coma, the hemiplegia with aphasia persisting, and the case pursues the usual course of localized cerebral softening.

TREATMENT. See General Treatment for Brain Troubles.

CEREBRAL ABSCESS.

SYNONYMS. Acute encephalitis; suppurative encephalitis.

DEFINITION. An acute suppurative inflammation of the brain structure, either localized or diffused, primary or secondary;

characterized by impairment of intellect, sensation, and motion.

CAUSES. Primary cerebral abscess is exceedingly rare. Pyaemia; glanders; embolus from ulcerative endocarditis. Secondary cerebral abscesses result from injuries to the cerebral tissues, to-wit: apoplexy, embolism, thrombosis, and injuries to the cranial bones. Chronic ear disease; chronic suppuration in some other portion of the body.

PATHOLOGICAL ANATOMY. Abscess of the brain affects the left side more frequently than the right. They are usually encysted or inclosed in a limiting membrane. Abscesses of the brain may be single or multiple, varying in size from an almond to an egg. It occupies a limited and well-defined region of the cerebral tissue, to-wit: either corpora striata, optic thalami, gray matter of the cortex, the cerebellum, or the white matter of the hemispheres.

"The initial stage at the site of the abscess is hyperaemia. Minute extravasations take place (capillary hemorrhages), giving to the inflamed area a dark, reddish color, whence the term red softening. Migration of white corpuscles, diapedesis of some red corpuscles and exudation of serum holding albumen and fiber in solution, occur simultaneously. The brain tissue, being soft and easily broken up, is rapidly dissociated and its elements disintegrated, and in a short time a soft, pultaceous, red mass results, which more and more assumes a purulent character, becoming first reddish-yellow, then yellow or greenish-yellow, ultimately almost white. The injury caused by an abscess is not limited to the portion of the brain inflamed, but the neighboring territory is in the condition of collateral hyperaemia and œdema." (Bartholow.)

SYMPTOMS. A concise description of the symptoms of abscess of the brain is very difficult, on account of the wide variations dependent on its location, and also the difficulty of isolating it from the affections to which it is secondary. The onset varies according to the cause, although all cases are associated with headache, irritative fever, vomiting, persistent and spreading paralysis, convulsions, optic neuritis, mental apathy, delirium, and coma. If following apoplexy, thrombosis, or emboli, there occur fever and delirium, the paralysis remaining and spreading with spasmodic contractions of the affected muscles.

Occasionally cases run a chronic course, the onset rather insidious; dull, persistent headache, changed disposition, peevish,

irritable, unreliable, with decline of moral sensibility; easily fatigued by mental work; inability to stand exertion; memory impaired; vertigo; dyspepsia, soon followed by slight palsies, which progressively increase, becoming general, with involuntary discharges, death following from exhaustion.

Of the focal symptoms, hemiplegia, of incomplete character, occurs in about one-half of all cases of abscess of the brain. A very constant symptom of diagnostic value, when hemiplegia is very marked, is exaggerated knee-jerk with pronounced ankle clonus.

DIAGNOSIS. A positive diagnosis is only possible by a close study of the causes and the clinical history, as the symptoms at times indicate meningitis and again cerebral tumor.

Purulent meningitis may follow trauma to the brain or chronic ear disease, making the diagnosis impossible. The chief points of distinction are, the subacute or chronic course of abscess (rarely an acute course), slight involvement of cranial nerves, hemiplegia, and the presence of an active, persistent, unilateral ankle clonus and exaggerated knee-jerk on paralyzed side.

PROGNOSIS. The usual termination is in death. The course depends upon the character and extent of the injury, varying from a few days to several months.

TREATMENT. See General Treatment for Brain Troubles.

INTRA-CRANIAL TUMORS.

SYNONYM. Cerebral tumors.

DEFINITION. Tumor of the brain is either a growth in the cerebral tissue, on the meninges, or in the vessels; characterized by symptoms of pressure upon the brain structure.

CAUSES. Injuries to the head; syphilis; changes in the vessels; tubercle and cancer; heredity.

PATHOLOGICAL ANATOMY. The size of tumors vary, and may become as large as an orange before they will give rise to symptoms. Tumors of the brain are of various kinds, to-wit: vascular tumors—aneurisms; parasitic tumors—cysticercus; diathetic tumors—tubercle or syphilis; accidental tumors—glioma. Whatever the character of the growth, it produces irritation of the surrounding parts, and by pressure, destruction of the tissues, or it interferes with the arterial or venous flow.

SYMPTOMS. Those common to tumors in general are, head-

ache, persistent and increasing in intensity, defects of vision, even blindness, due to an optic neuritis, a very constant symptom; defects of hearing, taste, and of speech, the result of paresis of the vocal cords; vertigo, associated with nausea and vomiting; convulsions, epileptiform in character, usually limited to one side of the body, occurring at regular intervals, or confined to the eyeballs (nystagmus), or one limb, with no loss of consciousness; palsies, beginning first as strabismus, ptosis and dilatation of the pupil, of the facial muscles, paraplegia and general hemiplegia; defects of sensibility, to-wit: sensations of numbness, and coldness in the limbs and body. Occasionally disturbances of equilibrium, manifested by a tendency to go backward or turn to the right or left; intellectual faculties well preserved until late in the affection, when the memory becomes impaired or lost for certain articles, and finally a gradually advancing imbecility.

DIAGNOSIS. Rarely can a positive diagnosis be made. The following points will aid: long-continued, persistent headache, without appreciable cause, epileptiform convulsions, unilateral, without loss of consciousness, difficulty of vision, hearing, and speech, associated with nausea and vomiting, and local and general palsies.

The location of the tumor may be determined by the more or less pronounced character of certain symptoms. The diagnosis of the character of the growth can only be determined by a close study of the history.

According to Herter, "the indications that suggest that the tumor is a syphilitic growth are as follows: Syphilitic history, symptoms of irritative disease of cortex rather than destructive, evidences of rapid growth at the onset, followed by a period of slow progress or stationary symptoms, gradual improvement under anti-syphilitic treatment, development between twenty and forty-five years of age."

Indications suggesting tubercular growth are: family history of tuberculosis in some other organ of the patient, rapid development of symptoms, indications of the growth in the cerebellum or in the pons, early appearance of the symptoms, especially before the tenth year, and history of injury to head.

Indications suggesting sarcoma or cancer are: the presence of a sarcoma elsewhere and rapidly failing health, with cerebral tumor symptoms in patient over fifty years.

Indications suggesting glioma: sudden loss of consciousness with exacerbation of all symptoms in the clinical history of

cerebral tumor, cortex irritative symptoms as in syphiloma, developing under fifty years of age, and the absence of all evidences of tubercle, syphilis, sarcoma, and cancer.

The focal symptoms of intracranial tumors are so important in diagnosis that the following summary is given of symptoms caused by brain tumors: Prefrontal Region.—Mental impairment, pressure in central region, causing aphasia, Jacksonian epilepsy, and disturbances of smell. Central Region.—Motor aphasia, monoplegia, partial anaesthesia, Jacksonian epilepsy. Posterior Parietal Region.—Word-blindness, homonymous hemianopsia, disturbed muscular sense. Corpus Callosum.—Progressive hemiplegia. Crus Cerebri.—Crossed paralyses of oculomotor nerve and limbs. Corpora Quadrigemina.—Oculo-motor paralyses, reeling gait, possibly blindness and deafness. Pons and Medulla.—Crossed paralyses of face and limbs, or tongue and limbs. Other lesions in cranial nerves. Cerebellum.—Marked cerebellar ataxia, vomiting, convulsions, coma. Base, Anterior Fossa.—Mental enfeeblement, and disturbances of smell and vision, exophthalmos. Base, Middle Fossa.—Impairment of vision; hemiplegia; oculo-motor disturbances. Base, Posterior Fossa.—Trigeminal neuralgia; neuro-paralytic ophthalmia; paralyses of the face and tongue; impaired hearing; crossed paralyses.

Diagnosis Between Cerebral Tumor and Abscess.—Both may have any or all of the following symptoms: headache, vomiting, double optic neuritis, and mental failure. Tumor has in addition, marked focal symptoms, monoplegia, hemiplegia, paralysis of cranial nerves, and marked optic neuritis; the absence of these favor abscess, or if hemiplegia the ankle clonus and knee-jerk are exaggerated. Fever and rigors point to abscess. The causes of abscess are very clear, those of tumor often uncertain.

Prognosis. Unless of syphilitic origin, unfavorable; but it is to be borne in mind that all syphilitic tumors of the brain do not have a favorable termination.

Treatment. See General Treatment for Brain Troubles.

APHASIA.

Definition. The inability to use spoken language or give vocal utterance to ideas. Amnesic Aphasia.—Or loss of the memory of words by which ideas are expressed. Ataxic Aphasia —The inability to combine the different parts of the vocal appar-

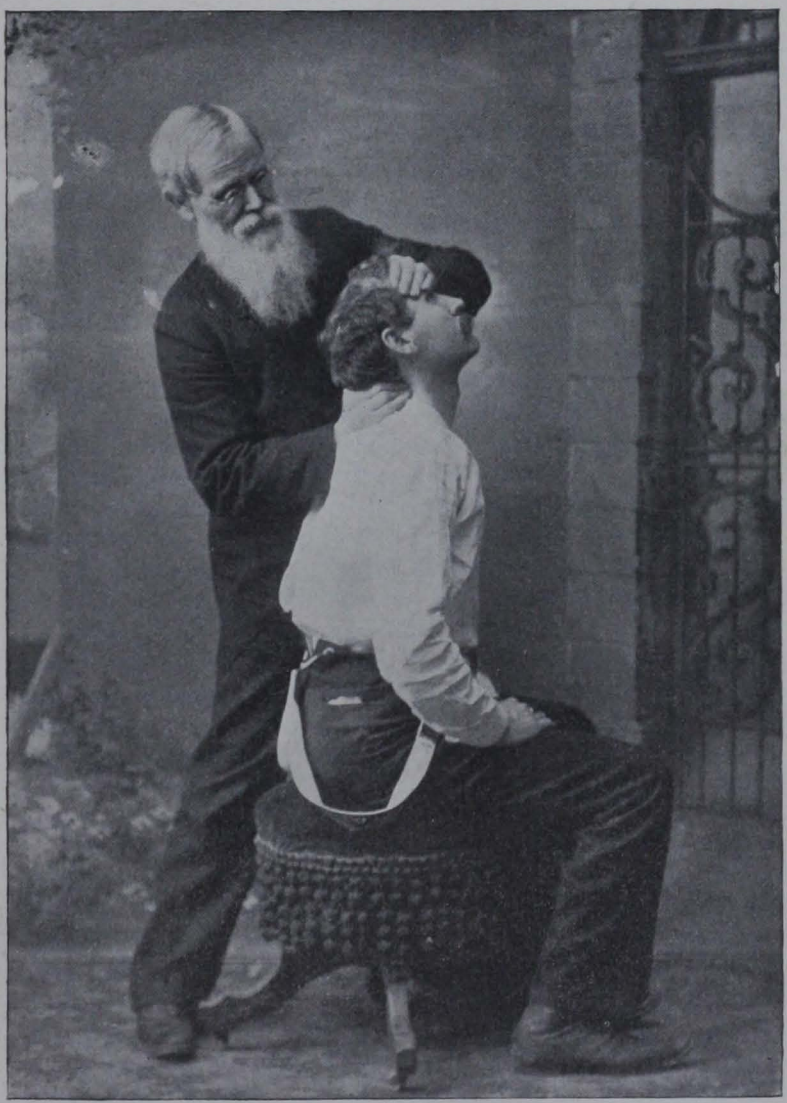


PLATE XLVII.—Manipulating Muscles of Back of Neck.

atus for vocal expression, although the memory of words still remains, so that the afflicted person can write his ideas intelligently. Agraphia.—The inability to recognize and make the signs by which ideas are communicated in written language. Amnesic Agraphia.—The inability to combine the muscular apparatus—"writer's cramp." Paraphasia.—The mental state in which the wrong words are used to express the idea. Paragraphia.—The state in which wrong or meaningless written signs are used to express the idea.

PATHOLOGICAL ANATOMY. The distinction between aphasia and aphonia must be clearly determined.

Aphasia is not the result of any one specific lesion, but occurs during the course of several, to-wit: occlusion of certain cerebral vessels; cerebral hemorrhage; cerebral abscess or softening; meningitis; tumors; mental or moral causes; hysteria.

It is now almost definitely determined that lesions of the left middle cerebral artery, island of Reil, third frontal convolution, and parts of the corpus striatum, are associated in the production of aphasia. The lesions are usually upon the left side of the brain, the aphasia being associated with right hemiplegia.

SYMPTOMS. The degree to which articulate language is impaired varies from the loss of a few words to complete inability to communicate ideas. The intellect does not suffer in proportion to the loss of words; for, showing the individual an article, while he may miscall it, if you call it by name he will recognize it. This inability to convey thoughts is a source of great mental suffering, in some leading to a suicidal tendency.

A strange clinical fact is the strong tendency to profanity shown by aphasic patients.

DIAGNOSIS. Aphonia, or loss of voice, should not be confounded with aphasia, or the inability to remember words. Paralysis of the tongue, or inability to move this organ, thereby interfering with articulate language, should not be confounded with aphasia, which, as a rule, is not associated with paralysis of the tongue.

PROGNOSIS. Controlled entirely by the cause. If the result of congestion of the brain or a syphilitic tumor, the prognosis is favorable. If associated with hemiplegia, the clot may undergo absorption, and recovery follow. If associated with softening of the brain, however, the disease grows progressively worse.

TREATMENT. See General Treatment for Brain Troubles.

VERTIGO.

SYNONYM. Dizziness.

DEFINITION. Vertigo or dizziness is a subjective state, in which the individual affected, or the objects about him, seem to be in rapid motion either of a rotary, circular, or a to-and-fro character.

CAUSES. The etiology of an attack of vertigo depends upon the particular variety.

Ocular vertigo results from the paresis of one or more of the ocular muscles, eye-strain, or astigmatism.

Aural or Auditory vertigo, or Meniere's disease, results from disease of the semicircular canals and cochlea. Meniere's disease, properly so-called, is a sudden severe vertigo, the result of either a hemorrhage or a serous or purulent exudation into the semicircular canals.

Gastric vertigo is the most common variety, and results from either stomachic or intestinal dyspepsia, disordered hepatic function, or constipation. "The mechanism of the vertigo is complex. There are two factors: one consists in the toxic effect of the imperfectly oxidized materials which accumulate in the blood; the other is reflex. An impression made on the end organs of the pneumogastric in the stomach is reflected over the sympathetic ganglia." (Bartholow.)

Nervous vertigo is associated with migraine, sick or nervous headache, and is also caused by physical or nervous excesses, also by the immoderate use of tea, coffee, alcohol, and tobacco. It is also a result of many of the organic diseases of the brain.

Senile vertigo is the result of the disordered cerebral circulation resulting from changes in the heart and vessels.

SYMPTOMS. In all varieties of vertigo, the symptom of a sensation of objects moving around the patient, or the patient moving around objects which remain stationary, is present in some degree. The attack of giddiness comes on suddenly, with an indistinctness of vision and slight confusion of the thoughts. The patient may fall unless he grasps something to steady himself. Nausea and vomiting and cardiac palpitation with tinnitus aurium are often associated with the vertiginous sensations. There is no loss of consciousness.

In the ocular vertigo the attack is usually the result of reading, writing, sewing, or other close application of the eyes, the ordinary symptoms of vertigo being preceded by headache, nausea, specks before the eyes, and pain in the eyeballs.

In Meniere's disease the vertigo is associated with serious tinnitus aurium and the vertiginous sensations are of various forms, such as a see-saw movement, a gyratory motion, right or left; a vertical whirl, or a sensation of rising and falling like unto the swell of the ocean. The symptoms are of long duration, becoming marked in paroxysms. The attack of aggravated vertigo is so sudden and overwhelming at times that the person is suddenly thrown to the ground as if struck with a blow, associated with nausea and vomiting. As the condition continues the character of the individual changes, becoming morose, irritable, and suspicious. Not all cases of Meniere's disease become permanent, but it may occur in isolated attacks, the interval being free from all sensations.

Gastric vertigo is by far the most frequent variety. Persons subject to vertigo of this kind live in constant dread of cerebral disease, which frequently results in true melancholia. The vertiginous sensations usually occur during the course of well-marked and long-standing stomach and intestinal disorders, such as pain or oppression after meals, nausea, pyrosis, heartburn, frequent eructations and constipation or rarely diarrhea. The abdomen is often distended with flatus. Great pain in the nucha is a very frequent occurrence. The attack may be associated with either hyperaemia or anaemia of the brain. The symptoms are not constant, but recur at intervals, sometimes remote, at others very close on each other.

In nervous vertigo the vertiginous symptoms are usually associated with more or less irritability of temper, restlessness, and insomnia. The onset is sudden, after some one of the etiological factors. In megrim there are headache, nausea, and vomiting. This form of vertigo often precedes or replaces the epileptic convulsion; it also often precedes softening of the brain.

In senile vertigo the vertiginous symptoms are the result of anaemia of the brain. The attacks are developed by any exertion, often by merely assuming the erect posture. There is a swimming sensation in the head, darkness falls on the eyes, with a sensation of chilliness and prostration.

Diagnosis. The diagnosis of the various forms of vertigo can only be determined after a close study of the history and course of attack. The existence of organic cerebral disease must always be kept in mind in solving any case.

Prognosis. This will be influenced by the variety of the vertigo. The prognosis is favorable in ocular and gastric vertigo.

Unless the result of organic disease, the prognosis is good in nervous vertigo. In auricular vertigo the prognosis is fair, but in genuine Meniere's disease the prognosis is unfavorable, as it is also in senile vertigo.

TREATMENT. For ocular vertigo, rest for the eyes and properly adjusted glasses. See General Treatment, etc., especially of neck and spine.

MIGRAINE.

SYNONYMS. Megrim; hemicrania; sick headache; bilious headache; blind headache.

DEFINITION. A unilateral paroxysmal pain in the head, periodical, accompanied with nausea, often vomiting, intolerance of light and sound and incapability of mental exertion, the brain for the time being temporarily prostrated and disturbed.

CAUSES. In the majority of patients the nervous predisposition to migraine is inherited, but whether inherited or acquired, it commonly develops before the age of thirty.

Among the many exciting causes are disturbances of digestion, irritation of the ovaries or womb, worry, exacting mental labor, sexual excesses and insufficient sleep, and eye strain. The causes of many attacks, however, are wrapt in mystery.

SYMPTOMS. Attacks of migraine occur in irregular paroxysms, the intervals between being free from pain or nervous disturbance. For a day or two preceding the paroxysm, it will be ascertained, on close questioning, that there was a feeling of fatigue without apparent cause, heaviness over the eyes, with some flatulency and indigestion. The attack proper is ushered in by chilliness, nausea, often vomiting, yawning, and general muscular soreness, with intolerance of light and noises in the ears and incapability for mental exertion and pain of a sharp, shooting character, of great intensity and persistency, localized most frequently in either the frontal, temporal, or occipital regions of the left side; at the same time there is tenderness over the whole side of the head. Rarely the pain is felt on the right side, and still more rarely on both sides at the same time. The nausea and other digestive symptoms may follow the onset of the pain instead of preceding it. There is more or less disturbance of the circulation, temperature, and secretions of the affected parts. At times there is a marked contraction of the vessels, when the face is pale, the eyes shrunken, and the pupils dilated; again, the vessels may

be dilated, when the face is flushed, the conjunctivae injected and the pupils contracted. Motion, sound and light aggravate the acute suffering.

The attack may continue with more or less intensity from a few hours to two or three days, the average duration being twenty-four hours.

DIAGNOSIS. The symptoms are so characteristic that an error seems impossible. It may, however, be confounded with anaemic headache, hyperaemic headache, dyspeptic or bilious headache, and neuralgic or rheumatic headache. The pains of organic brain disease must be excluded.

PROGNOSIS. While few cases of true migraine are permanently cured, the affection is free from danger to life. In a fair number of cases the susceptibility to attacks declines as the person advances in years, it being rarely seen after fifty years. "Cases of migraine of the ophthalmic variety appear to be not rarely followed by general paralysis of the insane." (Herter.)

TREATMENT. See General Treatment for Brain Troubles.

TREATMENT FOR ALL BRAIN TROUBLES.

Whether we consider congestion in the arachnoid or in the substance of the gray or white matter, or simply a venous congestion that produces megrim, the principles involved in the treatment are the same. To repeatedly specify the course to pursue in each particular affection would not only presume stupidity of intellect on the part of the reader, the student of "forces," who has traveled thus far with us in the application of this philosophy, but would increase the size of this volume out of proportion.

The thing to do is plainly stated in other parts of this volume —Take Off the Pressure! This is to be done in the neck, for the most part; for failure in emptying the veins causes all the troubles enumerated in the foregoing pages concerning brain trouble, from a slight headache to a cerebral abscess. To relieve the veins of engorgement means much.

The Manner of Treatment.

To relieve Common Headache occupies but a moment. It is accomplished as follows: The patient sitting down, lying down, or standing up, makes no difference. The operator places the fingers of both hands on either side of the cervical spines, the

upper portion, close to the occiput, and covering the vaso-motor area, with the thumbs on the side of the neck, covering the pneumogastric nerve (in sheath of carotid artery); now press gently on the neck with the fingers, and at the same instant with the thumbs press on the nerves above mentioned. Headache ceases.

Sick headache is treated in the same way, but may require further general treatment—stretching of the neck, manipulating the muscles of the neck, stimulating the spinal accessory nerve, raising the clavicles, treating the chest, liver, stomach and bowels; in fact, for chronic headaches a general treatment is always proper to administer.

Aphasia, Every Variety.—The treatment should be general, from the upper cervix to and including every part of the body. Remember that this affection, like paralysis, is due to extravasation of blood around the terminal footlets of the motor and sympathetic nerves, separating them, incoordinating communication of forces, or intelligence, from and through sympathetic to motor, hence confusion. The rational treatment is, therefore, to unite the footlets by removing the fluids that separate them. The proper way is to open the channels of outlet, the veins, let the venous blood be returned, let the waste material flow through the lymphatics into the veins, thereby removing pressure and reinstating normal conditions. The treatment should begin, then, at the back of the neck (vaso-motor area), then spring the neck strongly as recommended in general treatment, stretch the whole spinal cord, putting one hand at the occiput, the other under the chin; pull gently until the feet are seen to move, rotate the head while extension is being made, and at the same time the head is turned press the fingers of the hand under the neck strongly against the sides of the vertebrae, just back of the mastoid process. Change hands and repeat the process. Then give general treatment.

Locomotor Ataxia.—General treatment every other day is required for months (perhaps years) in long-standing cases. The higher up the lesion, the lower the effect. The treatment is to be thorough, freeing all pressure everywhere in the body. The only way to reach these lesions is through the sympathetic nerves, reflexly; and the great trunks coming out of the skull should be especially looked after. Spinal accessory, pneumogastric and cervical ganglia on both sides of the neck and clear down the spinal column, including ganglion impar, as well as those in the sphincters of the lower outlets of the body, remembering that

sympathetic nerve filaments exercise a controlling influence all over the body, and we reach the nerve centers only through the terminal ends or footlets of nerves. To specify each particular move seems superfluous, and, should the reader feel at a loss to know what to do in a particular case or condition, by reference to illustrations and studying them, the movements necessary in each indicated case may be readily selected, or the general treatment understood. Attention is always to be directed to the parts obstructed or structure involved, and how to reach it through the nervous system or circulatory apparatus easily, for the illustrations represent all the movements for the general and special, practical application of the science in the treatment of diseases. The general treatments covering the parts affected usually suffice in any given case. Therapeutic suggestion is a valuable means, deserving special consideration.

Cerebro-Spinal Ganglia.—The reflexes are forcibly illustrated in the irritation of the clitoris, sending a thrill all over the system, and when long irritated, exercises a prostrating influence upon the whole spinal nervous system. Moderately but firmly pressed upon, produces great contractive influence upon the fundus of uterus, producing expulsive parturient pains, both for delivery of foetus and afterward for disengaging and expelling the placenta. Contraction on the ends of the sympathetic filaments in the external uterine sphincter muscle causes sickness at the stomach. Sudden jerking of cilia (a wisp of) on the mons veneris immediately arrests uterine, post-partum hemorrhage. These actions show us some effects the sympathetic nervous system produces when we know how to use it, or suggest to it what our desires are, and how to utilize forces that are not found in medicines. These are only a few of the marvelous effects derivable from this same set of tubes called nerves. If the pressure on these end filaments produces such marvelous effects, imagine a pressure on millions of these nerves along the spinal column and over the whole body, for this same system of influences predominates everywhere in our bodies, superintending and controlling them whether we wake or sleep, provided the communication continues intact. To know how to use this marvelous system correctly, gives us complete control over disease.

