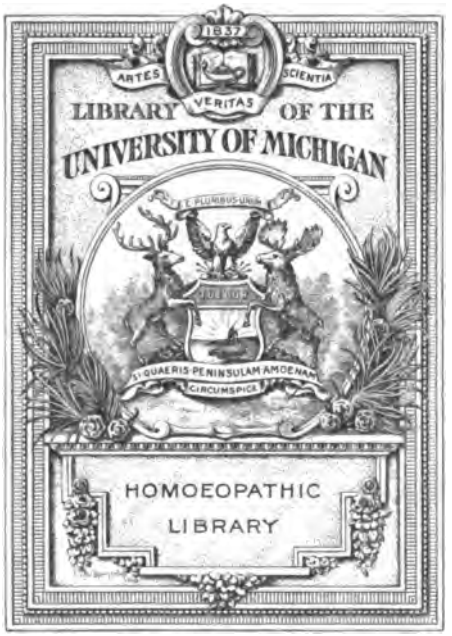
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PRACTICE of MEDICINE

By

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TO

WIFE, DAUGHTER, SON

PREFACE

This book has been many years in the making. For the past three years its preparation has occupied practically all of my spare time. It has been my aim to produce a book that omitted none of the essentials of practice, and yet one that was concise in statement.

The arrangement of the book, as a whole, gave me considerable concern. After various plans had been worked out the one adopted seemed to me the best. Certain chapters could appear in other sections than where they have been put, perhaps, but I have placed them in what seemed to me the most logical order.

Syphilis and Tuberculosis have been treated as general infections in the section on Infectious Diseases, and have been referred to in other sections treating of disease of special organs. Tonsilitis has been put in the section on Infectious Diseases, where, I believe, it belongs. Erysipelas and Septic Poisoning also appear in that section.

In the section on Poisonings and Intoxications there are a number of more or less common conditions often met with in hospital practice not usually described in books on practice.

There is also a section on Skin Diseases, a section not ordinarily found in books on practice. Every physician should be able to diagnose the more common skin lesions and treat them. Next to the eye and ear diseases, probably, skin diseases cause more mental perturbation on the part of the general practitioner than any others. Yet the names of the lesions are often more formidable than the lesions themselves.

There is a section on Mental Diseases and Psychoses. This includes descriptions of some of the more common forms of insanity, usually omitted in books on practice. It also includes conditions like Hysteria and Epilepsy, usually placed under Nervous Diseases. I have placed Heat Affections in this section

because the mentality is disarranged temporarily, and may be for a considerable time after sunstroke, or heat prostration.

The individual chapters have been arranged schematically for purposes of clearness and ready reference. Brief historical notes appear so that anyone interested may look up the original authorities if he so wishes. Mention is also often made of current literature.

It is impossible for anyone to write an entirely, or even a largely, original book on the practice of medicine. But it is possible at the bedside and in the autopsy room to verify and compare and learn much from experience. I have many thousand hospital case records with my own notes, and for more than twenty years I have recorded the symptoms and treatment of every case treated in private practice either in my office or at the bedside. I have