ALCOHOLISM.

VARIETIES. Acute alcoholism; chronic alcoholism.

SYNONYMS. Acute variety, temulentia; mania-a-potu. Chronic variety, delirium tremens; dipsomania or oinomania. It would hardly be correct to consider these terms interchangeable; they are rather names applied to various conditions due to acute or chronic alcoholic poisoning.

DEFINITION. Alcoholism is the term used to designate the physical and mental phenomena induced by the use of alcohol.

Temulentia, meaning drunkenness; mania-a-potu is an acute mental derangement, occurring in those of strong neurotic tendencies; delirium tremens is an attack of delirium associated with tremors in persons with the numerous changes resulting from chronic alcoholism. Delirium tremens results in alcoholics suffering from some form of nephritis, preventing the elimination of some poison developed from the ingested alcohol. Dipsomania or oinomania, an alcoholic insanity in which an individual at longer or shorter intervals has paroxysms of alcoholic desires, between which he neither wishes nor craves alcohol.

CAUSES. Predisposing causes are influences arising from unfavorable moral, social, and personal conditions. Heredity. Exciting causes are the immoderate use of alcoholic beverages, of which there are three groups: 1. Spirits, or distilled liquors. 2. Wines, or fermented liquors; and, 3. Malt liquors.

PATHOLOGICAL ANATOMY. Acute Alcoholism.—The brain is the seat of an active hyperaemia; the mucous membrane of the stomach and duodenum is markedly injected and covered with a ropy mucus slightly tinged with blood, and the gastric juice is altered in quality and quantity. The kidneys are also the seat of an active hyperaemia.

Chronic Alcoholism.—In this condition of the economy there are no organs or tissues which do not present morbid changes. The gastro-intestinal mucous membrane presents the changes of chronic catarrhal inflammation; the liver, the first organ to receive the poison after the stomach, presents the changes of congestion, cirrhosis, or fatty degeneration; the kidneys show chronic congestion, and often the changes are incident to chronic interstitial nephritis. The post-mortem results found in twenty-five cases of delirium tremens dying in the Philadelphia Hospital, were fourteen with the changes of interstitial nephritis, eight with chronic parenchymatous nephritis, and three with fatty kidney; all showed chronic gastric catarrh and changes in the myocar-

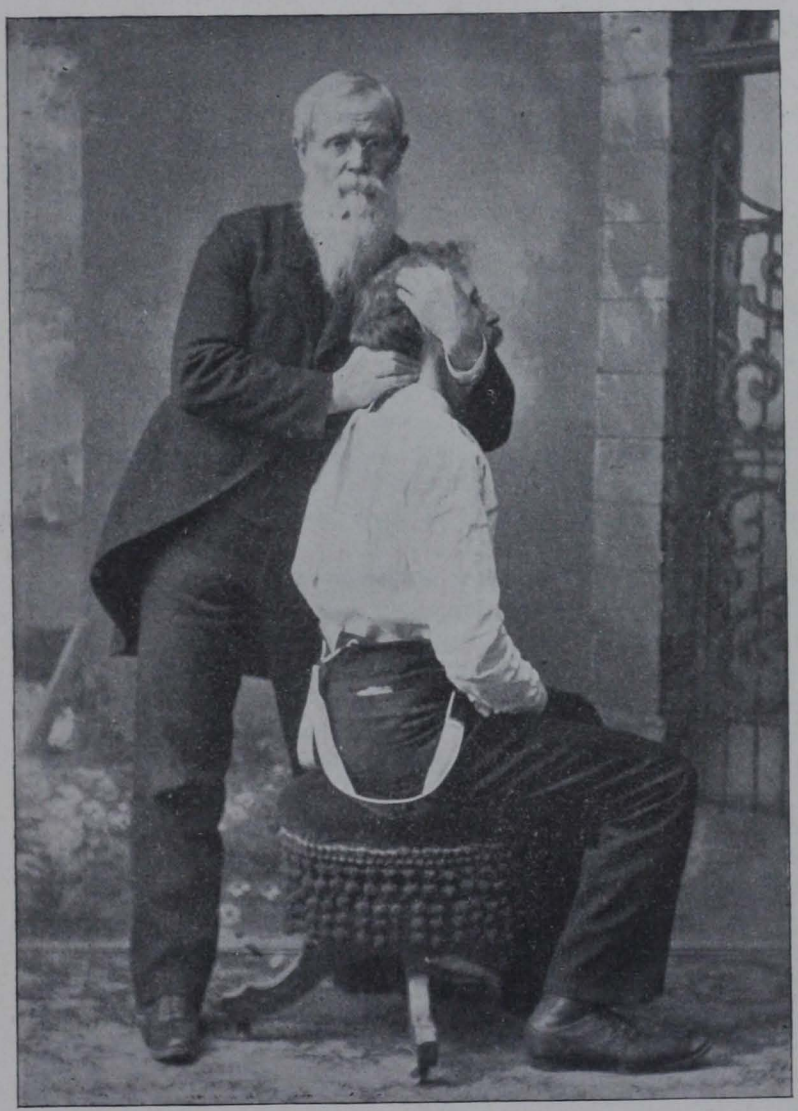


PLATE XLVIII.—Chin–Occipital–Neck Extension.

dium and the arteries of the heart, brain, and the aorta. The muscular structure of the heart may undergo fatty degeneration and the vessels the senile changes of the aged. The brain structure presents the changes of sclerosis in various stages, and there may be chronic meningitis and pachymeningitis with haematoma. The nerves are altered, atrophied, and hardened, and the neuroglia, vessels, and ganglion cells of the spinal cord show similar changes.

SYMPTOMS. Acute alcoholism, resulting from the use of a large quantity of alcoholic fluid, occurs with symptoms of mild intoxication, to drunkenness passing to acute delirium and acute coma. The condition begins with a period of exhilaration, passing to semi-delirium, and ending in an acute coma, when the breathing is stertorous, the face bloated and congested, the lips swollen and purplish, the pupils contracted, the pulse feeble and slow, the skin cold and clammy, the temperature depressed and frequently control of sphincters lost. An individual so affected is said to be "dead drunk." The cases of ordinary drunkenness do not often pass beyond the stage of exhilaration, ending in a mild coma or sleep.

Mania-a-potu—Or acute alcoholic delirium, is the direct result of alcoholic excess in those engaged in a sudden debauch, or who have drunk alcoholic beverages very "hard" for a comparatively short period. The individuals grow more and more excitable, lose all desire for food, are unable to sleep, become the prey of horrible hallucinations—"the horrors"—finally terminating in mania which resembles delirium tremens in all save the tremor, which is absent.

Chronic Alcoholism.—The condition to which this term has been given is truly a disease. It is the result of the continued use of alcoholic beverages until one or more of the morbid organic changes have occurred. These persons are markedly dyspeptic, with coated tongue, fetid breath, and early morning vomiting, straining, or retching, attended with much distress. There is a gradually developing muscular tremor, progressing to the ataxic gait, and insomnia. The face may either become pallid, flabby, and bloated, with an imbecile expression, or swollen, rough, and dusky, with great bladders under the eyes, with yellow injected conjunctivae. There are headache, vertigo, and attacks of hallucinations; the memory grows weaker, the judgment less accurate, the moral sense blunted, and the will power weak and erratic. These and many other symptoms add to the distress of

the individual, which he attempts to overcome by the use of more and more of the poison.

Delirium Tremens.—In the majority of instances delirium results from a prolonged debauch, in an old drinker. It begins by an increased tremor, insomnia, irritable, excitable manner, followed by the characteristic hallucinations and illusions, during which snakes and all forms of repulsive reptiles are seen, causing the most intense horror and abject fear. There also occur illusions of smell and hearing. This marked excitement is followed by great depression, the skin is cold and clammy, the pulse feeble, the muscular system weak, the mind in a condition of coma-vigil, and a febrile condition typhoid in character develops. Uraemic symptoms soon develop, the temperature suddenly bounding to 103, to 104 or 105 degrees F., with albumen and casts. The ordinary duration of an attack of delirium tremens is about two weeks in those recovering, although death may occur at any time from cardiac failure, uraemia, or alcoholic pneumonia. Convalescence dates from the beginning of refreshing sleep, the patient awaking with a clear mind and desire for food. Should the delirium subside, but the patient continue to mutter and pick at the bedclothing, the tongue become dry and cracked, and the regurgitation of dark brownish and bilious matter occur, the condition is critical and an early fatal termination may be expected.

Dipsomania or Oinomania is the inherited or acquired mental condition which craves the drinking of intoxicating liquors. This is a true mental disease. It manifests itself in periodical attacks of excessive indulgence in alcoholic drinking, or this symptom of this sad disease may be replaced by other irresistible desires of an impulsive kind, such as lead to the commission and repetition of various crimes, the gratification of other depraved appetites, robbery, or even homicide. Imbecility and dementia frequently result.

The paroxysms at first occur at long intervals, but gradually the intervals become shorter and shorter until the individual entirely surrenders himself to alcoholic and other excesses.

Diagnosis. Profound drunkenness or alcoholic coma may and often is confounded with apoplectic and uraemic coma. Von Wedekind suggests the following method for diagnosing drunkenness: "By simply pressing on the supraorbital notches with a steadily increasing force you may, with certainty of success, bring an unconscious alcoholic to his senses, and thus differentiate between alcoholic and other comas." The symptoms of chronic

alcoholism often bear a close resemblance to the following maladies: general paralysis, disseminated sclerosis, paralysis agitans, locomotor ataxia, cerebral and spinal softening, epilepsy, dementia chronica, and nervous dyspepsia.

In individuals whose habits are secret the question of diagnosis is attended with considerable difficulty. Anstie lays much stress upon the importance of the following four points, diagnostic of chronic alcoholism: insomnia, morning vomiting, muscular tremor, and causeless mental restlessness.

PROGNOSIS. In acute alcoholism the prognosis is good if the patient is manageable. In chronic alcoholism the organic changes, the direct result of the alcoholic habit, tend to shorten life by the production of fatty heart, Bright's disease, insanity, impotence, epilepsy, melancholia, and organic brain diseases. The danger in delirium tremens is heart failure or deepening coma. The association of chronic nephritis with delirium tremens, perhaps its cause, must always be taken into account in determining a prognosis. Acute lobar pneumonia is a very fatal complication of all forms of alcoholism.

THE TREATMENT.

The treatment for confirmed alcoholism is necessarily a variety treatment, to meet varied indications. Like for the poisonous effects of opium, the patient should be moved around continuously, or the manipulations made with a view to keep up a rapid and uninterrupted circulation of the blood. Strong divulsion of the sphincters sobers the victim at once, and the operator will immortalize himself in the estimation of some to do so, but there is either an inborn craving, or a cultivated one, that has a deeper significance than occasional habit of inebriety. Habit, like an armed foe, has transformed every tissue into servitude to the destroyer, and the will power of the victim must be turned in the opposite direction to make him a sober man. The diseased morbidity of his stomach should be intelligently looked after, rested, treated through the cerebro-spinal nervous system until all right. Non-stimulating diet and no breakfast enjoined. Complete hypnotism and suggestion cures a larger percentage than anything else. Use it. To sober up a person drunken, press on the supraorbital nerves.

HEAT STROKE.

SYNONYMS. Insolation; sunstroke; thermic fever; coup-de-soliel; heat exhaustion.

DEFINITION. A depression of the vital powers; the result of exposure to excessive heat. The condition manifests itself as acute meningitis (rare), heat exhaustion (common), and as true sunstroke.

CAUSES. Exposure to the influence of excessive heat, either to the direct rays of the sun or artificial heat in confined quarters, or diffused atmospheric heat without proper ventilation. Among the predisposing causes, which act by lessening the power of the system to resist the heat. are great bodily fatigue, overcrowding, and intemperance.

PATHOLOGICAL ANATOMY. The action of the heat upon the system is so sudden, and the malady so rapid in its course, that structural changes have not developed. The left ventricle is firmly contracted (Wood). The right heart and vessels are gorged with dark fluid blood. All the tissues and organs of the body are in a state of great venous congestion. The blood is dark, thin, and either but feebly alkaline or decidedly acid, and its power of coagulability is destroyed. The post-mortem rigidity is early and marked.

SYMPTOMS. Depending upon the variety.

Acute meningitis the result of exposure to heat is similar to that due to other causes.

Heat exhaustion develops with a rapid feeling of weakness and prostration, the surface cool, the face pale, the voice weak, the pulse rapid and feeble, the respiration increased, the vision growing dim and indistinct, noises develop in the ears, the individual overcome, becoming partially, or completely unconscious. with perhaps convulsions and tremors, and shrunken features.

Sunstroke.—The symptoms, developing suddenly, with or without prodromata, are, insensibility, with or without delirium, or convulsions, or paralysis, the surface flushed and hot, the conjunctivae injected, the breathing either rapid and shallow or labored and stertorous, the pulse quick and either bounding or weak, and the temperature in the axilla ranging from 105 degrees to 108 degrees, to 110 degrees, with suppression of all glandular action. Death occurring, the result of asphyxia, or from a slow failure of respiration and cardiac action.

DIAGNOSIS. It is of great importance, therapeutically, to distinguish at once between attacks of sunstroke and heat exhaus-

tion. Cases of sunstroke are to be differentiated from cerebral hemorrhage and alcoholic insensibility, for which purpose the clinical thermometer is indispensable.

PROGNOSIS. Attacks of heat exhaustion, if properly and promptly treated, favorable. The prognosis of sunstroke or heat fever is unfavorable in the majority of cases, death resulting in from half an hour to several hours. Unfavorable indications are, increased temperature, cardiac failure, convulsions, absent reflexes, followed by complete muscular relaxations. Favorable indications are, decline in surface heat and axillary or rectal temperature, stronger pulse, increased depth of respirations, restored reflexes, and return of consciousness.

THE TREATMENT.

The indications are apparent: Restore capillary circulation as soon as possible. The bivalve should be brought into requisition if possible, otherwise use the digits to divulse; then regulate the action of the circulation by vaso-motor stimulation, then follow up the treatment of spine, strongly extending the arms as the spine is treated. Use all the measures to establish and continue circulation, especially in the body. To draw blood from the head use the hemaspasia process—cording one of the lower limbs—and hold the venous blood there for half an hour, then remove the cord, and place it on other limb, same way and time. Induce vomiting of contents of stomach, if full. Use strong, rapid vibratory movements along the spine and over the abdomen, moving the muscular tissue profoundly for several moments. The neck muscles should be thoroughly manipulated, and the clavicles raised, so as to permit the venous blood to return from the brain. These measures will be found successful, except caused by alcoholism—and sometimes then. Remove all obstacles to deep inspiration and be sure to place the patient in airy quarters. Oxygen is essential in such cases. Enjoin deep breathing, artificial if necessary, until recuperation.

ACUTE HYDROCEPHALUS.

SYNONYMS. Acquired hydrocephalus; serous apoplexy.

DEFINITION. Strictly speaking, hydrocephalus signifies water in the brain; but it is here restricted to the presence of a serous fluid in the arachnoid spaces, in the pia mater, in the ventricles, and in the brain substance (œdema); characterized by the

more or less sudden development of cerebral excitation, followed by depression and usually death.

CAUSES. Most common between the ages of one and five, although it may occur at any age. "'The predominance of the nervous system in the bodily conformation" is a strong predisposing cause. Among the exciting causes are unfavorable hygienic conditions, dentition, eruptive fevers, blows on the head, mechanical causes preventing the return of the blood from the venae Galeni and the right sinus, compression of the jugular vein, diseases of the right heart, and Bright's disease.

PATHOLOGICAL ANATOMY. The effusion may be limited to the ventricles, although there is usually considerable distention of the subarachnoid spaces and œdema of the pia mater and neighboring portions of the brain, whence results more or less softening, especially around the ventricles. The choroid plexus is hyperaemic, and may be the seat of minute extravasations.

SYMPTOMS. There are three varieties of acute hydrocephalus with characteristic symptoms, to-wit: comatose, convulsive, and the ordinary.

Comatose.—Known also as "serous apoplexy," begins abruptly with the phenomena of apoplexy, the result of the sudden effusion. The pressure is usually so great on the medulla oblongata that it ceases to functionate, death resulting in a few hours, rarely lasting several days.

Convulsive.—The result of Bright's disease or a general dropsy, is ushered in with headache, nausea, and vomiting, followed in a day or two with convulsions, passing into coma, which usually terminates fatally, although rarely a remission may precede death for a day or two.

Ordinary.—The most common in children, begins with feverishness, headache, vertigo, photophobia, restlessness, nocturnal delirium, insomnia, twitching, and spasmodic contractions of the muscles and great hyperaesthesia of the skin. Such symptoms continue for several days, when convulsions occur, followed by death, or a continuance of the symptoms, followed by rigidity, stupor, and death.

THE TREATMENT.

In referring to causes, it will be seen that the nervous system is interfered with, or a predominance is in that direction. This will generally be found to be reflexly. Something will be found wrong with the genitals. Referring to the Orificial Surgery Journal, I find that cases have been successfully treated by the

performance of circumcision. That relieves the reflex irritation from that source, especially where phymosis exists. This should be corrected in cases demanded, and no one is excusable for neglect in this regard. The neck muscles are most generally involved also, and contracture of the muscular fibers closes the clavicles down, preventing venous return circulation, hence accumulation in the brain. Clear out the scavengers (the veins), then look for favorable results—not till then. The mild and persistent general treatment every day or two will be rewarded by favorable results in a larger per cent. of cases generally, than from other treatment. The spinal system of nerves is to be kept in normal condition, the renal splanchnic receiving special attention each treatment. The indications properly heeded, promise good results if treatment is properly applied. These directions apply to the chronic congenital as well. Treatment should be applied every other day, at least.

CONGENITAL HYDROCEPHALUS.

DEFINITION. An excessive accumulation of the cerebro-spinal fluid—a cerebral dropsy—in the ventricles—internal hydrocephalus; or in the meshes of the pia mater—external hydrocephalus, or in both—mixed hydrocephalus; characterized by enlargement of the head and more or less pronounced nervous phenomena. A disease of infants, or very young children.

CAUSES. Imperfect or arrested development of the brain or its membranes. Occurs in the offspring of tubercular, scrofulous, or syphilitic parents. Inflammatory changes in the ventricles and ependyma.

PATHOLOGICAL ANATOMY. Enlargement of the head is the chief external pathological condition, although there is no constant ratio between the size of the head and the amount of fluid, the quantity varying from an ounce to a pint or more. The liquid is transparent, of a straw color, containing a small amount of albumen and chloride of sodium. If the quantity of fluid be small the ventricles are simply distended, if the amount be large the optic thalami and corpus striatum are depressed and flattened, the roof of the ventricles thinned, and the foramen of Monro is greatly enlarged. The enlargement of the head may occur before birth and impede or prevent natural delivery, or the head may be normal at birth and increase afterward. As enlargement

progresses the bones are so thinned as to be translucent, the fontanelles and sutures are widened, the lateral portions of the cranium project, the forehead bulges out over the eyes, and the orbital plates are depressed, forcing the eyes outward and downward, producing a variety of exophthalmos; the head has an irregular, triangular shape, the base of the triangle being the top of the head. The scalp being stretched by the pressure within, becomes tense and thin, and but scantily covered with hair, the veins which ramify in it are unusually prominent and large, and the entire head is elastic on pressure, from the amount of liquid beneath.

Hilton, in "Rest and Pain," says: "In almost every case of internal hydrocephalus which I have examined after death I found that this cerebro-spinal opening (between the fourth ventricle and the spinal canal) was so completely closed that no cerebro-spinal fluid could escape from the interior of the brain; and, as the fluid was being constantly secreted, it necessarily accumulated there, and to my mind the occlusion formed the essential pathological element of internal hydrocephalus."

SYMPTOMS. The increased size of the head, with the emaciated condition of the child, who seemingly eats well, is what first attracts the attention. The head appears too heavy, the eyes are prominent and have a downward direction, the face is devoid of expression, old and wrinkled, the voice feeble; the mental development is not in keeping with the age. When the period for standing or walking arrives the power is found wanting. The further history is but a continuation and exaggeration of this, until convulsions occur, which sooner or later terminate fatally.

The course of congenital hydrocephalus is usually slow, but progressively worse. The majority terminate within the first year; cases are recorded, however, of ten and fifteen years' duration.

DIAGNOSIS. In rachitis the volume of the head is increased, due, in part at least, to a deposit of calcareous matter on the exterior of the cranial bones. Rachitis may be mistaken for hydrocephalus in cases in which the amount of liquid is small. The differential diagnosis is based on the shape of the head, round in rachitis, square or triangular or with prominences in hydrocephalus; with the persistent downward direction of the eyes and the elasticity of the head on pressure.

PROGNOSIS. Unfavorable. Arrest of progress and even cures have been reported. Spontaneous cures are reported fol-

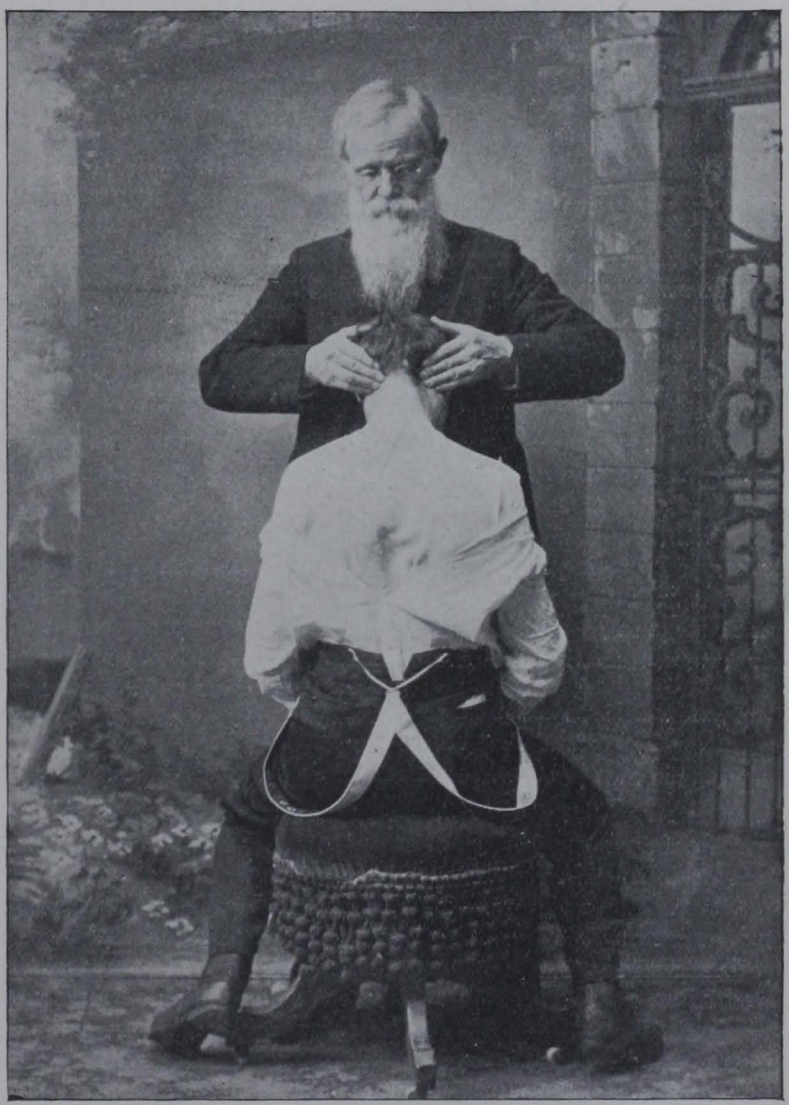


PLATE XLIX.—Vibratory Movements, Neck and Back.

lowing the accidental discharge of the fluid. But such reports are exceptional.

TREATMENT. See treatment for Acute Hydrocephalus.

DISEASES OF THE SPINAL CORD.

SPINAL HYPERÆMIA.

SYNONYMS. Spinal congestion; plethora spinalis.

DEFINITION. An abnormal fullness of the vessels of the meninges and cord; active when an arterial hyperaemia; passive when a venous hyperaemia; characterized by pain in the back, with more or less pronounced disorders of sensation and locomotion.

CAUSES. Cold and exposure; arrested menses; arrest of habitual hemorrhoidal discharge; malaria; protracted erect posture; injuries to the back; certain spinal poisons, as strychnina, picrotoxinum, and alcoholic excesses.

PATHOLOGICAL ANATOMY. Active.—The post-mortem appearances are congestion of the meninges and cord, the same vessels supplying both, with numerous points of extravasation, due to the rupture of capillary vessels. The spinal fluid is increased in amount. Passive.—A general bluish discoloration, owing to the abnormal fullness of the large anastomising vessels; the spinal fluid somewhat increased.

SYMPTOMS. Active.—Dull pain in the dorsal or lumbar region, shooting into the hips and thighs, persistent and increased by pressure; tenderness on motion; tingling sensations in the limbs and feet, and sometimes in the hands and arms; a feeling of constriction about the abdomen is often present, with rigidity of the abdominal muscles. Increased reflexes, with disorders of motility, and when the patient is in the recumbent position, jerking of the limbs. On attempting to walk it is accomplished with difficulty, from an incomplete loss of power. If the upper part of the cord be affected, dyspnœa and palpitation occur. There often occur painful priapism and frequent nocturnal emissions.

The above symptoms may be followed by a more or less

pronounced temporary depression, the sensation diminished, and the lower limbs benumbed and heavy, the movements weak.

The electro-contractility is preserved, and in many cases even increased or exaggerated.

DURATION. From a few hours to several days; if longer, myelitis may result.

DIAGNOSIS Anaemia causes more or less spinal irritability and tenderness; but the history, pallor, and general weakness, unassociated with defects of motility or sensibility, will prevent error. Spinal meningeal hemorrhage is more sudden in its onset, its violence, and its range of symptoms. Myelitis and spinal meningitis have symptoms in common with spinal congestion, which will be pointed out when discussing those affections.

PROGNOSIS. Favorable, recovery occurring in three or four days. If the symptoms show a tendency to linger, myelitis, more or less pronounced, will ensue.

TREATMENT. See Spinal Meningitis.

PACHYMENINGITIS SPINALIS.

SYNONYMS. Pachymeningitis spinalis interna; hypertrophic pachymeningitis; pseudo-membranous pachymeningitis.

DEFINITION. An inflammation of the inner surface of the spinal dura mater; characterized by violent pains in the head, back, shoulder, and arms, followed by contractures and paralyses of the upper extremities.

CAUSES. Exposure to cold and damp; alcoholism; syphilis; gout; injuries.

PATHOLOGICAL ANATOMY. Hypertrophic pachymeningitis is characterized by an exudation upon the inner surface of the dura mater, which gradually solidifies into a layer of compact connective tissue, which presses upon the spinal cord and nerves, producing a myelitis and an atrophic neuritis, resulting in muscular atrophy. The most frequent seat of this form is the cervical hypertrophic pachymeningitis.

In the pseudo-membranous form a membranous exudation also occurs, in which large numbers of blood vessels develop and rupture, the hemorrhagic extravasation forming a cyst—haematoma—which causes pressure on the cord and nerves.

SYMPTOMS. The onset is slow and gradual, with irregular chills and feverishness, violent pains, and stiffness in the head, neck, shoulders, and arms, continuous but subject to exacerba-

tions, and associated with a painful constriction of the upper thorax. Numbness and prickling occur in the arms, more marked in one than the other. Rarely nausea and vomiting occur. These symptoms may continue off and on for several months, the muscles of the painful parts atrophying, followed by spasmodic contraction, particularly of the hands and wrists, followed later by paralysis.

The paralytic stage develops gradually, with weakness in the arms, associated with contractures and rigidity. The pain continues with anaesthesia, hyperaesthesia, and trophic changes. Later paraplegia with rigidity, exaggerated reflexes, and spinal epilepsy develop.

The development of tuberculosis and nephritis during the progress of chronic cerebral and spinal diseases, which are the immediate cause of death, is a clinical observation.

The electro-contractility is lost.

PROGNOSIS. If early recognized and promptly treated, the hypertrophic form may be improved. Generally, however, the prognosis is unfavorable.

TREATMENT. See Spinal Meningitis.

SPINAL MENINGITIS.

SYNONYM. Leptomeningitis spinalis.

DEFINITION. Inflammation of the arachnoid and pia mater membranes of the spinal cord, either acute, subacute, or chronic; characterized by pain in the back, rigidity of the muscles, disorders of motility and sensibility. It may be acute or chronic.

CAUSES. The disease is rare, and is always due to an infection from tubercle, syphilis, typhoid fever, or septicaemia, or the result of a traumatism.

PATHOLOGICAL ANATOMY. Acute.—Hyperaemia of the membranes, with swelling of the tissues, the result of serous infiltration, followed by purulent and fibrinous exudations. The roots of the spinal nerves are covered with exudation, and are swollen and soft. The cord proper is more or less congested and œdematous.

Chronic.—Adhesion of the membranes, with more or less accumulation of fluid, resulting in atrophic degeneration of the cord from pressure. If the disease is secondary to tubercle, these

granulations are seen distributed over the pia, arachnoid, and inner surface of the dura.

SYMPTOMS. There are two stages, the first, the stage of irritation, the second, the stage of paralysis of motion and sensation, with atrophy. Although an inflammatory affection, yet its onset is usually subacute, the febrile reaction being moderate, with intense boring pain in the back, aggravated by motion, rigidity of the spine, and a sense of constriction around the body —"the girdle." Spasmodic contractions of the muscles innervated by the nerves originating at the seat of the lesion, with inability to straighten the limbs. If the lower part of the spinal membranes is the seat, there occur constipation and retention of urine; if upper part, dysphagia, dyspnœa, and feeble heart. The muscular contractions are excited or increased by motion, but uninfluenced by pressure. Reflex movements are not abolished. The rigidity and spasmodic contraction of the muscles are followed by paralysis, more or less complete, death following from paralysis of the muscles of respiration. If the inflammation extend to the medulla, the above symptoms are associated with disorders of speech, vomiting, and delirium.

Electro-contractility lessened or absent, both as to motility and sensibility, in the affected parts.

Chronic form succeeds to the acute or originates spontaneously, and presents the same form and order of symptoms—excitation or irritation, and depression or paralysis.

DIAGNOSIS. The points of importance are deep, boring pain in the back, aggravated by motion but not by pressure, with spasmodic contraction of the muscles, followed by paralysis.

Myelitis slight or absence of pain with earlier and more complete paralysis.

Tetanus may be confounded with spinal meningitis. The points of distinction are: in the former occur early trismus with rhythmical spasms excited by irritation of the skin, whereas irritation of the skin does not in spinal meningitis produce muscular contractions, but movement of the limbs does do so; progressively increasing, and not associated with fever; usually a clear history of an injury.

PROGNOSIS. Generally unfavorable. Death is either sudden, from paralysis of respiration and of the heart, or gradual, the result of exhaustion. Critical discharges, such as profuse perspiration, urinary flow, or epistaxis occur, and are followed

by rapid recovery. Cases recovering may have more or less pronounced partial or complete paralysis.

THE TREATMENT.

The treatment for spinal hyperaemia, pachymeningitis and spinal meningitis is modified according to circumstances, and applies to all. The important thing to be done is to take off the pressure from the veins. To do this, results in a removal of the difficulty—venous closure. The whole man demands our attention, hence general treatment, using the various manipulations recommended to restore normal circulation, by establishing capillary circulation, and removal of pressure from the terminal nerve filaments. The first attention is to be directed to the vaso-motor nervous system, and then the spine, all the way down. The neck should be duly stretched, the body extended, the spine stimulated, chest expanded, intercostals relieved of venous congestion, lower limbs flexed, rotated, extended, and every manipulation applied that is essential to the promotion of freedom of the circulation of the fluids in veins and lymphatics. This takes off the pressure from the nerves, reaction ensues at once, the difficulty is arrested, and health restored. Treatments should be repeated daily or oftener, or at longer intervals, according to indications. Promoting a free circulation all over the body is the thing to do. Use the means indicated.

ACUTE MYELITIS.

SYNONYMS. Acute or general diffuse myelitis; transverse myelitis; softening of the cord.

DEFINITION. An inflammation affecting the substance of the spinal cord, which may be limited to the gray or white matter, and involve the whole or isolated portions of the cord. When the gray matter alone is inflamed, it is termed central myelitis; when the white matter and the meninges, it is termed cortical myelitis; it may be ascending, descending, or transverse in its extension. The disease is characterized by more or less sudden and complete loss of motion and sensation.

CAUSES. Following spinal meningitis; exposure to cold and damp; injuries to the vertebrae; prolonged functional activity of the cord; typhus fever; rheumatism; syphilis; puerperal fever, or,

during the course of the exanthemata, arsenical or mercurial poisoning.

PATHOLOGICAL ANATOMY. Intense hyperaemia of the substance of the cord, with extravasations, giving the tissues a reddish-brown or chocolate tint, and also serous transudations, resulting in softening of the structure of the cord, the color changing to yellow and white, the nerve elements undergoing fatty degeneration, presenting the appearance and consistency of cream. The membranes also undergo more or less change.

SYMPTOMS. The severity of the symptoms depends upon the extent and location of the inflammation. The onset is usually sudden, with a chill, fever, 103 degrees, frequent pulse, with alterations in sensibility and motility, to-wit: pain in the back, aggravated by touch and by heat and cold, with sensations of formication ("pins and needles"), the limb feeling as if asleep, or else complete anaesthesia, associated with severe neuralgic pains.

The distinction between anaesthesia (insensibility to touch), and analgesia (insensibility to pain) must be clearly determined.

A sensation of constriction around the body and limbs, as if encircled by a tight cord, "the girdle pains"; rapidly developing paraplegia, complete in a few hours, with involuntary discharges. The reflex functions are usually abolished, as seen by attempting to cause movement of the limbs by tickling the feet or by striking the patella tendon; rarely are they diminished, very rarely exaggerated. The temperature of the affected limbs is lowered three or four degrees. Sloughs and bed-sores and muscular atrophy result if the anterior cornua—the trophic centers—are affected.

The above symptoms of loss of motion and sensibility with rectal and vesical paralysis, are associated with more or less pronounced vomiting, hepatic disorders, irregularity of the heart, dyspnœa, dysphagia, apnœa, and painful priapisms. The urine is markedly alkaline in reaction, finally developing cystitis. Among the late manifestations are shooting pains and spasmodic twitching or contractions of one or all of the muscles of the paralyzed parts.

The electro-contractility is abolished in the paralyzed parts.

DIAGNOSIS. Acute spinal meningitis is distinguished from acute myelitis by severe pains, increased by pressure, with muscular contractions increased by motion, followed by paralysis much less profound than the paraplegia of myelitis; in spinal

meningitis there exists cutaneous and muscular hyperaesthesia, which is absent in myelitis.

Congestion of the spinal cord is characterized by the mild character and short duration of all the symptoms.

Hemorrhage in the spinal canal is abrupt with irritative symptoms, slight paralysis, preserved reflexes and electro-contractility.

The principal diagnostic points of acute myelitis are the "girdle" around the limbs or body, rapid and complete paraplegia, loss of sensation, lowered temperature in the affected parts, early and persistent sloughing (bed-sores), and alkaline urine or cystitis.

Hysterical paraplegia shows no trophic changes, no altered reflexes, slight atrophy, irregular anaesthesia, and the presence of the stigmata of hysteria.

Lithaemic paraesthesia, tingling and numbness of fingers and toes, might lead to error if the cerebral symptoms of lithaemia are overlooked.

The diagnosis of the location of the lesion is made by a study of the height of the anaesthesia, the skin reflexes, and the distribution and extent of the paralysis.

PROGNOSIS. Varies according to the location of the lesion and completeness of the symptoms.

If the paralysis is of the ascending variety, death occurs within a few days, from paralysis of the muscles of respiration.

If the trophic centers are affected, there occur bed-sores, intense pyelo-nephritis and cystitis and changes in the joints; death from exhaustion in several weeks.

Central myelitis, or inflammation of the gray matter, is rapid in its progress, death occurring in a week or two.

The morbid process may be arrested and the general health restored, but some spinal symptoms will persist.

THE TREATMENT.

When the effects are considered, the cause is easily imagined —venous congestion. This congestion is due to the contraction of the muscular fibers, drawing fascia, perhaps capsular ligamentous tissue, taut across the nerves leading to the spinal cord, lessening or abridging or destroying nervous communication to the cord; hence paralysis of the connective tissue around and of the walls of the veins, hence extravasation of blood, hence pressure. The very nature of the results demonstrates this fact.

The results of the application of Osteopathic principles prove the correctness of this assertion. The extensiveness of the lesion at the various sections of the spinal ganglia is seen in effects on certain muscles supplied by the various nerves from it—supplying said muscles. The knowledge of the origin, distribution, and places of exit from the spinous foramina becomes a matter of much moment in the correct application of the principles of this science, manipulatory at least. It will be a stunner to the general practitioner to contemplate the probabilities of a possible benefit through the application of these principles to such a terrible state of affairs as we find in such cases! The applications prove alike successful in all the forms of paralysis known to exist. The more recent, the more potent for relief. We would state here: That venous obstruction relieved, myelitis ceases. The treatment given by the ordinary medical man sustains about as much relationship to cause and effect as tying a vein to stop arterial hemorrhage. It will invariably be found that failure to return the blood from the head through the internal jugular veins is the direct cause of the congestion that produces the pressure that results in the lesion—the extravasation—pressing upon the origin of nerves that supply certain muscles. Remember that.

The treatment must be directed to the place of obstruction —in the neck veins first. Then the pressure must be removed all the way along the lines of the various nerves from origin to termination. This takes off the pressure, lifts the weight from the venous walls, permits return circulation to the heart, lungs, where oxygenation takes place, renewal of the vital forces and a normal arterial supply. The treatment, then, should begin at the occiput. It should be continued all the way down the spinal column, every manipulation recommended in general treatment for the whole system used, and special regard to the proper inhalation of pure air, as well as due regard to the restfulness of the digestive tract, avoiding overcrowding the stomach. Rest is especially enjoined until amelioration ensues. Treatments applied systematically and intelligently at intervals of from a few hours to two days will be rewarded, usually, with perfect success.

INFANTILE SPINAL PARALYSIS.

Synonyms. Myelitis of the anterior horns; poliomyelitis anterior acuta; essential paralysis of children; atrophic paralysis of children.

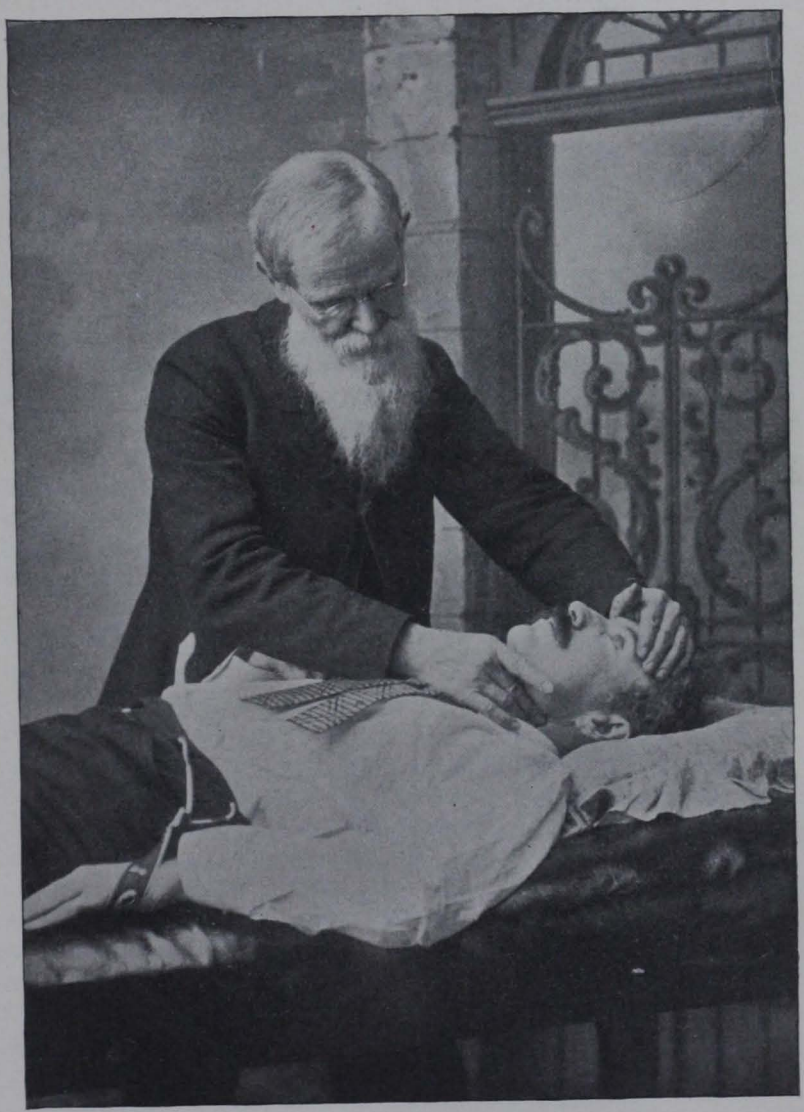


PLATE L.—Showing Manipulations of Neck and Throat.

DEFINITION. A rapidly developed inflammation of the anterior horns of the gray matter of the cord, occurring suddenly in children, at times in adults—acute spinal paralysis of adults—characterized by mild fever, muscular tremors and twitchings, and paralysis of groups of muscles.

CAUSES. Essentially a disease of early life—the second month to the third or fourth year. The fact of its having occurred in adults must be borne in mind. Cold and damp; dentition (?); injuries to the spine; developed during convalescence from the acute exanthemata.

PATHOLOGICAL ANATOMY. The early changes are: medullary hyperaemia, vascular exudation and inflammatory softening, although the naked eye may not recognize any changes. Microscopical examination reveals inflammatory softening of the anterior horns of the gray matter. Among other constant lesions are atrophic degeneration of the multipolar ganglion cells and of the anterior nerve roots.

The changes noted as occurring in the cord are usually limited to the dorso-lumbar and cervical enlargements. As a direct result of the changes in the trophic centers and the nerve degeneration of the muscular fibers supplied, there ensue changes in the bones and joints, leading to great deformities.

SYMPTOMS. The onset of the affection varies; it may be acute, subacute, or chronic; it is usually sudden, with an attack of mild fever of a remittent type, of a few days' duration, on recovery from which it is noticed that the child is paralyzed. Rarely the paralysis may be preceded by convulsions. The paralysis may affect both arms and both legs, the legs alone, or only one of the four extremities; it may, but very rarely, be a hemiplegia. As a rule, however, the leg suffers more frequently than the arm; in paralysis of the leg the muscles below the knee suffer more severely than those above. The bladder and rectum are not affected, or if so, only temporarily, nor can anaesthesia or numbness be detected. The temperature of the paralyzed limb is low and the appearance cyanosed. After a few days there is a slight improvement in the paralyzed parts, although the muscles show a rapid wasting, which is progressive until all muscular tissue is gone.

The reflex movements are impaired or abolished.

The electro-contractility by the faradic current is abolished in the paralyzed parts. With the galvanic or constant current the "reactions of degeneration" are developed. To fully under-

stand the meaning of this term a knowledge of the normal electrical reactions is necessary.

The normal formulae for the production of muscular contraction in the physiological state are as follows, the strength of the current being barely capable of causing fair contractions: First.—The most effective contractions are produced by the cathode (negative) pole on closing the circuit. Second.—The second most effective are produced by the anode (positive) pole on closing the circuit. Third.—The next most effective is by the anode pole on opening the circuit. Fourth.—Cathode pole contractions on opening circuit are rarely seen in the physiological state.

The "reactions of degeneration" are shown by any reversal of the regular formulae, to-wit: if the anodal closure shows stronger contractions than cathodal closure; still greater degeneration is shown if the anodal opening contractions are stronger than either of the above; and most complete degeneration is shown by the complete reversal of the normal formulae as shown by distinct cathodal opening contractions.

SEQUELÆ. Amongst the deformities resulting from the paralysis are the different forms of talipes. Talipes Equinas.—The result of paralysis of the antero-external muscular group of the leg. Equino-Varus.—The result of paralysis of the antero-external muscular group of the leg, together with the adductors of the foot. Talipes Calcaneus.—The result of paralysis of the muscles of the calf of the leg. Talipes Cavus.—"Pes cavus"—characterized by the hollowing of the sole of the foot, with prominence of the instep, the result of paralysis of the calf muscles with contraction of the long flexor of the toe or the long peroneus—the foot flexors.

DIAGNOSIS. The recognition of acute poliomyelitis is not always possible at the onset or during its early days, as localized paralyses are difficult of detection in children, but immobility of one leg or arm in children with febrile symptoms or following convulsions is always an indication of poliomyelitis. After the initial stage has passed, the presence of paralysis, wasting, presence of R. D. (reactions of degeneration), loss of reflexes, and the absence of anaesthesia, render the diagnosis very easy.

Hemiplegia from acute cerebral affections in children can be distinguished from infantile paralysis by the disorders of intelligence and the special senses, and the perseverance of the normal electro-contractility.

Paralysis of myelitis occurs in older persons, and is associated with disturbances of the genito-urinary organs and bed-sores.

Pseudo-muscular hypertrophy, with paralysis, begins gradually, becoming progressively worse with increase in the size of the limbs.

PROGNOSIS. More or less paralysis with muscular wasting always results, although there is no doubt that the extent can be greatly lessened by early recognition and treatment.

THE TREATMENT.

The same general principles apply here as in every case of paralysis. Take off and keep off the pressure. Restore normal blood supply and recovery ensues. There are more cases cured by the application of these principles than by any other means ever devised. The freedom of the flow of all of the fluids to and from the heart, means much—it means health. Follow the special indications as well as general. It requires time and patience in the chronic form of these affections. Persist with confident assurance of final relief. Marvelous consequences follow these treatments.

CHRONIC PROGRESSIVE BULBAR PARALYSIS.

SYNONYMS. Glosso-labio-laryngeal paralysis; bulbar paralysis.

DEFINITION. A chronic degenerative affection of certain nuclei of the medulla oblongata; characterized by a slowly progressive bilateral paralysis of the tongue, lips, palate, pharynx, and larynx, with atrophy of the tongue and lips.

CAUSES. Obscure. Rare before the fortieth year. Among many others may be named cold, rheumatism, gout, syphilis, and injuries about the neck.

PATHOLOGICAL ANATOMY. "Degenerative atrophy of the gray nuclei in the floor of the fourth ventricle; with atrophy and gray discoloration of the nerve roots from the medulla, especially of the facial and hypoglossal nerves." "Atrophy and disappearance of the motor ganglion cells is always to be noted. It may be the sole lesion."

"The nerves going to the muscles exhibit sclerosis of the neurilemma, and the degenerative atrophy is found in the nerve roots coming from the bulb."

SYMPTOMS. The disease begins insidiously. There is first noticed some difficulty in articulation, from want of precision in movements of the tongue, particularly in the use of the lingual consonants, *l*, *n*, *r*, and *t*, which increases until that organ is completely paralyzed. The paralysis gradually invades the soft palate and pharyngeal muscles, causing difficulty in deglutition, of the orbicularis oris, preventing closure of the lips, of the laryngeal muscles interfering with articulation. With the increasing loss of power in the tongue and lips is also a gradual atrophy of these muscles. When the disease is fully developed the condition of the patient is most pitiable indeed; articulation is impaired or impossible, deglutition interfered with, the lips remaining apart allowing the saliva to dribble from the mouth, and liquids to return through the nose if attempts are made to swallow them. As the malady progresses, the pneumogastric nucleus becomes involved, resulting in loss of voice, difficulty of respiration, and cardiac irregularity. The general health gradually suffers from insufficient nutrition and imperfect respiration, although the mind is clear until the end. The "reactions of degeneration" are present.

Besides the chronic bulbar paralysis, there are two acute forms which give the same symptoms as the chronic cases, only they develop suddenly, one, the result of hemorrhage into the medulla, which at the onset has vertigo, vomiting, loss of power in the limbs, and slight sensory disturbances, all of which disappear, leaving the glosso-labio-laryngeal paralysis; the second form comes suddenly, with fever, vomiting, and loss of power in the limbs, soon disappearing, leaving the characteristic bulbar symptoms; this variety is inflammatory and closely allied to acute poliomyelitis.

DIAGNOSIS. It can hardly be confounded with any other malady.

PROGNOSIS. Unfavorable. The duration is from one to five years.

THE TREATMENT.

This, like many other affections of the nervous system, is traceable to muscular contracture in the neck. Long-continued contractions of the fibers of the trapezius, splenius capitis, the levator anguli scapulae, and of the scaleni, mitigate the action of the terminal sympathetic nerve influence, cut off communication with the motor, and eventually produce paralysis. These contractures interfere also (and have perhaps for years) with the

venous circulation, squeezing down on the recurrent nerve filaments, as well as the spinal accessory and the glosso-pharyngeal nerves; hence paralysis of the tongue. That the other muscles (those in front of the neck) are implicated there is no doubt, when we consider that the venous blood is prevented from returning through the internal jugulars thereby, and thus press upon the base and posterior part of the brain. The release from contracture of all of the muscles of the neck is essential to the cure of the condition under consideration. The pneumogastric and the facial nerves need attention in that they affect respiration and the muscles of the face. The extension of the neck should be carefully done, the rotary movement as well; then all the muscles profoundly manipulated and vibrated on all sides of the neck, reaching high up under the chin and angles of the jaws, in front of the ears, on the cheeks, the sides of the nose, divulsing the nares as well. Continue treatments twenty or thirty minutes, and as often as every other day. General treatment.

PROGRESSIVE MUSCULAR ATROPHY.

SYNONYMS. Wasting palsy; chronic spinal muscular atrophy; chronic poliomyelitis; amyotrophic lateral sclerosis.

DEFINITION. A slow, gradual, progressive wasting and atrophy of certain groups of muscles, with symptoms varying in accordance with the variations in the pathological anatomy.

CAUSES. Most frequent in males between twenty-five and fifty years of age, and in many instances is hereditary. A predisposing cause seems to exist in those who habitually use one set of muscles (muscular strain). Exposure to cold and damp; lead; syphilis; injuries to the spinal column. Following such acute diseases as diphtheria, measles, acute rheumatism, typhoid and typhus fevers.

PATHOLOGICAL ANATOMY. Two theories as to the origin of the pathological changes are held: one that the initial lesion is in the cord (Charcot), the other in the muscular interstitial connective tissue (Friedreich).

The morbid alterations are of two groups—spinal and muscular. The spinal changes consist in the atrophy and degeneration of the anterior columns, wasting and disappearance of the multipolar ganglion cells of the anterior horns, with hyperplasia of the neuroglia; rarely the hyperplasia extends to the lateral

columns (amyotrophic lateral sclerosis); also wasting, atrophy, and degeneration of the anterior nerve roots. The muscular changes consist of a progressive wasting of the muscular tissue, with increase of the interstitial connective tissue. "The final result is, that the muscle is converted into a mere fibrous band with numerous fat-cells, the development of this latter material taking place outside of the muscular elements and in the newly-formed connective tissue." (Bartholow.)

SYMPTOMS. The invasion is gradual, the disease having been in progress some weeks or months before the patient is aware of its existence. Wasting begins usually in the hand, the first dorsal interosseus being the first to be attacked, then the muscles of the thenar and hypothenar eminence, then the deltoid, and so on from muscular group to group. Often, however, the extension is very erratic in its course, jumping from one group to another at some distance.

In the immense majority of cases the disease is permanently limited to one or a few groups of muscles in the upper, or more rarely, in the lower extremities. The only muscles not yet known to be attacked are those of mastication and those that move the eye-ball (Roberts.)

Fibrillary contraction is an early symptom, continuing more or less marked so long as any muscular fibers remain. It consists of wave-like movements of the muscles, excited automatically, by draughts of air or percussion. Coincident with the wasting are loss of power, disorders of sensation, coolness, and pallor of the surface.

The natural roundness and contour of the body and limbs are changed, the bones standing out in unaccustomed distinctness, giving the individual the appearance of a skeleton clothed in skin. The hand is frequently the seat of a very singular deformity—the "claw-shaped" hand.

The electro-contractility is preserved so long as muscular fibers remain.

DIAGNOSIS. When wasting palsy is fully developed its diagnosis is a simple matter. In its early stages a doubt may exist, but attention to the history, symptoms, and progress will determine the question.

Syringomyelia (abnormal dilatation of the central canal of the spinal cord) often begins with muscular atrophy as a marked symptom, and may be confounded with wasting palsy, the chief points of distinction between which are, the loss of power of per-

ceiving heat, or, often, to distinguish between heat and cold, and the appearance of trophic changes, such as a dusky or purplish hue of the hands, with a uniform thickness resembling myxœdema, the development of blebs and ulcers, and changes in the nails. Arthropathies are sometimes met with.

PROGNOSIS. Very unfavorable, although the danger to life is often very remote. The disease may be arrested and remain stationary for years.

THE TREATMENT.

Place one hand under the occiput, the other under the chin, extend and gently rotate the head; treat the muscles of the neck thoroughly, deeply; raise the clavicles, chest, arms, and liberate the intercostals; treat the sides of the spine from above downward, in dorsal and lumbar regions, manipulate, extend and flex the lower limbs, and treat profoundly the special muscles involved, using a vibratory manipulation deeply so as to be sure to empty all the venous blood for the time, freeing the larger veins, stretching the muscular fiber in all the muscles implicated. Stimulate the spinal cord by pressure with thumb and fingers, while the limbs are raised upward or pulled backward, either way, as directed elsewhere for that purpose. See also that the terminal end filaments are freed in sphincters of the lower outlets of the body, which aids greatly in promoting capillary circulation in the whole body. Freedom of circulation of all of the fluids must be restored before good can result. This can only be done by the means recommended Osteopathically. Three-fourths of the trouble may be removed by the carrying-out of the directions for treating the neck. The brachial plexus (or region) requires special and thorough manipulations. All the movements applicable to the accomplishment of this purpose may be necessary in any given case. Osteopathy is the *sine qua non* in all obstructed fluid-flow in all manner of pathological conditions, effectual when properly and scientifically applied. Treatment every third day, gently, thoroughly.

SPINAL SCLEROSIS.

SYNONYM. Duchenne's disease.

DEFINITION. A myelitis; an increase in the connective tissue of the spinal cord, with atrophy of the nerve structure proper.

CAUSES. Generally a hereditary neuropathic diathesis;

syphilis; alcoholism; mineral poisons; shock or injuries to the cord; exposure to cold and wet; mostly occurring between the ages of thirty-five and fifty-five; males more liable than females. It is said that railroad enginemen and firemen, as well as conductors and other trainmen, suffer from this and other spinal diseases by reason of the continual concussion of railway travel. The freedom from the disease in the negro has been noted by Mitchell.

PATHOLOGICAL ANATOMY. The changes in the cord are gradual in their development and follow a longitudinal instead of a transverse direction. The form, consistency, and color of the cord are altered, it being atrophied, indurated, and of a grayish color. The changes are hyperplasia of the connective tissue, with granular degeneration, atrophy, and disappearance of the proper nerve elements. The nerve roots undergo the same fibroid change. The joints undergo remarkable atrophic degeneration—the arthropathies or Charcot joints consisting of an osseous hyperplasia, the joint enlarging to an enormous extent.

VARIETIES. 1. Lateral sclerosis; 2. Posterior sclerosis, or locomotor ataxia; 3. Ataxia paraplegia; 4. Cerebro-spinal sclerosis.

I. PRIMARY LATERAL SCLEROSIS.

SYNONYMS. Antero-lateral sclerosis; spasmodic tabes dorsalis (Charcot); spastic spinal paralysis (Erb).

DEFINITION. A degeneration of the lateral columns of the cord; characterized by paraplegia, contractures of the muscles, with exaggerated reflexes.

PATHOGENY. The exact morbid condition is still a subject of discussion. The site of the lesion is the lateral white columns, in some cases extending to the anterior horn, and involving the whole length of the cord. The changes consist in an interstitial hyperplasia of the connective tissue, and an atrophy of the nerve elements.

SYMPTOMS. The onset of the disease is very gradual, with increasing feeling of heaviness and weakness in the limbs, progressing of a complete paraplegia. There is also jerking and twitching, with cramps and stiffness of the muscles of the paretic limbs. The spasms of the legs gradually increase in extent as the power lessens, until at last the legs, whenever extended, pass into a condition of strong extensor spasm, rigidly fixing them to

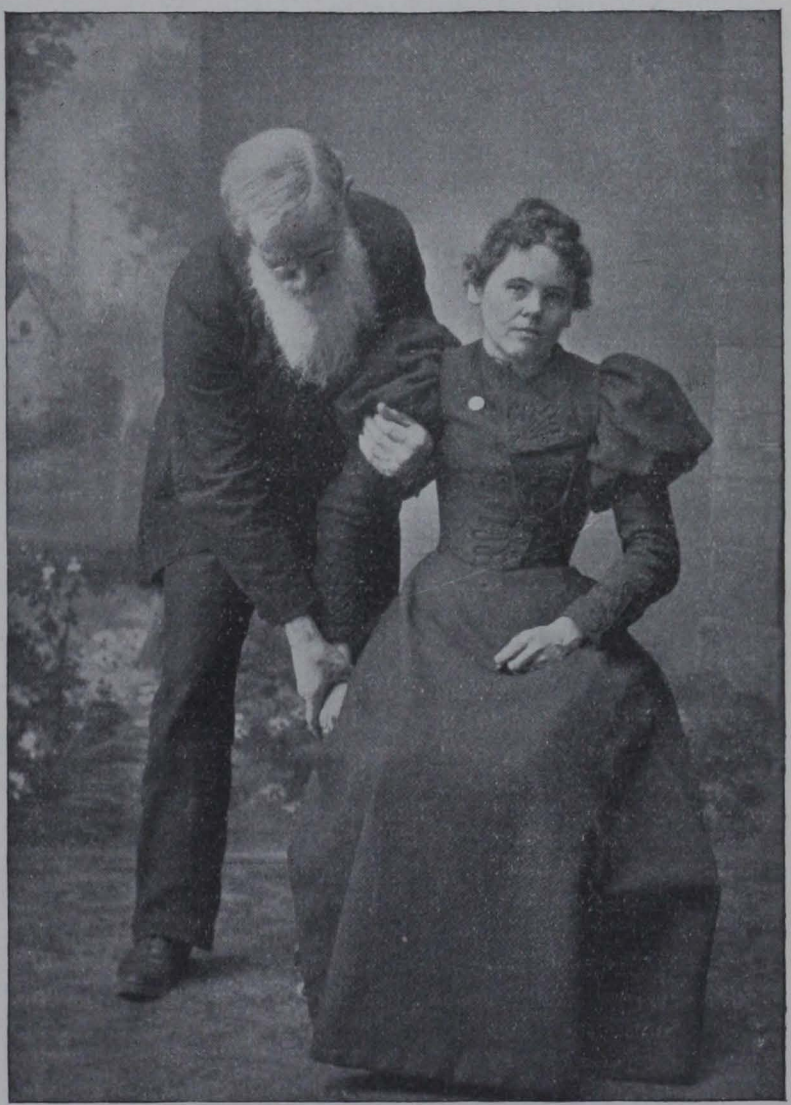


PLATE LI.—Shoulder and Arm Raising.

the pelvis, so that the patient lies rigid; if one leg is lifted from the couch by the observer, the other leg is moved also. The spasm may be such that the knee can not be passively flexed by any force that can be applied to it until the spasm has become less. When flexed the limb is comparatively supple; but if it is then extended, the spasm instantly returns, making the limb rigid, and often completing the extension, just as the blade of a knife opens out under the influence of its spring, "clasp-knife rigidity." Occasionally there occur brief flexor spasms, drawing the legs up. The knee-jerk is greatly exaggerated, and there can also be developed rectus-clonus and ankle-clonus. The spastic gait is characteristic, termed by Hammond "'the waddle"; the legs drag behind and are moved forward as a rigid whole, the toes catching against the ground, the patient showing a tendency to fall forward. Sensation is unaffected. As the morbid process extends upward the superior extremities suffer in the same manner as those of the lower.

Electro-contractility early impaired and gradually declining until abolished.

DIAGNOSIS. The gradual development of weakness in the legs, excess of myotatic irritability and spasms with developing spastic gait render the diagnosis clear. If the symptoms develop suddenly or acutely, the morbid condition is not of the degenerative variety.

PROGNOSIS. Complete recovery rare. If the condition is early recognized its progress may be held in check for a long time.

TREATMENT. See Cerebro-Spinal Sclerosis.

II. LOCOMOTOR ATAXIA.

SYNONYMS. Posterior spinal sclerosis; tabes dorsalis.

DEFINITION. A chronic degeneration of the posterior columns of the spinal cord and the posterior nerve roots, characterized by loss of coordination, neuralgic pains in the limbs, loss of sensation and reflexes, and visceral and trophic changes.

PATHOGENY. "A progressive destructive process which has a selective influence on certain tracts in the posterior columns with their roots and ganglia and to a less extent on the peripheral nerves, particularly the optic. The nerve fibers of the cord are first involved. Their destruction is not a simple wasting, but is accompanied with evidence of irritation, such as swelling

of axis cylinders, and, secondarily, proliferation of connective tissue and slight congestion." (Dana.)

SYMPTOMS. Locomotor ataxia may be divided into three periods: 1. Disturbances of sensation; 2. Loss of coordinating power; 3. Paralysis. The onset of the disease is gradual, by sharp, darting, electric-like pains in the lower limbs, with disorders of the gastro-intestinal and genito-urinary tracts. Associated with the pains is a loss of sensation in the feet, the patient being unable to distinguish between hard and soft substances in walking, and, if the upper portion of the spinal cord be affected, is unable to coordinate the muscles of the fingers sufficiently to button his clothing. A sensation of formication over the surface, especially over the lower limbs, and about the waist, the knee, and the ankle, is present; there is nearly always a feeling of constriction about the trunk—the girdle.

Loss of coordination or ataxia, the subject being unable to walk upon a straight line with his eyes closed, and with difficulty if his eyes are opened. Inability to preserve the erect position with the feet close together, the body swaying widely and the patient falling on standing with closed eyes (Romberg's symptom), and as the malady progresses he throws his feet and legs in the most grotesque manner. Although the patient is unable to coordinate the muscles, their power is not lost, for, on being supported, he can kick or strike with his usual force.

The sight is early impaired, due to atrophy of the optic nerve, either double vision or inability to distinguish between different colors. Very early there is loss of pupil reflex to light, the reaction to accommodation being present (Argyll-Robertson symptom). As the disease progresses the sensation becomes more and more blunted and pain is slowly felt, in cases it being several minutes until the sticking of a pin is appreciated. A characteristic sign of the disease is the abolition of the patellar tendon reflex (Westphal's symptom), as well as other reflexes in the lower limbs. Loss of the sensation of temperature also occurs. The electro-contractility is decreased in the affected limb. General emaciation is marked.

Either early or late in the disease occur disturbances in micturition and loss of sexual power and often desire. There also occur in a fair number of cases, painless swelling and disintegration of various joints, particularly the knee and elbow—the tabetic arthropathies, or Charcot joint.

At any period of the disease peculiar crises, or neuralgic

attacks occur: if griping pains in stomach with vomiting, gastric crises; if renal pain or colic with disturbed urinary flow, nephralgic crises; if pain in bladder, vesical crises; if pain in rectum with hemorrhoids, rectal crises; if severe paroxysm of coughing, bronchial crises; if constriction of throat with dyspnœa, laryngeal crises; if cardiac pain and tachycardia, cardiac crises.

Paralysis finally ends the suffering of the patient. There is generally an entire absence of cerebral phenomena.

DIAGNOSIS. There are three pathognomonic symptoms of locomotor ataxia whose presence render the diagnosis positive; they are Westphal's symptom—absence of patellar reflex; Romberg's symptom—swaying of body and inability to maintain erect position with closed eyes; and the Argyll-Robertson symptom—loss of pupil reflex to light, but reaction to accommodation retained. Another important point is the history of syphilis five to twenty years before. Chronic myelitis is characterized by paralysis, and the courses of the affections are otherwise so different that an error should not occur. Diseases of the cerebellum present symptoms of disordered coordination, but they are the result of vertigo, and associated with headache, nausea, and vomiting, and neuralgic pains and eye symptoms absent. Paraplegia is a true paralysis, while sclerosis is not. Neuralgic pain is not a symptom of paraplegia. Multiple neuritis gives loss of power with pain, but does not present the three pathognomonic symptoms mentioned above.

PROGNOSIS. Unfavorable. Few if any recoveries are recorded, although rarely the progress has been retarded for a long time. There are some claims of recoveries of locomotor ataxia in the early stage, but that a cure of a genuine case, extending to the second stage, is ever effected, seems very questionable.

TREATMENT. See Cerebro-Spinal Sclerosis.

III. ATAXIC PARAPLEGIA.

SYNONYMS. Combined lateral and posterior sclerosis; antero-lateral sclerosis.

DEFINITION. A chronic degeneration of the lateral pyramidal tracts and of the posterior columns of the spinal cord; characterized by gradual developing paraplegia, with ataxia, and spasms of the limbs.

CAUSES. The causes are not so well determined as in other varieties of spinal sclerosis.

PATHOGENY. A sclerosis of the lateral and posterior columns of the spinal cord. It is to be noted that the posterior columns show the morbid changes higher up than in locomotor ataxia—the dorsal rather than the lumbar regions—and that the root-zone of the postero-external column is much less involved. Nor do the lateral tracts show the same degree of involvement as in spastic paraplegia.

SYMPTOMS. The onset is slow and gradual, with loss of power in the lower extremities. The muscles involved are particularly the flexors of the thigh and knee. One leg may be weaker than the other. There is also ataxia, the patient being unsteady when standing with feet together, and he tends to fall if the eyes are at the same time closed. Spasms of the lower extremity gradually develop and finally become as marked as in spastic paraplegia. The knee-jerk reflex is increased, quick and extensive, and rectus and ankle clonus can be developed. The sexual power is early lost. Incontinence of urine is frequent. Sensation is unimpaired and neuralgic pains are absent, as are eye symptoms.

DIAGNOSIS. The conditions ataxic paraplegia is most liable to be mistaken for are locomotor ataxia and spastic paraplegia. The presence of knee-jerk and loss of power in lower extremities are of value in discriminating from locomotor ataxia. Spastic paraplegia is not associated with ataxia—indeed, ataxic paraplegia is spastic paraplegia plus incoordination.

PROGNOSIS. As a rule favorable.

TREATMENT. See Cerebro-Spinal Sclerosis.

IV. CEREBRO-SPINAL SCLEROSIS.

SYNONYMS. Multiple sclerosis of the brain and cord; cerebral sclerosis; spinal sclerosis; disseminated sclerosis (Charcot).

DEFINITION. A degenerative disease of the brain and spinal cord; characterized by pains in the back, disorders of sensation, loss of coordination, tremor on motion, scanning speech, and some mental impairment.

PATHOGENY. The disease consists of the development of patches of grayish, translucent, tough nodules, varying in size from a minute microscopical object up to the size of a walnut, varying in number and widely distributed in the white matter of

the hemispheres, ventricles, optic thalamus, corpus striatum, peduncles, pons, and cerebellum, while in the cord they are found in both the white and gray matter and in the columns. The deposits are also found in the nerve roots and nerve trunks. The nodules are composed of the neuroglia, much altered, and a newly-formed connective tissue. The result of the growth of the nodules is pressure upon the nerve structure, ending in its degeneration.

SYMPTOMS. Charcot divides this disseminated sclerosis into three varieties, depending upon the site of the marked changes, as the brain, the cord, or a combination of the two. The latter variety is the more common.

Rarely, the malady is ushered in with apoplectiform symptoms, but generally the onset is insidious, with pains more or less severe in the limbs and back, which are attributed by the patient to rheumatism. Also a feeling of formication, itching, and burning in the limbs. Loss of coordination of the hands in writing, or the feet in walking, or a jerky coordination, followed after a time by paresis, more or less general, with contracture of the muscles. Voluntary movements of the paretic limbs develop a tremor—the shaking tremor—which subsides when the limbs are at rest—intention tremor, with shaking of head. An early and frequent condition is nystagmus. The loss of coordination, with tremor and with contractures of the muscles of the legs, has given rise to the "waddle" or "hop" gait when walking. There are also present headache, vertigo, mental impairment, with an unnatural contentment of the feelings and with the surroundings, a scanning or slurring speech, disorders of vision and hearing, sexual disturbances, vesical disorders, gastric and other crises and often the development of bed-sores. Knee-jerk and muscular reflexes are exaggerated. The disease is progressive, the symptoms developing as the various nerve tracts are invaded.

DURATION. Ranges from a year to twenty years, an average being five or ten years.

DIAGNOSIS. Paralysis agitans may be mistaken for disseminated sclerosis. The chief points in diagnosis: the presence in paralysis agitans of the fine tremor continually without shaking of the head, with a peculiar flexion and rigidity of the hand, while in cerebro-spinal sclerosis the tremor is produced only on movement of the muscle, and is associated with shaking of the head. Paralysis agitans, a disease of middle life, sclerosis under forty years. Changes in the voice, speech, and vision are present in

cerebro-spinal sclerosis, but absent in paralysis agitans. Tumor of the pons or crus is accompanied with wild, jerky incoordination closely resembling disseminated sclerosis, but tumor also has headache, optic neuritis, local spasm, and local paralysis. General paralysis of the insane and disseminated sclerosis are frequently confounded, as are locomotor ataxia and primary lateral sclerosis.

PROGNOSIS. Unfavorable. The disease slowly but steadily progresses, chronic nephritis or tuberculosis frequently developing and causing death.

THE TREATMENT.

Without regard to differentiation in the special manifestations of the diseased conditions of the cord, under the name of spinal sclerosis, including primary lateral sclerosis, posterior sclerosis (locomotor ataxia), ataxic paraplegia, and cerebro-spinal sclerosis, we would endeavor to impress on the mind of the reader that every variety of effects characteristic of the pathological conditions of the spinal cord, under whatsoever name classed, is the result following obstructed venous circulation. That removed early and kept removed means a cure of the disease, arrest of progress, a restoration of health, a normal state.

In the incipiency of these affections there will be observed an increase of temperature of the head a little above the normal, and a tendency to contracture of the muscles of the neck, a tenderness at the sides of the cervix, all indicative of pressure. This pressure is the direct result of venous obstruction; this starts in the large veins, caused by exposure to lower temperature, damp atmosphere, producing enervation of nerve force from pressure on the walls of the nerves, resulting in partial or complete paralysis at the ends, hence stasis of blood in capillaries and in venoles and lymphatics, so that waste material can not escape. Decomposition ensues, chemical changes take place, and destruction of the connective tissue is the result. From this picture, what course suggests itself but to take off the pressure? To enter into the profundity of these phenomena would not comport with the present mental status of the profession, and therefore would be "beating the air," and the effects nil. We therefore proceed to demonstrate our philosophy by "works" rather than spend time in a vain effort to open the eyes of those who "won't see" until they "feel the grip" of actual observation.

Our direction for general treatment covers the ground neces-

sary, and we therefore suggest a thorough general treatment, starting with extension and rotation of the cervix, with thorough neck manipulations, so as to open all the channels (the veins and lymphatics) that, obstructed, cause the difficulty under consideration, raising the clavicles, arms, chest muscles, treating the spine all the way down, manipulating the lower limbs, flexing and extending them, freeing the circulation all over the body, equalizing the nerve forces, taking off all pressure according to directions given for that purpose; and do this repeatedly, at least three times each week, steadily, persistently, until new tissue can be formed.

The Tissue Elements may have to be supplied in some instances, such as Kali phos., Silicia and Calc. fluoricum. The reasons for these will be found elsewhere in this work.

DISEASES OF THE NERVES.

SIMPLE NEURITIS.

DEFINITION. An inflammation of the nerve trunks; characterized by pain and paresis of the parts supplied by the affected nerve trunk.

CAUSES. Wounds and injuries or compression of nerves; cold and damp; syphilis (?); lead.

PATHOLOGICAL ANATOMY. Hyperaemia, followed by exudation into the nerve sheath and connective tissue, "which becomes softened and ultimately breaks down into a diffluent mass." Migration of white corpuscles takes place into the neurilemma. Recovery may occur before destruction of the nerve elements is produced, absorption of the exudation occurring. "It is important to note that when inflammation occurs in a nerve it may extend from the point first diseased upward (neuritis ascendens), or downward (neuritis descendens)."

SYMPTOMS. The onset may be accompanied with febrile reaction. The most decided symptom is pain along the course of the nerve trunk and its peripheral distribution, of a burning, tingling, tearing, intense character, increased by pressure or motion. If the affected nerve be a mixed one—sensory and motor—spasmodic contractions and muscular cramps occur, followed by impaired motion, terminating in paresis of the muscles

innervated by the affected trunk. If the inflammation proceed to destruction of the nerve trunk, wasting and degeneration of the muscular tissue ensues. Various trophic changes also occur, such as cutaneous eruptions, and clubbing of the nails.

The electro-contractility is impaired or lost.

DIAGNOSIS. Myalgia or muscular pain is not associated with paralysis, nor does the pain follow the course of a nerve trunk.

Neuralgia has the pain, but, as a rule, not the tenderness of neuritis.

PROGNOSIS. Generally favorable, with proper treatment.

TREATMENT. See Multiple Neuritis for treatment.

MULTIPLE NEURITIS.

SYNONYMS. Polyneuritis; peripheral neuritis; disseminated neuritis; degenerative neuritis; pseudo-tabes; alcoholic paralysis; beri-beri (Brazil and India); kakke (Japan).

DEFINITION. A parenchymatous inflammation of a number of symmetrical nerves, simultaneous or in rapid succession; characterized by pain, numbness, loss of power, or ataxia, with muscular atrophy. Mental symptoms are often associated.

CAUSES. Alcoholism; syphilis; malaria; lead, arsenic, or silver; following diphtheria, typhoid fever, and rheumatism.

Beri-beri and kakke are epidemic varieties of multiple neuritis and the result of a special poison. The probability is that the various cases named develop in the blood a poison, having a particular susceptibility or "selective action" for nerve fibers.

PATHOLOGICAL ANATOMY. The affection is generally bilateral and symmetrical. An important characteristic is its peripheral distribution, the inflammation being most intense at the extremities of the nerves, lessening progressively toward the center, usually terminating before the nerve roots are reached. The inflammatory process affects the nerve fibers primarily and the sheath and connective tissue secondarily—a parenchymatous inflammation. The affected muscles are paler and smaller than normal, the fibers reduced in size and undergoing granular changes.

SYMPTOMS. All plans yet suggested for classifying the varieties of multiple neuritis are imperfect. The onset may be sudden, even overwhelming, causing rapid death, but is usually

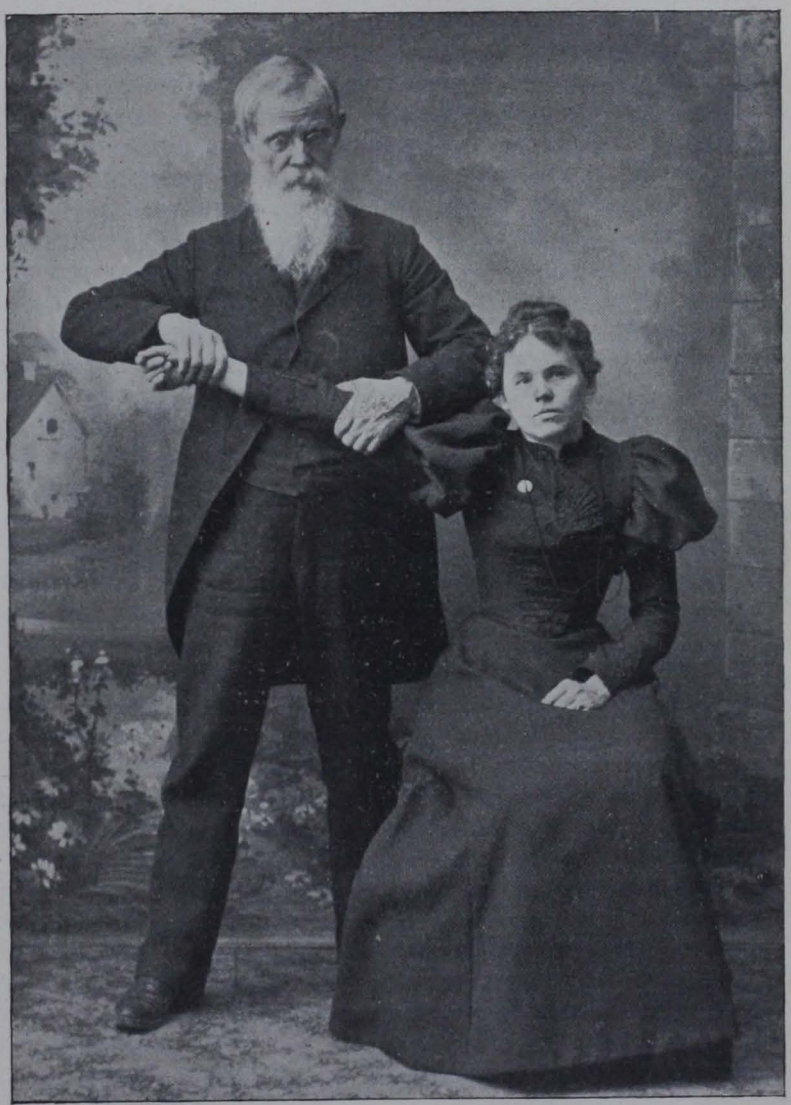


PLATE LII.—Showing How to Strain Elbow Joint.

subacute or chronic in its course, the symptoms being widespread in proportion to the acuteness, intensity, and cause of the malady. The symptoms may be described under three forms—a motor, a sensory, and an ataxic form. The motor form shows motor weakness, chiefly involving the flexors of the ankles, the extensors of the toes, and the extensors of the wrist and fingers in the forearms. Inflammation of the anterior tibial or peroneal nerve in the leg, and the radial branch of the musculo-spiral in the arm, resulting in the double "wrist-drop" and "foot-drop" so characteristic of this disease. Any nerves of the body may be affected, the symptoms varying with the particular nerves. The sensory form shows pains, tenderness, tingling, and numbness, with loss of cutaneous sensibility. The ataxic form shows incoordination with or without sensory disturbances, but with loss of the muscular sense. The forms may all be associated, in greater or less extent, in any one case.

Muscular atrophy begins early and progresses with the disease. The knee-jerk is feeble or absent. The electro-contractility is feeble or lost. In alcoholic cases, there may be delirium, mania, and delusions, associated with tremors.

Trophic changes may occur in the nails, hair, and skin. The characteristic glossy condition of the skin, with some œdema, is due to involvement of the vaso-motor nerves. Rarely the vagus, optic, and laryngeal nerves are involved.

The disease may be ushered in with fever, 101-103 degrees F., rapid, feeble pulse, headache, nausea, vomiting, with delirium or confusion.

The alcoholic variety affects chiefly all the limbs; the malarial, the legs; diphtheria, the pharyngeal and motors of the eye; rheumatic, the face; and lead, the arms.

DIAGNOSIS. In no disease is an early diagnosis so important from a therapeutical standpoint. Early treatment may prevent months of suffering and idleness.

Since the symptoms of this wide-spread affection have been properly separated from disease of the spinal cord, with which they were formerly always associated, the diagnosis is very readily determined.

PROGNOSIS. As a rule favorable if early and proper treatment be instituted.

THE TREATMENT.

In simple and multiple neuritis we have further demonstration of the results of the most serious character from undue press-

ure on the tissue, caused by contracture of muscle fibers. Whether due to over-stimulation of the nerves controlling the fibers of muscles, or the pressure sufficiently strong to paralyze the nerves controlling said fibers, the results are stasis of blood in the vessels, lymph in spaces, thus increasing pressure by the accumulation of fluids, separating the nerve footlets, cutting off communication—hence death in the parts, with everything involved. To repeat the directions for the treatment indicated seems superfluous. The philosophy holds good everywhere, in all pathological conditions; the only thing to become familiar with is the tracing out to a legitimate conclusion the real condition existing, and remove the pressure. That done, wait on Dame Nature to perform her accustomed, unerring duties—rebuild the "waste places." Whether we find the condition acute or chronic, the cause is the same, and requires the same means to remedy—may be a longer course in the latter than the former, but the removal of the pressure must be done in the one case as in the other. Persistence in the treatment, intensifying it in parts needed, lessening in others according to indications, should not be lost sight of, for amelioration can be obtained in no other way so surely as this way. The cranial congestion is removed by freeing the pressure on the neck veins, by relieving muscular contracture.

NEURALGIA.

DEFINITION. A disease of the nervous system, manifesting itself by sudden pain of a sharp and darting character, mostly unilateral, following the course of the sensory nerves.

CAUSES. Hereditary; anaemia; malaria; syphilis; metallic poisons; anxiety; mental exertion; exposure to cold and damp; injuries of a nerve trunk.

PATHOLOGICAL ANATOMY. The old axiom of neuralgia being "the cry of the nerves for pure blood" is perhaps only part of the truth. The changes in the nerve trunks or centers have not as yet been determined. A fair number of cases present the changes of neuritis.

VARIETIES. 1. Neuralgia of the fifth nerve; 2. Cervico-occipital neuralgia; 3. Cervico-brachial neuralgia; 4. Dorso-intercostal neuralgia; 5. Lumbo-abdominal neuralgia; 6. Sciatica.

NEURALGIA OF THE FIFTH NERVE.

SYNONYMS. Tic-douloureux; Fothergill's disease.

SYMPTOMS. Paroxysmal pain, of a sharp, darting, stabbing character, most common at points along the course of the supra- and infra-orbital branches of the fifth nerve of the left side, and attended with an increased lacrymation. When of any duration, nutritive changes are observed in the nervous distribution, to-wit: œdema along the course of the nerve, gray eyebrows and convulsive twitches of the muscles, termed "tic-douloureux," tenderness at the infra- and supra-orbital foramina as well as along the course of the nerve distribution.

CERVICO-OCCIPITAL NEURALGIA.

SYMPTOMS. Paroxysmal pain, of a sharp and lancinating, or deep, heavy, tensive character, along the course of the occipital nerve upon one or both sides, extending from the vertex, and on the neck as far down as the clavicle, and upward and forward to the cheek. May be associated with hyperaesthesia of the skin, and with cramps in the cervical muscles, and with attacks of herpes. A sensation of cracking at the nape of the neck is an annoying symptom in many cases.

CERVICO-BRACHIAL NEURALGIA.

SYMPTOMS. Paroxysmal pain, of a severe, boring, burning, or tensive character, with sensations of numbness and weakness of the arm, hand, shoulder, scapula, and mamma, with tenderness along the cervical plexus. Œdema of the arm and other parts along the distribution of the cervical plexus occur if the neuralgia be of long duration, the result of nutritive changes, the limb at times becoming pale, the skin glossy, dry, and harsh.

DORSO-INTERCOSTAL NEURALGIA.

SYMPTOMS. Paroxysmal pain of a sharp and lancinating character, along the fifth and sixth intercostal spaces, often associated with the development of herpes, the so-called herpes zoster, or "shingles." Tenderness at the points where the nerves emerge from the intervertebral foramina at the sides of the chest and at points in front.

LUMBO-ABDOMINAL NEURALGIA.

SYMPTOMS. Paroxysmal pain of a sharp and lancinating, at times heavy and dull character, following the course of the ilio-hypogastric nerve, ilio-inguinal and external spermatic nerve, supplying the integument of the hip, the inner side of the thigh, the scrotum and labium.

THE TREATMENT.

If there is any condition that calls loudly for deep breathing (oxygenation of the blood), neuralgia does. The first thing to be done in all cases should be to induce deep inspirations. If the patient is lying down, on the back, let the operator take hold of one arm, draw it strongly up to the side of the head, stretching all of the intercostals, chest muscles and ribs upward and outward on either side, placing finger-ends to the sides of the spinous processes, beginning at the last cervical and extending down to the eighth dorsal vertebra. Then raise the clavicle, extend the neck, manipulate the neck muscles; then, lastly, treat the face, sides of head, and use moderate pressure and vibration over painful localities for two to five minutes, until relieved. The treatment should be slowly done, modified according to the susceptibility of the impressions of the patient, and intensity of suffering from the affection. The above directions apply to all neuralgias of the face. The occipital neuralgia may require variations in the treatment as conditions indicate. Intercostal and lumbar neuralgia succumb to movements of the chest and the dorsal muscles involved.

SCIATICA.

DEFINITION. A neuritis. Pain following the course of the sciatic nerve. The sacral plexus is made up of the fourth and fifth lumbar and the first two pairs of sacral nerves.

SYMPTOMS. Sciatica usually follows an attack of lumbago, the pain becoming fixed in the sciatic nerve; at times it is a true neuritis.

The pain is sharp, tearing, shooting, or lancinating in character, increased upon motion, shooting along the course of the nerve into the hip, inner side of the thigh, calf of the leg, ankle, and heel, at one or all of these points, in paroxysms lasting from a few hours to twenty-four hours or longer. The tactile sensation in the foot and motility in the limbs are impaired, and if of long duration, wasting of the limb occurs.

DIAGNOSIS. Rheumatism, so called, is the only condition likely to be confounded with neuralgia. The history of the attack, the character of the pain, with its localized spot of tenderness, should prevent such an error.

PROGNOSIS. If promptly and properly treated, unless the

result of pressure of an exostosis, aneurism, or other tumor favorable.

THE TREATMENT.

Sciatica—the dread of most practitioners who depend on medication—is usually relieved, modified, as well as cured, by Osteopathic treatments, continued for a reasonable time. The whole system should be treated down to the seat of the pain, then the lower limbs demand special attention. Strong flexion of the limb on the side of the affection is to be made, with the fingers of one hand pressing on the sides of the spines of the lower lumbar vertebrae, abducting the limb against the fingers, so as to press the muscular tissue of the sciatic filaments, and follow the course of the nerve as far down as possible, between the ischium and the trochanter as low as the lower portion of the ischium; then raise the limb, holding it by the ankle with one hand, and the other pressing strongly above the knee, endeavoring thereby not only to stretch the sciatic, but the muscles of the dorsal and lumbar regions. This same object may be accomplished by placing the ankle over the shoulder and both hands locked above the knee, as shown in illustration elsewhere. All the muscles of the hip and upper portion of the thigh should be manipulated deeply, from side to side, opening the saphenous vein, so that the engorgement of veins may be reduced. The usual treatment of adducting and abducting and flexing and extending of the lower limbs should receive due attention. The vibratory dorsal and lumbar treatment should be used, especially over the lower lumbar and gluteal area. Select any means illustrated in this work for the relief of the patient, using all recommended as indicated in a given case, to accomplish results. Treatments should be given every day or every other day, requiring a longer or shorter time to cure, according to the nature of the case, from one to a various number, ranging over several weeks. Much benefit may be assured, and a large number of the very worst cases promised a certainty of being cured in a few weeks.

FACIAL PARALYSIS.

SYNONYM. Bell's palsy.

DEFINITION. An acute paralysis of the seventh cranial—the facial nerve, the great motor nerve of the muscles of the face—the nerve of expression.

CAUSES. Exposure to a current of cold air against the side of the face—over the pes anserinus—is the most frequent cause. Also due to injury or disease of the middle ear. Syphilis.

SYMPTOMS. The facial nerve supplies the muscles of the face, the muscles of the external ear, also the stylo-hyoid, posterior belly of the digastric, the platysma, one muscle of the middle ear, the stapedius, and one palate muscle, the levator palati; by means of the chorda tympani branch it controls the secretion of the parotid and submaxillary glands, and, possibly, the sense of taste. It also furnishes motor power to the azygos uvulae, the tensor tympani and the tensor palati muscles. The onset is usually sudden, with tingling of the lips and tongue, and upon looking into the mirror the patient is surprised by the perfectly blank, motionless side of the face; the corner of the mouth is depressed, the eyelids open, the face drawn toward the well side, and the patient is unable to expectorate, whistle, or swallow. Any of the muscles innervated by the nerve may participate in the paresis.

The electro-contractility is feeble or lost. The reflexes are abolished.

DIAGNOSIS. Paralysis of the muscles of the face occurs in hemiplegia; the points of differentiation are the presence of cerebral symptoms and the normal reflex excitability.

Facial palsy with otorrhœa, imperfect hearing, obliquity of the uvula, and loss of taste, determine its origin within the aquæductus Fallopii. It is due to peripheral neuritis if the taste be normal and the uvula straight. If other nerves are also involved the origin is central.

PROGNOSIS. Favorable.

THE TREATMENT.

The treatment for this affection is necessarily complicated, and covers a good deal of surface. The aim at first should be to take off the pressure around the stylo-mastoid foramen (the exit of the nerve or nerves from the aperture), and from the parotid gland. The stretching of the muscles of the sides and back of the neck, and rotary movements, are important, whether the trouble lies along the line of course after emerging from the foramen, or whether the trouble begins in the pons, as its supposed origin. The freedom of contracture all along the line is essential. Whether this be in the tympanic muscle, stylo-mastoid foramen, or at its origin, the cause is the same—pressure. That is the thing to remove, hence overcoming, relaxing the

muscular fibers of all of the muscles of the neck, to give freedom to the venous return blood, is essentially important. Extension of the neck, rotation gentle, firm and steady, should constitute the first movement. The freedom of all muscular fibers along every fibrilla, denominated the facial nerve, must be the order, and deep inhalations made by the patient at stated intervals, to oxygenate the blood. Strong and long-continued manipulations high up under the angles of the lower jaw are needed, pulling the muscles in various directions, manipulating the parotid glands, the submaxillary as well as the sublingual glands. In this case, as in all others, the outlets as well as inlets (the tubes leading in and out) everywhere, should be normal, then normal action will exist. Treatments to be repeated every other day.

General or Nutritional Diseases.

CHOREA.

Synonyms. St. Vitus' dance; insanity of the muscles.

Definitions. A functional (?) disorder of the nervous system; characterized by irregular spasmodic movements of groups of muscles, with muscular weakness, more or less approaching paralysis of the affected parts.

Causes. Essentially a disease of childhood; hereditary; reflex, from dentition, worms, masturbation, or fright; probably the result of rheumatism in many cases.

Pathological Anatomy. As yet there has been no constant anatomical lesion discovered, the theory of emboli having, however, many advocates.

Symptoms. The onset is usually gradual, the child seemingly grimacing or jerking the arm or hand, as if in imitation, followed soon by decided irregular jactitations of the muscles of the face (histrionic spasm), of the eyelids (blepharospasm), eyeballs (nystagmus), and the shoulder, arm, and hand, finally extending to the lower extremities, interfering with motility; in severe cases, inability of self-feeding or of holding anything in the hands. The speech is often unintelligible, the tongue constantly moving in an irregular manner.

The heart's action is tumultuous and irregular, associated

often with a soft, blowing, systolic murmur, most distinct at the base The muscles are usually quiet during sleep, although this is not always the case. The mind is somewhat blunted, the temper irritable, the memory impaired. If the irregular muscular movements are confined to one side of the body, it is termed hemi-chorea.

DIAGNOSIS. Chorea was confounded with epilepsy until the points of distinction were pointed out by Sydenham.

Paralysis agitans has general muscular tremor, beginning in one limb, gradually progressing, uninfluenced by treatment; a disease of the elderly. Post-hemiplegic chorea is the choreic movement of a paralyzed limb.

PROGNOSIS. The vast majority of cases recover, but relapses are very frequent.

THE TREATMENT.

It will generally be found that accompanying this affection nerve waste from the genitalia constitutes the largest factor. The sympathetic nervous system, the terminal filaments, are bound. When the terminals of the sympathetic and the motor footlets are separated, "insanity of muscular action" is to be looked for as a natural sequence. Orificial examination should be attended to, correcting whatever is needed in the case there, then manipulations Osteopathic will be beneficial. In this day of discovery, advancement, intelligence along the lines of knowledge of the human system, the practitioner becomes culpable who neglects his duty in this regard. No wonder it is a "disease of childhood," for the ordinary practitioner depends so largely upon the *vis a tergo* of his small quiver of one-sidedness in faith and practice, that a new departure to him is a "shameful violation of the code," and deserves immediate excommunication from men or mortals of the "vile" wretch who would attempt to digress. Take off the "fig leaf" long enough to learn the condition of the patient, then a rational treatment may be instituted, unless the doctor should, after finding "pinched sympathetic nerve filaments," conclude the efficacy of some "high potency" is adequate to the emergency, and giving it, await "the time with patience" for months, and let his patient suffer it out. Such a course is worse than reprehensible. Carry out the motto—"Take off the Pressure."

It will be found upon examination that there is more or less spinal tenderness about the second lumbar vertebra, and at the sides of the neck at the exit of the spinal accessory as it emerges

PLATE LIII.—Raising Clavicle, Patient Sitting Up.

at the posterior border of the sterno-cleido mastoidei. More or less hyperaesthesia all along the spine is present, and the reflexes readily induced. The Osteopath will overcome much of the trouble by a systematic course of rectal divulsions, either with digits, or, better by far, with a bivalve speculum. Pratt's is a first-class instrument for that purpose, and every Osteopath should have one. Many conditions will present themselves where the use of it will afford perfect satisfaction, not attainable otherwise. It will assist in taking off the pressure, and do it effectually, as desired, and arouse latent energies that have slept for years, perhaps. Its use starts up capillary circulation and regulates the heart's action, sometimes, better than through the vaso-motor filaments at the upper cervix. These divulsions may be repeated at longer or shorter intervals *ad libitum*, if not too much at a time—it is a good plan to do so gradually; and in constipation it is an effectual means, dilating each sitting a little more, until contracture is overcome and regularity ensues. It also is needed in many cases where a dormant, relaxed state exists, re-establishing normal vitality, and restoring the lost powers in an incredibly short time. Use it.

EPILEPSY.

DEFINITION. A chronic disease, of which the characteristic symptoms are a sudden loss of consciousness, attended with more or less general convulsions.

CAUSES. Heredity; rarely, worry, anxiety, depression, or fright. Pressure from a tumor at the periphery, or thickening of the membranes of the brain, causing pressure; dyspepsia (?); syphilis; uterine diseases.

PATHOLOGICAL ANATOMY. There are no constant anatomical lesions, as yet, associated with essential epilepsy.

In "Jacksonian," "cortical," or "partial epilepsy," however, the "motor cortex" is irritated by disease and there occur tonic and clonic spasms of the same character as in general epilepsy, confined to a single arm, or an arm and half the face together, or may be the entire half of the body. These epileptiform attacks furnish precise data as to the locality of the lesion; spasms affecting the distribution of the facial nerve point to the lower third of the central convolution; of the arm, the middle third of the cen-

tral convolution; of the lower extremity, the upper third of the central convolution.

VARIETIES. 1. Epilepsia gravior, *le grand mal;* 2. Epilepsia mitior, *le petit mal.*

SYMPTOMS. *Le grand mal* is preceded by a more or less pronounced and curious sensation, the so-called aura epileptica.

The attack proper is sudden, the subject suddenly falling, with a peculiar cry, loss of consciousness, and pallor of the face, the body assuming a position of tetanic rigidity, succeeded after a few moments by more or less pronounced clonic convulsions followed by a coma of several hours' duration. The subject awakens with a confused or sheepish expression, with no knowledge of what has occurred, unless he has injured himself during the attack, either by the fall, or, what is very common, has bitten his tongue during the convulsions.

Le petit mal is manifested either by attacks of vertigo, the consciousness being preserved, or by a passing absent-mindedness, either form being associated with slight convulsive phenomena, followed by slight coma or mental confusion of short duration.

The mental functions are not, as a rule, injured by attacks of epilepsy, unless they recur very frequently. Indeed, when at wide intervals, the subject seems relieved by them, "the sudden, excessive, and rapid discharge of gray matter of some part of the brain on the muscles," the so-called "electrical storm," having cleared the cerebral atmosphere. The great majority of epileptics suffer from chronic gastric catarrh, and have at the same time an inordinate appetite (boulimia); indeed, an attack of gluttony may immediately precede a fit.

DIAGNOSIS. Uraemic convulsions closely resemble an epileptic attack; but the dropsy or general œdema and albuminous urine, increased temperature, of the former should guard against error.

Feigned epilepsy often misleads the most practical expert.

Jacksonian epilepsy begins as a spasm of a limb or some portion of a limb, and is confined there or may gradually extend until even a general convulsion occurs.

PROGNOSIS. The vast majority of cases will not recover under treatment, but have the frequency and severity of the attacks greatly ameliorated, but sooner or later returning with

their former severity. Cases the result of the various reflex causes usually recover when the cause is removed.

THE TREATMENT.

Many cases have been radically cured by this treatment. The treatment is to be directed to the neck first; not that "the atlas is dislocated" every time, but the contracture of the neck muscles prevents normal circulation in the brain. The spine needs special treatment from start to finish, the sphincters relieved and special attention given to the general circulation. Treatments should be given every other day. The splanchnics are greatly at fault, and are to be looked after; the renal splanchnic righted, and the stomach should be rested by doing without breakfast, and observing not to eat too much at one time. Deficiency of neurine substance is supplied by the tissue element known as Calcarea Phosphoricum. As much as five grains thrice daily is requisite. A systematic cording of the lower limbs persistently has cured many cases. Hypnotism has also lessened the attacks in number and severity by Therapeutic Suggestion during the "profound stage," after reflexes are corrected. The intelligent Osteopath will get good results from his treatment, scientifically applied three times each week. This, with orificial surgery where indicated, should satisfy a majority of cases. We surely commend this treatment to the consideration of those interested, as accomplishing better, more satisfactory results than any other. Pay special attention to the reflexes. Take off the pressure everywhere in the body. Set the atlas, if dislocated.

HYSTERIA.

DEFINITION. A nutritional disorder of the nervous system, of the nature of which it is impossible to speak definitely; characterized by disturbances of the will, reason, imagination, and the emotions, as well as motor and sensory disturbances.

CAUSES. A morbid condition confined almost exclusively to women. Young girls, old maids, widows, and childless married women are the most frequent subjects of the disorder. The paroxysms frequently develop during the menstrual epoch. The menopause is another frequent period for its manifestation. A peculiar condition of the nervous system, either inherited or acquired, is responsible for the phenomena of hysteria, the peculiar

manifestations being excited by the disturbances of either the sexual, digestive, circulatory, or nervous system.

Hypochondriasis, a peculiar mental condition, characterized by inordinate attention on the part of the patient to some real or supposed bodily ailment or sensation, a continual introspection, as seen in males, is a condition much like the hysteria of the female.

PATHOGENY. Structural alterations have thus far not been detected in cases of hysteria; it is thus a functional disturbance of the nervous system. It should, however, be borne in mind that hysterical manifestations frequently develop during the prevalence of organic diseases.

SYMPTOMS. These will be considered under the headings of the hysterical paroxysm, and the hysterical state.

The Hysterical Paroxysm or fit occurs nearly always in the presence of others, and develops gradually with sighing, meaningless laughter, causeless moaning, nonsensical talking, and gesticulations, or a condition of fidgets followed with a sensation of choking dyspnœa, and a ball in the throat—the globus hystericus. These and similar symptoms precede the fit, during which the unconsciousness is only apparent, the patient being aware of what is transpiring about her. During the paroxysm the patients may struggle violently, throwing themselves about, their thumbs turned in and their hands clenched. Again, spasmodic movements occur, varying from slight twitching in the limbs to powerful general convulsive movements, and to almost tetanic spasms. The paroxysm ends by sighing, laughing, crying, and yawning, and a sensation of exhaustion. During the attack it will be noted that the surface and face are normal, showing absence of respiratory embarrassment, the breathing varying from very quiet to spluttering and gurgling sounds, the pupils not dilated, the pulse normal, the temperature normal, and absence of foaming at the mouth and wounding of the tongue.

The Hysterical State is shown by disturbances of the mental and sensory-motor functions respectively. It may be a permanent condition or occur at intervals with greater or less severity.

Mental Disturbances.—The patients are emotional, erratic, excitable, impatient, and self-important, showing marked defects of will and mental power.

Sensory Disturbances.—This is either a condition of exaggerated sensibility or hyperaesthesia, as shown by the marked effects from the slightest irritation and the cutaneous tenderness

along the spine, or a condition of anaesthesia as shown by the apparent absence or recognition of pain after severe irritation, or a perverted sensibility, as shown by the feeling of tingling, numbness, and formication. Sensibility to heat or cold is often absent. There is great perversion of the special senses in many of the cases.

Charcot, referring to the ovarian hyperaesthesia of hysteria, says: "It is indicated by pain in the lower part of the abdomen, usually felt on one side, especially on the left, but sometimes on both, and occupying the extreme limits of the hypogastric region. It may be extremely acute, the patient not tolerating the slightest touch; but in other cases pressure is necessary to bring it out. The ovary may be felt to be tumefied and enlarged. When the condition is unilateral, it may be accompanied with hemianaesthesia, paresis, or contracture on the same side as the ovarialgia; if it is bilateral, these phenomena also become bilateral. Pressure upon the ovary brings out certain sensations which constitute the aura hysterica, but firm and systematic compression has frequently a decisive effect upon the hysterical convulsive attack, the intensity of which it can diminish, and even the cessation of which it may sometimes determine, though it has no effect upon the permanent symptoms of hysteria."

Motor Disturbances.—These phenomena embrace every variety of motor disturbance, from exaggerated excitable movements to defective or complete loss of power. With the paralysis that may occur, neither nutrition nor sensation is constantly impaired. Hysterical paralysis is liable to frequent and sudden changes, the loss of power often disappearing suddenly. Aphonia, from paralysis of the laryngeal muscles, is a frequent form of paresis. Some hysterical patients refuse to even make an attempt at speech.

"A curious enlargement of the abdomen is observed sometimes, constituting the so-called phantom tumor. This region presents a symmetrical prominence in front, often of large size, with a constriction below the margin of the thorax and above the pubes. The enlargement is quite smooth and uniform, soft, very mobile as a whole from side to side, resonant, but variable on percussion, and not painful. Vaginal examination gives negative results, and under chloroform the prominence immediately subsides, returning again as the patient regains consciousness."

Among the numerous other symptoms that may develop in

a hysterical patient are disturbances of digestion, circulation, respiration, and disorders of micturition and menstruation.

Among other phenomena that belong to the hysterical state are to be mentioned Hystero-epilepsy, a condition of hysteria to which is superadded the convulsion, epileptic in form; Catalepsy, a condition in which the will seems to be cut off from certain muscles, and in whatever position the affected member is placed, it will so remain for an indefinite time; there may or may not be unconsciousness and loss of sensation; Trance, the individual lying as if dead, circulation and respiration having almost ceased; Ecstacy, a condition in which the individual pretends to see visions and acts in a most ridiculous manner.

DIAGNOSIS. The hysterical state is so general in its manifestations that it is to be borne in mind in diagnosing all ailments occurring in women. The diagnosis is attended with great difficulty, however, and requires the display of all the skill of the clinician to prevent error.

PROGNOSIS. Death from either a hysterical fit or the hysterical state is the rarest of events, if it ever occurs. The ultimate recovery of a hysterical patient is of frequent occurrence. Marriage has cured many cases, although it can hardly be advised by the physician.

THE TREATMENT.

This is another one of the affections resulting from "tightened sphincters," and relieved by taking off the pressure. The bivalve is the most effectual source of relief ever devised by mortals. It is always a matter of merciful gratitude that wells up in my inmost soul toward Professor Pratt for his discoveries of reflexes and the marvelous uses of the bivalve. I have had universal success with its use in hysteria. It acts like magic—at once. The coordination of the nerve force is established and the patient experiences the most salutary effect. All the foolishness is at an end (generally at the lower end), and when we take off the pressure of the internal sphincter there is a "calm and peaceful rest" comes over the mind of the patient that beggars description. When I was younger than I am now, it was my painful service to sit up all night, watching over women whose lives were supposed "to hang in the balance" and liable to "wink out" momentarily, with medicine in hand (*a la* spoon), in readiness to pour down the victim's throat, through closed, clenched canines, as often as possible, and the most incongruous compounds that Allopathic

ingenuity could mix together—"Hoffman's Anodyne," for instance; assafoetida, *et id omne genus*—when, lo! I would succeed in "curing my patient," get the honor, and sometimes pay, for it. But I have lived in a better time, learned a new idea, adopted it, and it does not require nasty drugs, anxious thoughts, nor impatient waiting. It effectually answers. I could give the reader a long list of cases that it has been my lot to witness, suffering with this affection, and narrate many laughable instances associated therewith. I was called to see a 17-year-old unmarried, beautiful blonde, living about two miles from my office. It had rained all the night before; the road was the kind we have in Illinois under those circumstances. The case was one of an urgent nature—the girl was thought to be dying, and horse-flesh suffered ere I got there. I approached the bedside of that bundle of youthful, marriageable loveliness with feelings of profound caution and solemnity, carefully felt the pulse, ascertained that death was far away, and had no sooner let go the wrist than my supposed dead or dying patient suddenly arose, started for the door, reached it, plunged forth into the rain, out into the pasture, made direct headway for a pond of water of considerable area two hundred yards from the house. She rushed out into the center of the body of water, where it was at least eighteen inches deep, and as she got to this particular spot, two young men who had followed her, caught her, brought her back to the house, where she seemed as well reconciled as if nothing had happened. I ordered a mustard poultice, placed it the whole length of the spine, put her on a pallet on the floor, and as signs of restlessness began to appear in my patient, I ordered a clyster of sapo, salt, castor oil and terebinth, fully a quart, altogether, used. That settled the difficulty; relaxation of the sphincters ensued. Relief was immediate. Since then I have had many cases, and taught by that simple lesson, have had no difficulty in relieving every case. The bivalve saves all the trouble of fixing up and using compounds. It induces immediate response, satisfactory to patient and doctor. Properly used, there is not the slightest danger. Some cases are extremely contracted, and a small bivalve is the kind needed at the start, then a larger one. Whichever is used, let it be well oiled, introduced carefully, then, by turning the screw attached until it is spread out about half an inch, the blades apart, the handles are gently squeezed, then relaxed; then spread a little more, gradually divulsing the sphincter until the patient is relieved of the spell of hysteria.

I had a case of this sort in my practice out in Denver, Col., that was restored in one minute. Another in Kansas City, Mo., who would drop dishes and throw them across the room involuntarily, that two or three sittings cured effectively, and more grateful beings you could scarcely find. Thanks for this marvelous discovery—the bivalve.

NEURASTHENIA.

SYNONYMS. Spinal irritation; nervous prostration; nervous exhaustion.

DEFINITION. A debility of the nervous system, causing an inability or lessened desire to perform or attend to the various duties or occupations of the individual. Professor Bartholow describes it as consisting "essentially in an exaggerated susceptibility to bodily impressions and false reasoning thereon."

CAUSES. It may result from various chronic diseases; mental worry or emotion; overwork, as "whenever the expenditure of nerve-force is greater than the daily income, physical bankruptcy sooner or later results." (Jackson.) Neurotic temperament; sexual excesses; alcohol; tobacco.

SYMPTOMS. Nervous debility may affect any organ of the body. It is a condition of nerve-tire or exhaustion, and hence the nervous energy necessary for functional activity of any particular organ may be wanting, a fair example being seen in cases of nervous dyspepsia.

One of the earliest manifestations of nervous exhaustion is an irritability or weakness of the mental faculties, as shown by inability to concentrate the thoughts, and efforts to do so causing headache, vertigo, restlessness, fear, a feeling of weariness and depression, together with the army of symptoms attendant on nervousness.

There may be ocular disturbances, cardiac palpitation, coldness of the hands and feet, chilliness followed by flashes of heat, followed in turn by slight sweating. Patients are troubled with insomnia, or fatiguing sleep, accompanied with unpleasant dreams.

In the male there are genito-urinary disorders, with pains in the back, giving the dread of impotence. In females, painful menstruation, ovarian irritation, and irritable uterus.

DIAGNOSIS. It is of importance to determine between a true

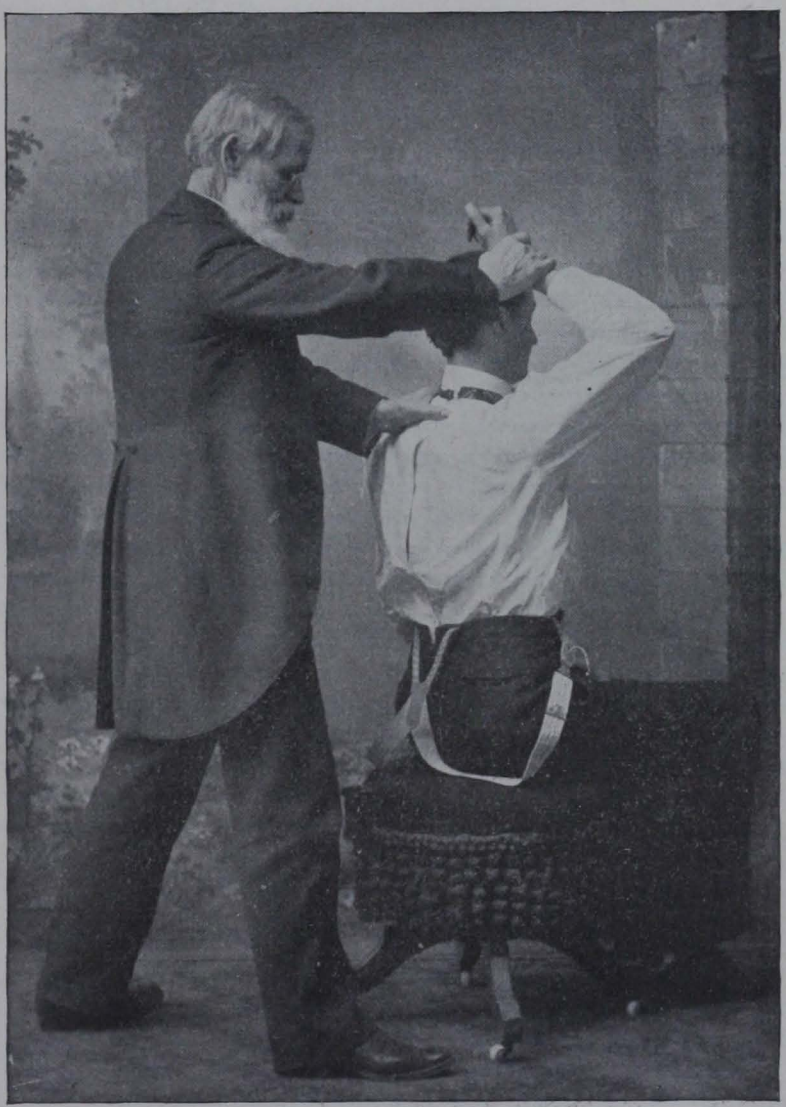


PLATE LIV.—Raising Chest Muscles and Treating Spine.

nervous exhaustion and nervous debility, the result of organic disease. A study of the history of the case, together with the symptoms, should prevent error.

PROGNOSIS. Unless there be a tendency to mental disorders, the prognosis is good.

THE TREATMENT.

This condition is remediable, so far as the system in general is concerned, according to the ability to assimilate the lost elements. Arrest in the first place the waste causing it, then supply the elements. This is done in various ways—through direct exhibition per oram, in the form of food. Re-establish the nerve force by properly stimulating arterial peristalsis and capillary activity by the manipulations recommended therefor, the vasomotor area receiving due attention. Vibratory manipulations along the spine, stretching the spinal cord gently, liberating all of the muscles of the body, start up new forces throughout the system. Do not neglect the orifices of the body (the sphincters) in these affections. Stimulate the glandular system. If there is ever a general all-over treatment given, do so in these cases. Manipulate gently but deeply all of the muscles, stimulate the spinal area all the way from cervix to coccyx, as patient is able to bear the treatments—as often as every third day.

Ferrum phos. and Calc. phos. are often indicated. It will appear plausible to a rational being to supply these elements when it is known that certain proportions of the sixteen elements constitute the physical organism, and a deficiency of either one or more causes disturbance in all the rest. The body is not a whole body without the elements of which it is composed, and it requires certain ones to render the rest active and assimilable.

EXOPHTHALMIC GOITRE.

SYNONYMS. Graves' disease; Basedow's disease.

DEFINITION. A disease of the nervous system; characterized by protrusion of the eyeballs, enlargement of the thyroid gland, dilatation of the arteries, and palpitation of the heart.

CAUSES. An undemonstrable condition of the nervous system, either inherited or acquired, is the predisposing cause of Graves' disease. Among the exciting causes are anaemia, shock, fright, chagrin, worry, and reverses of fortune.

It is more common in women than in men.

PATHOLOGICAL ANATOMY. "Some structural alterations have been found, in a majority of cases, in the sympathetic ganglia, and especially in the inferior ganglia." (Bartholow.) The veins and arteries of the thyroid gland are dilated, the result of a vaso-motor paralysis. The enlargement of the gland is the result of the dilated vessels, and a serous infiltration of its tissues, followed, if long continued, by hypertrophy. A considerable increase of fat behind the eyeballs has been observed. In the majority of cases more or less anaemia exists.

SYMPTOMS. The development of the quaternary of symptoms may occur suddenly, the result of some great shock to the nervous system, but in the majority of instances the symptoms develop slowly and insidiously, with cardiac palpitation, with paroxysms of more marked acceleration, tachycardia, the pulse rate varying from 90 to 120, 150, and rarely as high as 200 beats per minute; soon pulsations of the vessels of the neck and thyroid gland may be felt and seen. The enlargement of the thyroid gland—the goitre—appears gradually after the development of the circulatory disturbances, although rarely it may be the first symptom observed. The goitre is elastic, rather soft, and has a thrill similar to an aneurism. The degree of enlargement varies in different cases, and in none ever attains a very great size. Following the development of the goitre occurs the protrusion of the eyeball—the exophthalmos—which may be confined to one eye, but usually occurs in both. Prominence of the eyeball may be the first symptom observed, but usually it does not develop until after the appearance of the goitre. The degree of protrusion varies from a slight staring expression to a point so great that the eyelids can not cover the balls. Associated with the protrusion of the eyeballs is incoordination in the movements of the eyelids and the eyeball, the sign of Graefe, so that when the eyes are quickly cast down the eyelids do not follow them, the sclerotic being visible below the upper lid. Vision is unimpaired. Conjunctivitis may arise, the result of the imperfect protection of the protruding ball by the eyelids.

Associated with the pathognomonic symptoms are nervousness, irritability of temper, headache, insomnia, vertigo, fits of despondency, aphonia, and cough the result of pressure of the goitre, disorders of digestion, increase of temperature, anaemia, and loss of flesh.

DIAGNOSIS. The fully developed disease presents no difficulties in diagnosis, but during its incipiency, before the char-

acteristic symptoms have appeared, the disease may be confounded with such conditions as cardiac disease, neurasthenia, lithaemia, malaria, or incipient phthisis.

PROGNOSIS. Recovery occurs in a fair number of cases, but is slow and tedious. The disorders of the circulation lead to dilated heart in many cases, and ultimately death occurs from this cause. Relapses are frequent.

THE TREATMENT.

This is another of those affections that manifest motor and sympathetic separation of terminal filaments. This is caused by muscular contraction, so that partial paralysis occurs, or infiltration of blood and lymph in the tissues, separating footlets and producing the incoordination of forces.

The treatment consists in gently extending the neck muscles, with rotary motions of the head, thoroughly manipulating the face and neck muscles, stimulating the vaso-motor area, re-establishing nerve connection between the sympathetic and motor nerves, start the fluids out and regulate the heart's action and arterial circulation. Open the channels—the veins of the thyroid —by the same manipulations as recommended for Goitre. Treat the patient according to indications, as in other affections that complicate this one. Free the obstructions and take off nerve pressure repeatedly, every two or three days, and good results soon begin, and a rapid amelioration ensues.

Enlarged thyroid gland is the simplest thing to cure in our practice, usually.

The engorgement in the recent stages is due to obstruction to the venous blood in the thyroid veins. These may be relieved by raising the clavicle and then manipulating the gland upward. sidewise, and pressing the gland in the various ways that knead the substance of the gland itself, freeing all of the muscles of the neck thoroughly and keeping the clavicles raised. This should be done by the thumb of the operator close to the sternal end of the clavicle, pushing outward as the arm is raised upward and backward in either of the various ways shown in this work; steadily holding the clavicle out for a moment greatly facilitates emptying the venous blood therein. The treatment should be given every other day. Many cases of recent origin are cured very soon. Those of long duration, and that have become indurated, require many months' treatment to effect a cure. The cure of the latter kind will be more easily effected by the aid and the

introduction of the tissue element that has a special affinity for glandular indurations—Calcarea fluoricum (6x), three or four grains three times a day. It will be found to be the case with those troubled with goitre that the clavicles are extremely close down to the chest walls, preventing venous return circulation, by pressure of the sternum and the clavicles on the jugulars and other small veins in that region. Take off the pressure, then work the blood out of the gland. The thymus gland does not seem to be involved in goitre. Persons very young are not troubled with goitre; as a rule, those over 12 years old have it.

TETANY.

SYNONYMS. Tetanilla; intermittent tetanus.

DEFINITION. A succession of tonic, usually bilateral, painful muscular spasms, occurring at irregular intervals, without loss of consciousness.

CAUSES. It has been observed in those having a family history of nervous disorders.

PATHOLOGY. The disease is very rare in America.

SYMPTOMS. Tetany is the occurrence of intermittent spasms in the muscles of the arms, hands, legs, or feet, or rarely the face and larynx (laryngismus stridulus), associated with pain.

The hands are thrown into a position such as they assume in writing, or such as is taken by the hand of a midwife; or the hand may be tightly closed, or one or more fingers may be cramped. The elbows and shoulders may be, at times, affected. In the feet the toes are drawn down and the instep upward, like in equinus. The knees may be cramped or the legs extended. Any muscles may be involved. Trousseau pointed out that in those suffering from tetany, pressure upon the affected extremities at certain points will excite the spasms.

The duration of the spasms varies from a few moments to several hours, the intervals being from an hour to a day or more. A certain periodicity is noticed as to the hour of the day or night.

The electro-contractility is increased, as are also the reflexes. The consciousness is always preserved, although the patients are very nervous.

DIAGNOSIS. Tetanus and tetany may be confounded, and yet trismus is rare in the latter, and always present in the former.

PROGNOSIS. Favorable.

THE TREATMENT.

The strangest thing we have to contend with in educating the people is to get them out of their old ways of thinking. It does not seem to dawn on the minds of some that any other way of thinking than "our fathers" thought should be permitted! This age has demonstrated a few facts that would be profitable to consider, even those pertaining to the extent of lessening our sufferings, lightening our burdens and expanding the horizon of our zenith intellectually, physically and spiritually.

The old way of treating such cases was to further paralyze the muscular fiber (that which was not affected) in order to procure harmony in such cases as the one under consideration; but now we would lift the pressure off of the already spasmodically paralyzed nervous system and harmonize action that way, without producing a worse condition than found to exist. Truly, "the world do move." The spinal nervous system is at fault in this affection. When righted tetany subsides, as a matter of course. Beginning at the vaso-motor area, we stimulate all along the whole course of the spinal column, then remove pressure from the sympathetic nerve terminals. The measures recommended to re-establish normal tension everywhere in the body should be perfectly familiar to the reader at this juncture, and there should be no difficulty in rightly applying them. Magnesium phosphate introduced in this affection will be found adapted to conditions admirably.

TETANUS.

SYNONYMS. Lockjaw; trismus; cephalic tetanus.

DEFINITION. An acute or subacute infective disease, characterized by muscular rigidity, with paroxysms of tonic convulsions, the mind remaining clear. Idiopathic tetanus when no open wound is discoverable. Traumatic tetanus when an open wound is present. Tetanus neonatorum when it attacks infants. Lockjaw or trismus when the jaw alone is involved. Cephalic tetanus when the throat and face are affected.

CAUSES. The result of a specific bacillus, which usually gains access to the system through an abrasion.

PATHOLOGICAL ANATOMY. In the post-mortem examinations which have been made, no uniform morbid appearance was discovered, on microscopical examination.

The brain, cord, lungs, and muscles are markedly congested, and show minute hemorrhages, such as are met with in all cases of death from convulsions, and which occur chiefly during the process of death.

In four post-mortem examinations of cases dying from tetanus, at the Philadelphia Hospital, marked chronic nephritis was observed. Probably the future may show some connection between nephritis and tetanus, by which the specific poison is not eliminated as it might be were the kidneys normal.

SYMPTOMS. The onset is rather sudden, with stiffness of the jaw, neck, and tongue, and some difficulty in swallowing, which increases in extent, the stiffness passing down the spinal muscles to the legs, which are held in a firm spasm.

Gradually tonic spasms develop, which, involving the jaw muscles, cause "lockjaw"; the face muscles, "risus sardonicus"; neck and trunk muscles, "opisthotonos"; these tonic convulsions are associated with intense pain, and the patient suffers the greatest distress, particularly if the chest muscles are involved. Usually the febrile reaction is slight, but in many cases 102 deg. F. to 104 deg. F. is reached, and in some instances, as death approaches, 108 deg. F. to 110 deg. F. may occur, rising still higher after death. The mind remains clear till carbonic acid poisoning occurs. Usually a wound, not severe, can be found, the symptoms developing some two weeks after its occurrence.

The tonic spasms are developed by any sources of irritation, a draught of air, shaking of the bed or floor, suddenly opening the door of the room, the presence of a visitor, or attempts at speaking or movement.

DIAGNOSIS. The symptoms are so characteristic, with the addition of a history of a wound, that an error seems hardly probable.

Tetany in the spasms chiefly affect the extremities, the muscles being free in the interval and trismus a late or very rare condition.

Strychnine poisoning often closely resembles tetanus, but there is no beginning trismus and more rapid development of the symptoms. No history.

Hydrophobia does not have trismus, but respiratory spasm, excited by attempts at swallowing, with increasing mental symptoms.

PROGNOSIS. Unfavorable. The great majority die.

THE TREATMENT.

All forms of tetanus are amenable to Osteopathic treatment, properly applied; but where it is caused by a healed-up wound from a sharp pointed-instrument(a rusty nail or thorn), it will be essential to open the wound afresh, take off the pressure there, and that settles the matter with that case—cures it. All the manipulations that might be made without the means recommended would prove unavailing. The sudden spasmodic characteristics of tetanus indicate pressure on terminal-end sensory filamentous footlets. Certain stages in the circulation of the fluids seem to increase the pressure, and then the spasm supervenes, lasting until the mechanical effort moves off the pressure, and then the spasm ceases. The affection seems to follow wounds of the feet or hands, where numerous tendons, fasciae and small muscular fibers unite, and where terminal nerves are the most pressed upon by movements of structure, hence suggestive of spasmodic action, reflexly.

The treatment should begin at the back of the neck, the vaso-motor area, then extend the neck gently, remove muscular tension in the neck muscles, raise the clavicle, treat all the way down the spine, thoroughly relaxing all of the muscles of the body, removing all venous obstructions everywhere in the body. Avoid mental excitement as much as possible. Give general treatments once or twice daily, if indicated. Remember the reflexes and terminal nerve filaments in the sphincters.

OCCUPATION NEUROSES.

SYNONYMS. Professional neuroses; artisans' cramp.

VARIETIES. Writers' cramp; piano-players' cramp; telegraphists' cramp; violin-players' cramp; dancers' cramp.

DEFINITION. A group of affections of the nervous system, characterized by the occurrence of spasm (cramp) and pain in groups of muscles, in consequence of overuse or frequently repeated muscular acts.

CAUSE. Undetermined. It has been noticed that many persons suffering from occupation neuroses have a family history of nervous affections.

SYMPTOMS. The symptoms of any of the varieties named generally develop gradually and slowly, by a feeling of stiffness in the used member, the part feels fatigued and heavy, until it is

impossible to use it, from the occurrence of spasmodic contractions; pain on using the affected muscles, often associated with tremor, and in many cases with an actual paralysis. Associated with the loss of power to follow the usual occupation are nervousness, mental worry, and often depression. There is often the sensation of prickling and numbness in the crippled member.

The electro-contractility is preserved until the atrophy of non-use develops.

DIAGNOSIS. Calling to mind the history of the case and its results, in being limited to one member, the nature of the condition is evident.

PROGNOSIS. Often unfavorable. Some recoveries are reported.

THE TREATMENT.

The neck muscles, the cervical ganglia, and down to the middle of the dorsum should be treated, stimulating the brachial area thoroughly, profoundly, several times. The vibratory manipulation of the muscular system over the upper chest, shoulders and neck and back, and raising the clavicles, arms, ribs, etc., attended to in their order, as directed elsewhere for general and special effects, every day or two, and a cure is to be expected in a short time. A change in the manner of holding the pen, brush or instrument, if practicable, will be well to consider.

Our position is that all kinds of paralysis is due to direct pressure at the origin of, or along the line of the nerve supplying tissue or muscle. The nervous system that originates motion comes out of vertebral foramina, and is distributed to every part of the body. Many obstructions are to be found at their exit from these foramina, through muscular contracture, and that removed cures the difficulty. A constant pressure eventually results in permanent paralysis, sometimes denominated "progressive paralysis."

PARALYSIS AGITANS.

SYNONYMS. Shaking palsy; Parkinson's disease.

DEFINITION. A nervous disease of unknown pathology, characterized by tremors, progressive loss of power in the affected muscles, moderate rigidity, with alterations in the gait and at times mental changes.

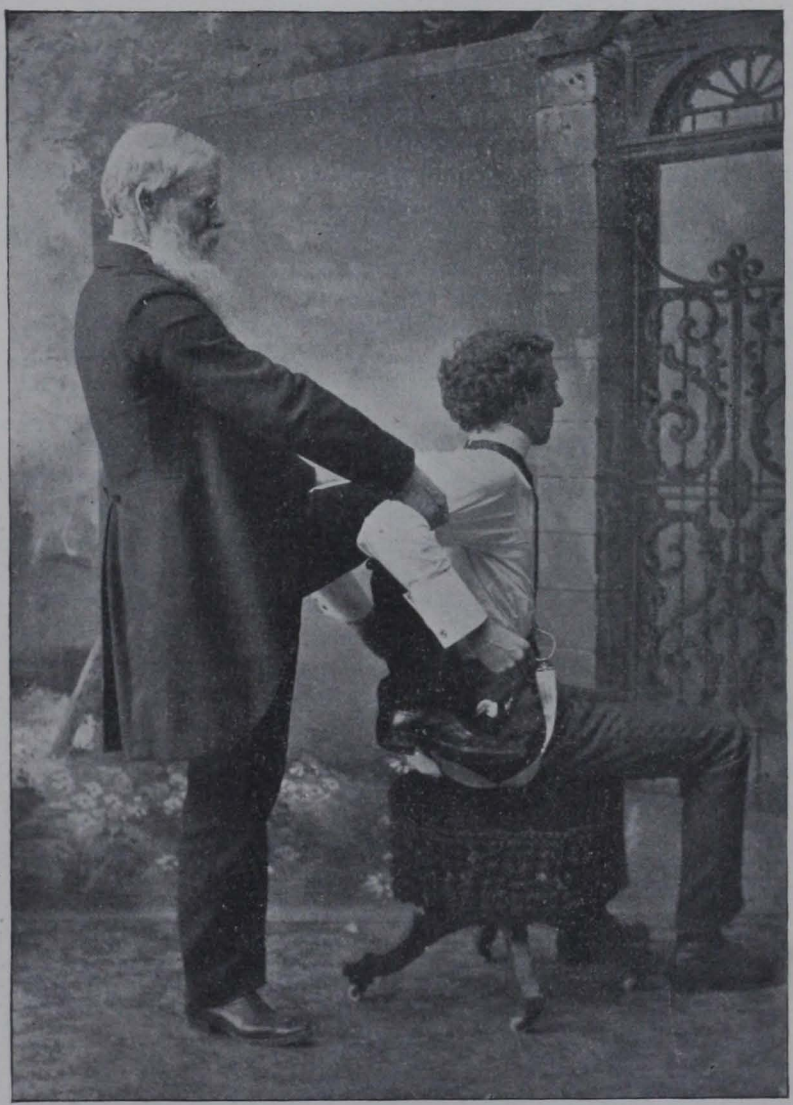


PLATE LV.—Knee–Chest Expansion Process.

CAUSE. Age seems to be an etiological factor, most cases developing after fifty years. Most frequent in women.

PATHOLOGICAL ANATOMY. No characteristic lesion yet determined. It being a disease of past middle life, there is probably an interstitial hyperplasia of some layer of the cortex, from alterations in the intima of the vessels.

SYMPTOMS. The onset is gradual, the tremor beginning in one of the extremities, oftenest the hand and forearm. At first it can be controlled by the will, for a time at least, and is suspended by voluntary movement. The disease gradually extends until an entire side or the upper or lower limbs are involved. The face and head rarely present tremors, but are not exempt. A peculiar rigidity of the affected muscles is characteristic of the advanced stage. "At this stage of the disease the hands are apt to assume the so-called bread-crumbling position, *i. e.*, the thumb and the fingers approximate and move restlessly over one another, as in the act of crumbling bread. There is often a tendency on the patient's part to go forward—so-called propulsion—and this is sometimes so marked that if the patient is once started in a walk forward, his gait becomes more and more rapid, and he can not stop himself." (Gray.) The patients are usually restless and annoyed with insomnia. The general health is fair. The mind is generally retained, although melancholia and mild dementia have been noted in a few cases.

DIAGNOSIS. Disseminated sclerosis has a tremor, but only on voluitary movements—intention tremor. There are also scanning speech and ataxic gait, with mental enfeeblement, as shown by an unnatural contentment with physical condition and surroundings.

Chorea is a tremor, but the movements are general, and particularly involving the muscles of the face. Again, chorea is a disease of children and young adults.

PROGNOSIS. Radical cure not seen. Improvement often results from early treatment. The disease does not tend to shorten life.

THE TREATMENT.

The general treatment, restoring normal circulation, is the proper thing, and many cases are greatly benefited and cured if treatment is had in the early stages. The motor nerves are at fault, and are to be reached through the spinal column. Treat the whole length of the spine and restore normal circulation.

MENTAL DISEASES.

MELANCHOLIA.

SYNONYMS. Depression of spirits; psychalgia.

DEFINITION. A variety of mental alienation, characterized by more or less profound depression of the emotions, with either no marked intellectual disturbance, or the presence of more or less incoherence, and the association of hallucinations and delusions. The cerebral mechanism developing a condition of supersensitiveness, all impressions are exaggerated, and a state of abnormal self-consciousness existing.

VARIETIES. Melancholia simplex; melancholia hallucinatory; melancholia agitata; melancholia attonita; chronic melancholia.

CAUSES. Hereditary predisposition. Failing health. Grief. Domestic and financial worries. Neurasthenia. Menstrual irregularities, pregnancy, childbirth, or lactation. Climacteric. Gastric and intestinal irregularities. Alcoholic and sexual excesses. Organic brain diseases. Religion rarely causes insanity, though it frequently gives color to it. Most common in females and in the young.

PATHOLOGY. The alterations in the nerve structure, underlying an attack of melancholia, are undetermined. Anaemia and sluggish nervous energy are constant phenomena, but are hardly the only conditions disturbing the cortex.

SYMPTOMS. Melancholia may be the initial stage of mania, delusional insanity, or paretic dementia, or a stage of *folie circularis*.

Mental.—The cardial condition is a feeling of depression, misery, or mental anguish or pain, for which no adequate cause exists. The onset is usually gradual, with a disposition to neglect duties and self, the patient worrying over a something they can not explain. The world is dark and gloomy, with a foreboding of some awful calamity that is to affect or wreck the patient or his family. Suspicion, distrust, and often fear of wife, children, relatives, or friends. Insomnia is a constant and stubborn symptom. The memory is maintained, and the reasoning faculties are usually intact, except upon the painful sensations.

The patient may sit quietly or be restless, according to the character of the emotions affected.

Physical.—The patient presents either an anxious or a woe-begone expression. Headache, and particularly a post-cervical ache, is a very constant symptom. The skin is dry and harsh, the respirations superficial, the cardiac action slow and feeble; there are gastric catarrh, constipation, and scanty, high-colored urine. The tongue is flabby and coated, and the appetite is poor. The refusal to take food is most characteristic.

Hallucinatory melancholia is an aggravated form of the disease, where, in addition to the painful mental reflexes, are distressing hallucinations and illusions, the patient living in a realm of terror. The attack may be the result of a delusion, but much more frequently the depression and foreboding give rise to the delusion. The delusions of melancholia are usually of self-accusation, self-abasement, and justified persecution; the patient feels that he is being punished for some transgression, imaginary or otherwise.

Melancholia agitata are those sad cases seen in continual agitations, in which the fearful and distressful thoughts and imaginations cause wringing of the hands, and prayers beseeching help, with tears flowing down their cheeks, crying out for assistance and protection. Incoherence and violent impulses are frequent.

Melancholia attonita, or melancholia with stupor, the patients seeming to be overwhelmed, sitting mute, motionless, and expressionless, refusing to assist themselves in any way whatever, often requiring mechanical feeding. Memory is usually impaired in this form; attacks of violence may occur.

Chronic melancholia is the continuation of the depression over a long period, the individual living in the fear of impending danger or punishment for supposed acts for months, often with apparent lucid periods.

Suicidal impulses are present in a fair proportion of cases of melancholia, and unless there be everlasting vigilance the patient will succeed in his insane desire.

Diagnosis. The cases of simple melancholia are readily determined. Melancholia agitata is frequently mistaken for acute mania. Melancholia attonita closely resembles acute dementia, a condition, it is but fair to mention, many alienists deny the existence of.

Prognosis. A typical attack of melancholia runs a definite

course, not unlike the typical course of a fever. Favorable in the mild cases of all forms not associated with organic disease, and who have not reached the climacteric. Pronounced cases of melancholia attonita are more apt to terminate in dementia than any other variety.

THE TREATMENT

In these cases there will seldom be found any changes in the nerve structure. The incoordination of mental action is the result of the improper assimilation of the elements, lack of motor nerve force to execute orders from the sympathetic, hence lack of proper capillary circulation, and a diminution of normal supply for the nerves or brain. Trouble in spine—twelfth dorsal.

There is no continuity of force when neurine is not furnished to direct the thought. Every element must be contained in the blood, and this blood must permeate every tissue, and it must have special nerve force executed along the lines and in ganglia where nerve substance is supplied from the blood in order to furnish the particular elements used in the manufacture of neurine. The importance of the unobstructed circulation of the fluids of the body becomes more impressively apparent the more we are permitted to see into the structure itself. Mind is the product, the result of divine creation, and there are certain processes that seem to be essential to its establishment, as well as continuance of action in any sense, and in this case normal sense.

The lifting off of the pressure throughout every part of the body impresses itself upon our mind as the essential thing to do, whether we find it in a contracted sphincter, or at the farthest confines of the sympathetic nerve filamentous structure. Repeat the process until cured. Osteopathy stands in the front ranks of remedial agencies.

MANIA.

SYNONYMS. Insanity; madness.

DEFINITION. An intense mental exaltation, with great excitement, loss of self-control, with, at times, absolute incoherence of speech, and loss of consciousness and memory. (Clouston.)

A mental condition in which there is an emotional exaltation, accompanied by illusions, hallucinations, delusions, great mental and physical excitement, and a complete loss of the inhib-

itory power of the will; in acute cases, and frequently in chronic forms of the disease, there is marked destructiveness and tendency to violence. (Wood.)

An attack of mania may be acute, subacute, or chronic.

CAUSES. Inflammation or other organic disease of the brain or its membranes. Mental shock or strain. Worry—domestic, moral, or financial. Excesses in alcohol, venery, or tobacco. Ovarian disease, or menstrual irregularities. Climacteric in those of nervous disposition. Pregnancy, parturition, or lactation. Nephritis. Anaemia. Syphilis. Hereditary predisposition.

PATHOLOGY. There are no constant morbid changes associated with mania. In all varieties of acute insanity there exists vitiated nervous energy or impaired vitality, the result of over-excitement or over-stimulation, motor disturbance, or auto-infection, the result of the imperfect elimination of the products of tissue waste. "There is no reason why a mere dynamical brain disturbance should not kill and leave no structural trace, any more than that it should for months abolish judgment, affection, and memory, and then pass off and leave the brain and all its functions intact." (Clouston.)

If death follow acute symptoms, the vessels of the brain and membranes are engorged, but in the majority of instances the brain structure is normal.

If death occur in chronic mania, the most frequent change found will be a thickened and adherent dura mater. As observed, any form of organic change may be found post-mortem in those dying of any form of mania.

SYMPTOMS. Acute Mania.—The onset may be abrupt, or following a period of emotional depression, associated with lassitude, feeling of unrest, disinclination to work, and disorders of the gastro-intestinal canal, with insomnia and an introspection; these symptoms are termed the melancholic stage of mania.

The maniacal stage is characterized by loud talking, intense egotism, violent motions of the limbs and body, great restlessness, and excitement; the thoughts flow in wonderful freedom and with amazing rapidity, the condition often resembling the symptoms of early alcoholic intoxication; as the condition continues the patient becomes either sullen, irritable, and angry, offering violence to those around him, or he becomes garrulous, talking of his personal affairs, is confidential and communicative to strangers, often making egotistic offers, passing frequently into

incoherence of language and action. Sexual passions are frequently exalted, and acts of masturbation practiced. Delusions are an almost constant symptom, of a superficial or transitory character, changing with every new appearing mood. The maniacal patient is sleepless, or may have short naps, at once continuing his chatter on awakening.

Any attack may show all of the symptoms mentioned, or any one or more of them, but the great majority of cases show intense egotism, loud talking, violent motion of limbs or body, hurry, excitement, insomnia, incoherence, and incessant noise.

The course of an attack is periods of remissions and exacerbations, with nocturnal crises; loss of flesh and mental weakness are often marked as the attack progresses.

Acute delirious mania, typhomania, is a psychosis of sudden onset, attended with increased bodily temperature, and marked by delirium with sensuous hallucinations, marked incoherence, restlessness, refusal of food, loss of memory, and rapid bodily wasting, terminating frequently in death.

Mania amenorrhoeal is applied to attacks of mania occurring at the menstrual epoch. Homicidal, suicidal, and various hysterical impulses are frequent.

Mania-a-potu is an attack of acute delirium, due to alcoholic excesses in those engaged in a sudden debauch, or who have drunk heavily and eaten little, for a comparatively short period.

Mania asthenic, in which there is a general anaemia associated with neurasthenic symptoms.

Mania Chronic.—A condition of continual mental exaltation, the acute symptoms having continued in a chronic course. The line that distinguishes between an acute and chronic mania must always be somewhat arbitrary and unscientific. The duration of the mania beyond twelve months is usually considered sufficient to determine the condition, and this is well, as it precludes the possibility of terming the condition incurable. If the term chronic mania was restricted to those cases in which, between the exacerbations of restlessness, excitement and destructiveness, were evidences of dementia, less confusion would occur.

Mania dancing is a hysterical mental state in which, through sympathy and imitation, dancing of a most grotesque and extravagant character occurs. Usually epidemic.

Mania delusional is the result of fixed delusions, either causing or associated with the maniacal outbreak.

Mania erotic, erotomania, presents systematized delusions of an erotic character, not necessarily accompanied by animal sexual desire. Nymphomania is a morbid, irresistible impulse to satisfy the sexual appetite, peculiar to the female sex.

Mania epileptica follows an epileptic paroxysm, and is often of a most violent kind, the maniacal acts being of the most treacherous and malicious character.

Mania hallucinatoria presents visual, auditory, olfactory, and other sense hallucinations.

Mania homicidal is any variety of mental disease in which there is a desire or an attempt on the part of the patient to commit murder. The condition may be the result of delusions that the persons attacked either are persecuting, or going to kill the patient, or of the excessive excitement that vents itself in destructiveness, combativeness, or desire to kill, or there may be a morbid desire, impulse, or craving to do murder, or the homicidal act may be unconsciously done during an acute delirium, or a paretic, or epileptic maniacal impulse.

Morphiomania is the insane craving for the stimulating action of morphia—a moral insanity.

Mania puerperal is the maniacal outbreak as seen in the puerperal woman. This is now thought to be of septic origin, although the mental strain through which the female has been passing is a predisposing factor.

Mania recurrent, or chronic mania, with lucid intervals of longer or shorter duration. Generally of alcoholic origin.

Mania transitoria, or ephemeral mania, is a rare form of maniacal excitement of sudden onset, violent and decided in character, accompanied by great insomnia, incoherence, and more or less complete unconsciousness of familiar surroundings. The attack as suddenly terminates, the duration being from a few hours to a few days.

Mania senile is the mental exaltation occurring in persons with senile arterial changes, or senile cerebral atrophy. Soon followed by dementia.

A maniacal outbreak may present any one or a number of the varieties named.

Terminations of Mania. About fifty per centum of acute manias, not due to organic disease, recover after periods varying from one month to several years. A fair proportion of cases make a partial recovery, and are able to return to their work, but always showing some alteration in character or affection, or

some eccentricity, or a slight mental weakness. About twenty per centum of cases terminate in dementia or mental death, and this is always the fear in each case. Two per centum of cases die, either the result of exhaustion or from the organic condition causing or associated with the attack.

PROGNOSIS. The question of recovery, partial or complete, is always difficult to determine, depending upon the cause, temperament, disposition, education, nationality and the normal mentality of the individual. Recovery is usually gradual; rarely sudden restoration occurs.

Favorable indications are: sudden onset, short duration, youth of patient, absence of fixed delusions, good appetite, increasing hours of sleep, moderate or no increase in temperature, pulse, and respiration; no evidences of mental weakness, no paralysis or alteration of pupils or articulation, no epilepsy, no unconsciousness to the calls of nature, and no former attacks. Unfavorable indications are the opposite of these, and also the presence of organic brain disease, or a strong hereditary tendency, or the possession of an excitable disposition, or nervous diathesis.

THE TREATMENT.

The Osteopathic experience along this line of mental pathology is somewhat limited, for there has been a peculiarly characteristic dearth in thought manifest, and the intense egotism of most of those who have heretofore studied the science has debarred the investigation of other and more salutary measures. The taxation of mental genius to discover a universal panacea for these conditions is hardly probable; and even if it were known, the obtuseness of mentality of those who claim to be and are recognized as sane precludes adoption. Madness is kaleidoscopic in manifestations.

Inharmony is apparent. The harmony is severed. How? The inequality originates from overstimulation of the nerve centers, or nerve terminals. Reflex action intensified or aborted accounts for the results. Mental suggestion under peculiar circumstances produces "mother's markings." Why not suggestions peripheral, from an undue pressure on the terminal nerve filaments, exercise an influence that culminates in intense manifestation of abnormality?

The experiments of Orificial surgeons demonstrate what we have said, and our own observation approbates their recorded conclusions. The genital organs, including the lower outlets,

PLATE LVI.—Side of Neck and Shoulder Treatment.

sphincter muscles, will be found to exercise marvelous influences in a large percentage of cases of mania, and these properly attended to, will greatly lessen the number of victims and cure cases abandoned by other methods, and by specialists and experts on nervous diseases. We have seen and treated persons, wild and insane, successfully by Orificial means, that manipulations Osteopathically were inadequate to modify even. Whether we invite the censure of Osteopathic practitioners or not, in our approval of means not recognized as Osteopathic (but really carrying out the principle), we shall take the liberty to recommend Orificial Surgery wherever indicated, and as a "twin sister" to Osteopathy, and worthy the most rigid scrutiny, profoundest investigation and our heartiest approval, when intelligently applied.

The general and special manipulations to remove obstructions, take off the pressure, establish nerve force, and coordinate the system with itself, demand our wisest consideration. Life is far too precious and too short to while it away in egotistic expostulation or denunciation of the means that relieve mankind, from whatever source derivable, or where found, or by whom discovered. Hemaspasia should not be lost sight of for lessening the blood pressure in the brain, when necessary. Hypnotic suggestion is remarkably effective in many mental states, changing the whole life at once.

Epileptic Insanity comes under the above treatment, and may be greatly modified thereby, persistently applied, generally and locally, as indicated.

EPILEPTIC INSANITY.

DEFINITION. A mental condition caused by or the result of epilepsy.

CAUSES. The careful study of the brain of those dying having epileptic insanity has failed to determine why some epileptics suffer from any of the insanities and others have their normal mentality, and another group are better after a convulsion.

VARIETIES. Pre-epileptic mania; post-epileptic mania; dementia epileptica; imbecility with epilepsy.

SYMPTOMS. The mental changes constituting epileptic

insanity, save in the cases of epilepsy with imbecility or idiocy, develop after some years of the ordinary epileptic paroxysms.

Pre-epileptic mania has attacks of mania some days or hours preceding the epileptic convulsion. The patient is morose, irritable, and threatening, often making homicidal attacks on those around him, be they friends or foes. Rarely the epileptic seizure is replaced by various insane, or so-called hysterical acts, as fits of dancing, laughing, crying, screaming, swearing, or scolding.

Post-epileptic mania follows the epileptic paroxysm, either taking the place of the comatose state or following after it. The maniacal acts during these outbreaks are often of the most desperate and impulsive character, many an asylum physician and attendant carrying scars, the result of attacks of post-epileptic maniacs.

Epileptic dementia is the terminal mental obliquity resulting in about thirty per centum of insane epileptics, who do not succumb before to nephritis or tuberculosis.

Epileptic imbecility is a congenital condition in which the two conditions are associated.

PROGNOSIS. The great majority of cases of epileptic insanity develop, sooner or later, either nephritis or tuberculosis. Recovery from epileptic mania is a rare occurrence. Thirty per centum of epileptic maniacs progress to dementia in from five to ten years.

TREATMENT. Apply the general treatment. Intensify locally—along cervex and spine.

CIRCULAR INSANITY.

SYNONYM. Folie circulaire.

DEFINITION. A mental disease characterized by regularly alternating and recurring periods of mental exaltation, depression, and sanity.

CAUSES. Hereditary predisposition. The exciting causes are any of those conditions which depress the brain or general system.

PATHOLOGY. There is no characteristic lesion associated with circular insanity.

SYMPTOMS. Essentially a chronic condition and probably incurable. The disease usually begins as a melancholia, the depression being an apathy and torpor rather than a mental pain;

and suicidal feelings and impulses are rare; this condition is soon succeeded by a mania, a mental exaltation, with hyperaesthesia and exaggeration of nervous functions, the reasoning power well retained; this is in turn followed by a lucid interval, often giving promise of recovery, to be sooner or later followed by another cycle. These periods follow each other with remarkable regularity, each being of the same duration. Rarely the various periods are of irregular duration.

The general health is well maintained, the patient gaining in flesh during the stages of depression and lucidity and losing during the period of exaltation.

DIAGNOSIS. The regularity of the different periods soon establishes the diagnosis.

PROGNOSIS. Generally incurable.

TREATMENT. See treatment for Mania.

KATATONIA.

SYNONYMS. Alternating insanity; Kahlbaum's insanity.

DEFINITION. A mental disease, characterized by irregular cyclical symptoms, ranging from melancholia to mania, followed by stupidity and confusion, with cataleptoid phenomena, followed by lucidity for a time, recovery, or passing to a dementia.

CAUSES. Hereditary predisposition. The exciting causes are usually the result of some excess. Rarely associated with organic brain disease.

PATHOLOGY. No characteristic lesions have been found associated with katatonia.

SYMPTOMS. A typical case begins as a melancholia, the mental depression, uneasiness, and distress followed after a variable period by mania, associated with hallucinations and delusions. This period is followed in turn by a condition of attonita, or rigidity and immobility, or a cataleptoid paroxysm; any of the stages may be followed by confusional symptoms, or a true dementia may develop. During the maniacal stage there is a tendency, in many cases, to histrionic and sermon-like declamation, or the speech may be of the verbigeration character—that noisy, incoherent, and meaningless speech seen in many manias, composed largely of the constant repetition of a few words or phrases. During the stage of attonita the presence of the so-called mutism or mutacismus, "a pathological tendency to be

silent," may continue for days, weeks, or months, or it may be interrupted by periods of verbigeration.

The immobility or rigidity so characteristic of a period of katatonia is frequently alternated with automatic, incessant, and monotonous movements—the stereotyped movements.

Patients suffering from katatonia often refuse food for days at a time and then suddenly present symptoms of boulimia. Vaso-motor and trophic changes are frequent, one of the most constant being cyanosis of the hands and other peripheral parts. Haematoma auris, insane ear, or perichondritis auriculae, is frequent. Epileptiform attacks may usher in the disease or occur during any of its stages.

DIAGNOSIS. It may be diagnosed as melancholia, mania, or a dementia, depending upon which of the cycles be first observed, but after being under observation long enough to observe a complete cycle, the diagnosis is readily determined. Katatonia differs from circular insanity in the absence of a genuine lucid interval, and the presence of the stage of attonita and catalepsy.

PROGNOSIS. The disease may continue for a number of years and recovery follow, but as a rule the prognosis is unfavorable.

TREATMENT. See treatment for Mania.

DELUSIONAL INSANITY.

SYNONYMS. Delusional mania; delusional melancholia; primary delusional insanity.

DEFINITION. A mental state, with fixed or partly systematized delusions, associated with either brain exaltation or excitement without maniacal acts, or a mental depression, minus the somatic symptoms of melancholia.

"An insane delusion is a belief in something that would be incredible to sane people of the same class, education, or race as the person who expresses it, this resulting from diseased working of the brain convolutions."

CAUSES. Cerebral and bodily exhaustion, the result of overwork, neglect of personal hygiene, or alcoholic, tobacco, drug, or sexual excesses—a neurasthenia. Impairment of the nervous centers, the result of fevers or shock. Climacteric period, worry, and insufficient food.

PATHOLOGY. Delusional insanity is a subacute, or chronic

condition; death seldom occurring, and when it does is the result of an intercurrent physical malady. In the few such cases in which post-mortem examinations have been made, the vessels of the brain were found torpid or dilated—a vaso-motor paresis causing an imperfect cerebral circulation.

SYMPTOMS. Either following an attack of acute mania or melancholia, but more commonly without either of these conditions, occurs a set delusion or delusions, which, to the patient, are so real that no amount of argument can dispel his or her belief. These cases are often classed as manias or melancholias, but, as they do not run the ordinary course of either of these conditions, they are best classed clinically by themselves. The acuteness or subacuteness of the attack distinguishes them from paranoia. The majority of the delusions are of an egotistical character, but lack the conduct or appearance of the position due to the character of the delusion. A patient with ragged clothing will assure you that he is worth millions, and yet sees nothing inconsistent between his delusion and his personal appearance.

DIAGNOSIS. Delusional mania and delusional melancholia are confounded with delusional insanity, the points of distinction being the absence of severe maniacal and melancholic acts; the patient simply possesses his insane delusion and may never refer to it unless questioned. Paranoia or monomania and delusional insanity have many symptoms in common, but in the former, if the patient believes he is Christ, he wishes to be so respected, and considers himself wronged if not so treated, while the delusional patient will say he is Christ and immediately drop the subject. There are, however, many border-land cases in which the diagnosis is difficult.

PROGNOSIS. Recovery the rule, although the delusions may exist for a number of years. Many patients who make a complete recovery will still believe that their delusions were facts.

TREATMENT. See treatment for Mania.

PARANOIA.

SYNONYMS. Monomania; chronic delusional insanity; reasoning mania; verruecktheit.

DEFINITION. A chronic mental disease characterized by fixed logical or systematized delusions of persecution, unseen or impossible agencies, or of self-exaltation, the emotions and mem-

ory being only paroxysmally defective, while, however, the life of the individual is dominated by the delusions.

The term paranoia, as it is now commonly used, to cover a group of insanities which are degenerative in origin, chronic in course, and characterized by systematized delusions, with little impairment of the emotional faculties, is not generally accepted as a synonym for monomania.

CAUSES. There is generally a hereditary predisposition to insanity in monomania or paranoia. The exciting cause may be the result of an acute mania or melancholia, or the result of alcoholism, or the result of malnutrition in those who have had a struggle to keep their position in the world. Extreme worry in individuals, with mental instability. Following primary or acute delusional insanity.

SYMPTOMS. The course of monomania is essentially chronic, the delusions becoming perfectly fixed and unchanging upon one particular subject or set of subjects, which in turn dominate the life of the individual. The most common character of these systematized delusions are, delusions of persecution or suspicion, delusions of exaltation or of pride, and delusions of unseen agents or influences.

The range these assume are most wide and varied, but always associated with the ego. The patient is being persecuted not because, as in melancholia, he has committed some sin, or thinks he has, and deserves punishment, but because the persecutors wish to deprive him of his rights, titles, or estate, or degrade him or in some way injure him.

DIAGNOSIS. In the diagnosis of monomania there are three points to ever keep in mind: first, the duration; the fixed, systematized delusions must have existed for over one year; second, the absence of symptoms of mania or melancholia; and third, the presence of systematized delusions affecting the personnel of the individual.

PROGNOSIS. Monomania is an incurable disease. Unless tuberculosis develop within a few years, dementia results.

TREATMENT. Symptomatic, and all means that promote constructive metamorphosis. Suggestive therapeutics. See treatment for Mania.

DEMENTIA.

SYNONYM. Acquired feeble-mindedness.

DEFINITION. A progressive general weakening of the mind, characterized by a loss of reasoning capacity, a diminution of feeling, a weakened volitional and inhibitory power, failure of memory, associated with lack of the power of attention, interest, and curiosity, in varying degrees, in an individual who at one time possessed these mental qualities.

FORMS. Dementia acute; dementia alcoholic; dementia apoplectica; dementia chòreica; dementia chronic, or secondary; dementia epileptica; dementia organic; dementia paralytica; dementia partial; dementia primary; dementia secondary, sequential, or chronic; dementia senilis; dementia syphilitica; dementia toxica.

CAUSES. Deficient or feeble mental inheritance; age; atheroma; following mania, melancholia, paranoia, and other forms of insanity; the result of organic brain conditions; alcoholism; syphilis; developmental changes; climacteric.

PATHOLOGY. In acute dementia the changes are dynamic. In the primary dementia there is probably atrophy of certain cells from overstimulation, the tissues being normally deficient. In secondary dementia the chief changes are, "alteration in the size of the vessels, owing to thickening and distention, the thickening being most marked in the deep layers, and in the walls of the vessels are fatty granules and haematoidin. The perivascular canals are enlarged. The changes in the cells may be described as deficiency in the number of pyramidal cells, and a want of distinctness of outline and branches, the nuclei being larger, but changed in form, and only capable of slight carmine staining." In senile dementia there is general atrophy and degeneration of all the tissues of the brain.

SYMPTOMS. The onset, extent, and variety of the impaired mentality differ greatly. In some patients the evidences of the failing mind are seen with the subsidence of the mania, melancholia, or other insanity, or soon after the development of the particular cause, while in another group of cases the development is slow and insidious. The difference in the intensity is marked; in one case the changes being scarcely noticeable, the patient being simply less active than before, showing a slight indifference to his environment, while in others the patients remain for hours alone, making no effort at movement and with little or no expression of the face, while another class of cases are

oblivious to the demands for food or drink, or the calls of nature, existing "in the darkness of perpetual intellectual and moral night." Between these symptoms are all varieties and degrees of mental enfeeblement. The physical symptoms of dementia vary with the particular cases, many enjoying the best of health, eating and sleeping well, while others are always unwell, first one organ and then another, while another group suffer from chronic diarrhoea, which finally causes death. Dementia patients seem predisposed to tuberculosis, nephritis, and apoplexy.

Acute dementia, or "stupor with dementia," is to be distinguished from "stupor with melancholia." The onset is rather sudden, with or without mania or melancholia, after some brain or bodily exhaustion, shock, or fright; the patient, a young person, "is horror-stricken, paralyzed in mind, not merely deranged, not depressed or excited, but deprived of feeling and intellect; his movements, if there be any, are automatic, but frequently he is motionless, standing or sitting, staring at vacancy for hours and days." (Blandford.) These patients will not converse, and do not reply to questions, or but slowly, and in monosyllables, and their faces have a blank expression.

Dementia alcoholic, the mental weakness resulting from excessive use of alcohol. Inebriety is a form of dementia, there existing an uncontrollable alcoholic habit, with weakened or absent will power, and impaired mentality.

Dementia apoplectica, an organic or terminal dementia, due to the cerebral changes sometimes following a severe apoplectic seizure.

Dementa choreica is a feeble-mindedness associated with chronic chorea, or, in some cases, probably the result of chorea.

Dementia chronic is the designation applied to all forms of dementia that have existed after one or more years.

Dementia epileptica is the slow mental impairment resulting from long-continued and frequently occurring epileptic convulsions.

Dementia organic, the mental deterioration resulting from gross organic brain lesions, such as sclerosis, tumor, embolism, or trauma.

Dementia paralytica is a synonym for general paralysis of the insane.

Dementia partial is an incomplete form of dementia, in which the mental enfeeblement is associated with such a degree

PLATE LVII.—To Raise Clavicle, Arm Leverage.

of intelligence and memory that the qualifying term "partial" is correct.

Dementia primary is seen most frequently in the young, developing slowly and insidiously, without any symptoms of mania or melancholia, usually in a youth who has given promise of a bright future, by a slowly progressive indifference to his former occupation, studies, or surroundings, with developing carelessness and negligence of person and proprieties, no amount of external stimulus serving to rouse the receding mentality, until finally the downward course ends in dementia so decided that, but for the history of the individual, the case would be classed as congenital.

Dementia secondary, sequential, or chronic, is the most common variety of mental impairment following mania, melancholia, and other insanities. According to Bevan Lewis, twenty per centum of manias, and fifteen per centum of melancholias, become permanent dements.

Dementia senilis, the result of cerebral atrophy, with its consequent failing mental power. Loss of memory for recent events is one of the most common symptoms. The disease often begins as a senile mania, melancholia, or delusional insanity.

Dementia syphilitica is the feeble-mindedness resulting from cerebral syphilis. This group of patients are always sanguine and assert they are "all right," "never sick in my life," and yet unable to assist or care for themselves.

Dementia toxica is the mental failure produced by the long-continued and excessive use of opium, cocaine, and chloral. Chronic plumbism is also given as a cause.

Diagnosis. Acute dementia is often misnamed melancholia with stupor, but if the patient is in the teens the probabilities are that it is a case of the former, while if past forty it is almost certainly the latter.

The distinction between dementia and idiocy or imbecility must always be determined. Esquirol's graphic description is well worth remembering: "The dement was a rich man who has become poor; the idiot, on the contrary, has always been in a state of want and misery."

Prognosis. Acute dementia is generally favorable. All other varieties are incurable. The average lifetime of dements is placed at about twelve years, the great majority dying of tuberculosis, nephritis, or apoplexy.

Treatment. See treatment for Mania.

The remarks under the head of Mania apply sometimes very forcibly to the various stages of insanity. Whatever name is applied, or however peculiar the manifestations, there is but one cause in our opinion for the whole trouble—impeded circulation. In the oncoming ages posterity will discover that man is a Cosmos, and that within his body the elements of the universe preside, and a disturbance of the elements changes the character most wonderfully, producing incoordination mentally as well as physically. Obstructions to communicating nerve tubules disturb forces, and these are made to exercise themselves in various ways. The mind is controlled according to suggestions, and the character of these suggestions governs the results. Mind travels through the nerve tubules as electricity traverses the electric wires, they being the media of communication; and when the wires are crossed confusion reigns. The same thing occurs when undue pressure prevails anywhere along the line, and as there are but two things causing the incoordination, to-wit: muscular contraction or blood stasis, the indications become apparent. Restore the nerve force or lessen the contracture, or both, if they exist. This sort of treatment persistently applied promises all that can be expected.

There are many things to consider in treating persons afflicted with mental troubles—from the hereditary origin to the exciting cause, whatever it may be. Judgment must be exercised in all of the cases presenting themselves for treatment. Some "sin" lies at the door as a cause. Transgression of a human or a divine law stands up before the victim, speaking in unmistakable tones that an enemy has done this. Evil habits must be corrected, the effects of them met and neutralized; present physical conditions corrected, and a new life started and lived; environments changed; the pressure taken off and kept off; new tissues permitted to take the place of the old, worn-out material; new thoughts, if possible, formed, maintained; dormant forces aroused, and new suggestions given to start new channels of thought and habits.

General Osteopathic treatment should be given as often as three times a week, and these should be as thorough as possible, arousing every nerve and liberating every pressure in the body.

Orificial Surgery and Suggestive Therapeutics occupy a prominent place in such cases, and are needed in a great many cases. Dementia may be arrested many times by changing the environments, the influences, and starting up a new life of hope;

and expectation and desire will strengthen the hope and make it the governing principle in due time, culminating in love, the promoter of all life and happiness.

GENERAL PARALYSIS.

SYNONYMS. Paralytic dementia; general paresis; general paralysis of the insane; dementia paralytica; paresis; paretic dementia.

DEFINITION. A subacute, or chronic, degenerative disease of the brain, sometimes involving the spinal cord; characterized by alterations in the intellectual and moral character, with the development of unsystematized ideas of self-importance, or delusions of grandeur, finally merging into dementia (preceded by either a mania or a melancholia), and the gradual development of tremor, slurring speech, pupillary changes, ataxia, trophic changes, and finally paresis.

CAUSES. General paralysis occurs chiefly between thirty and fifty-five years of age, and in the male more frequently than in the female. It usually affects the robust, middle-aged individual, rapidly destroying all intelligence and judgment, leaving him to exist, often for months, as a demented human automaton.

Predisposing Causes.—Hereditary; an ambitious overstraining for prominence, learning, or wealth; forced intellectual activity in those with imperfect or improper early training; cranial injuries; atheroma.

Exciting Causes.—Alcoholic and sexual excesses; syphilis; mental and physical overstrain; worry. "In many cases I think the middle-aged general paralytic is suffering for the sins of his youth." (Clouston.) "General paralysis is not a penalty of high cerebral development, but the expression of a discrepancy—an inadequacy of some brains to sustain the strain to which the race, as a whole, is subjected." (Spitzka.)

PATHOLOGICAL ANATOMY. A condensed description of the pathological basis of general paralysis is difficult. It may be described as a chronic diffuse cortical encephalitis. The microscopical changes in the cortex, according to Mendel, as quoted by Folsom, are as follows:

1. Increase of nuclei and new cell formation, some nuclei small, some large, and with such varying reactions to coloring agents as to suggest dissimilarity of origin. The stellate or

"spider" cells are increased in the upper layer of the cortex, where some may be normally found, and extend to lower layers, as is not the case in normal brains; they, too, may be several times the usual size and also push through the white substance to the ependyma of the ventricles. Proliferation of neuroglia or connective tissue, and in time sclerosis of the cortex, which involves the medullary substance also in a greater or less degree.

2. The larger blood vessels may or may not be atheromatous; in the capillaries there is an increase of nuclei in the walls, with thickening and hyaloid degeneration.

3. In the nerve cells, the ganglion cells, granular and fatty degeneration of protoplasm, sclerosis, atrophy.

4. Atrophy and final disappearance of the nerve fibers, not limited to the cortex and found in other brain diseases also—senile dementia and epilepsy, for instance.

5. Focal lesions of the most various kinds, and degenerative changes in the spinal cord, the several forms of sclerosis and myelitis.

The spinal cord undergoes atrophy, with gray degeneration in posterior and postero-median columns, and of posterior spinal nerve roots.

SYMPTOMS. For clinical convenience the disease is divided into three stages, prodromal, maniacal, rarely melancholic, and the stage of dementia, although there is seldom a marked division between each.

Prodromal Stage—May exist unrecognized for months or longer. It begins by an alteration in the habits and character of the individual; the patient has spells of irritability and obstinacy, which will not admit of contradiction or opposition; there is a general feeling of elation and *bien-etre*, or egotism shown by the exalted opinion of his own attainments and importance, and a great laudation of his family. He becomes boastful, untruthful, dishonest, and forgetful, neglecting engagements, business, self, and family. He frequently makes extravagant purchases, and may waste large sums of money before his condition of irresponsibility is recognized, or may unwittingly resort to dishonest means to obtain money to squander on new-made friends. In many instances the patient develops ideas of an enterprising character, and resorts to all forms of expedients, which, to his mind, are going to improve his or her family's station and worldly condition: he determines to change his occupation or business, or attempts to instruct the authorities in what he conceives

should be their duties. The moral lapses of paretics are most frequent during this stage, consisting of acts of theft, drunkenness, violent impulses, or indecent assaults, in individuals who have possessed a good moral character. They become profane and vulgar, and often resort to sexual excesses. Associated with any of the above symptoms may be any one or more of the following physical conditions: tremor of the muscles about the mouth, naso-labial folds, and of the tongue, causing a slight slur or hesitating speech; alterations in the pupils, or one pupil becoming somewhat larger than the other; attacks of vertigo, or epileptiform or apoplectiform seizures; the gastric, intestinal, hepatic, and nephritic secretions are disturbed, and there may be headache and insomnia. After a variable duration, continuing in a mild degree for many months, is ushered in the—

Second, or Maniacal Stage—Which is much the same as a severe attack of acute mania, plus the physical signs of paresis and the delusions or ideas of grandeur. The patient is excessively restless, boasting of his great wealth, intentions, prospects, and influence; one moment the most important of individuals, the next giving away thousands, and if doubt is expressed as to his ability to do so, making it millions and often billions, presenting houses and lands, titles and offices, with unstinted liberality. It is to be noted that these so-called delusions of the paretic are in reality conceptions, or an expansive delirium; for when contradicted the patient makes no effort to defend them; and they seem to be really assertions and reassertions, continuing until incoherency restrains the airy imagination. The patient is sleepless, noisy, destructive, with attacks of blind, uncalculating violence, resisting all who attempt to restrain or molest him; the violent impulses of paretics are similar to the furious excitement of the post-epileptic maniac. The physical signs are more pronounced, the characteristic hesitating and slurring speech increases, the pupillary changes become more marked, the tremor of the tongue and lips increasing, and spreading to the upper extremities, the gait ataxic, the patellar reflex increased, or, rarely, diminished, the sphincter of the bladder disordered, and there often occurs paralysis of the anal sphincters. During the progress of the second stage are developed cerebral crises,—syncope, petit or grand mal, apoplectiform attacks, or paralytic seizures. Few cases but show one or more of these conditions. There also occur myosis and loss of light reaction, and increased wrist and

elbow jerks. The maniacal stage is of shorter duration than any other, and is usually succeeded by the—

Stage of Dementia—The patient presenting all the evidences of failing mentality, with paralysis, trophic changes, as shown by the occurrence of bed-sores, cystitis, diarrhoea, and arthropathies, or Charcot's joints, the patient emaciating rapidly, death closing the scene within a few months.

Rarely the maniacal stage is preceded or replaced by a condition of melancholia with expansive hypochondriacal delusions. In a few instances a genuine lucid interval has followed either the prodromal or maniacal stage. The spinal form of general paresis is fairly frequent, in which symptoms of spinal sclerosis are added to the mental ataxic phenomena of the usual form.

"Of the many divisions of general paralysis into several clinical types, all of them naturally more or less arbitrary, I know no other so satisfactory as Meynert's eight." (Folsom.)

1. Simple progressive dementia, with the usual motor impairment which accompanies it, but, excepting hypochondriacal depression, not necessarily exhibiting other mental symptoms than dementia.

2. With the expansive delusions and the distinctive motor disturbances which appear simultaneously and are progressive, constituting the "classic" form of general paralysis. The mental state is usually of self-satisfaction and exultation, but there may be depression.

3. Of the same type as the last, but failing its steadily progressive character through arrest of the active process. The remissions, which seldom last so long as a year, raise hopes of recovery, but still manifest unmistakable impairment of the reasoning faculties. The psychic disturbances are much greater than can be accounted for by the atrophy of the brain alone.

4. Cases in which the characteristic exultation and grand delusions reach such an astounding height that manifest motor symptoms are looked for with confidence from day to day, and yet may not appear even for a year, any slight incoordination naturally being obscured by the general muscular disturbance. Meanwhile there may be such an improvement that the patient leaves the hospital for a while, once, rarely twice, on the responsibility of his family, but to return with marked motor, as well as mental signs.

5. A very rare form, with alternate symptoms of exaltation and depression of the type of circular insanity.

6. With early furious delirium, painful hallucinations, confusion and incoherence somewhat resembling acute delirium.

7. Progressive general paralysis, in which the characteristic indications appear secondary to other forms of insanity; for instance, after paranoia or melancholia, first described by Hoestermann.

8. The combined form, with sclerosis in the whole cerebro-spinal tract, the symptoms of tabes or spastic paralysis predominating, according as the posterior or lateral columns of the spinal cord are chiefly involved. The ascending type, in which the cord is first affected, is rare. Optic neuritis, ending in atrophy and paralysis, especially of the ocular muscles, may precede marked mental symptoms.

DIAGNOSIS. The development of the following symptoms removes all difficulties in diagnosis: mental—alteration in character, loss of memory, defective will power, changed moral sense, insomnia, violent impulses, melancholia or mania, unsystematized delusions of expansive character, with an exalted sense of well being, gradually ending in dementia; physical—hesitating, slurring speech, tremor of the lips, tongue, and upper extremities, pupillary changes, myosis, loss of light reaction, exaggerated wrist, elbow, and knee jerk, attacks of syncope, vertigo, epileptiform or apoplectiform seizures, ataxia, trophic changes, and finally paralysis.

Paralytic insanity, or organic dementia, is not the same condition as general paralysis. It is the form of mental failure succeeding to gross brain lesions, such as apoplexy, tumors, softening, trauma, and sclerosis.

PROGNOSIS. Unfavorable. Remissions very, very rarely occur.

THE TREATMENT.

We do not desire to have the reader to understand that when the condition resulting in tissue, structural changes, has been reached, that we recommend any treatment, but before the cord undergoes atrophy we claim that Osteopathic treatment offers better results than can be derived from any other source known, for the reason that blood (arterial blood to the parts) is the only tissue builder and preserver, Nature's own remedy.

The careful study of the nature of the malady and the stage, effects and condition of the patient at the time of examination should be made, and then an intelligent application of the principles of this science usually results in great good, in that it stays

its progress in many cases and cures others not gone beyond human limits—structural changes. As there are all grades of paralysis, from its simplest to its gravest form, the Osteopath will be extremely guarded in his prognosis as well as in his promises to the patient regarding results. Many cases thought to be incurable are cured, and others which seem easy, not serious seemingly, are not benefited by treatment. In all cases of paralysis the structural conditions determine the consequences. Wherever arterial blood circulates and nerve pressure can be removed, the case may be regarded as favorable.

The general treatment should begin at the neck. Extension, rotation, careful, deep, persistent, long manipulations of the muscles of the neck will be found to be necessary to relax them, and it sometimes, in some cases, requires months to accomplish this. In all cases of paralysis, of every form, we find rigidity of the muscular fibers to exist, especially those of the neck. The same condition extends all the way down the spine, in the dorsal region, and a persistent course of manipulations are necessary to relieve the rigidity. As the neck muscles yield, the others do in proportion, and amelioration shows itself. The manipulations require much patience on the part of the operator, for the rigidity is often intensified by attempts to manipulate, and greatly interfere with movements. A coaxing of the muscles, as well as the patient, to relax, constitutes a large part of the first few attempts to treat a patient. We speak especially of cases where the rigor is predominant, after the paralytic stage is partly changed. The incoordination is characteristic. Rigidity prevails in some cases, relaxation in others. Finally general paralysis, and death ends the scene. Early treatment should be instituted to promise benefit. Osteopathy promises more than any other treatment, if begun early and persistently applied, and yet early application is important. Do not promise to raise the dead with Osteopathy.

PLATE LVIII.—Manipulation for Goiter.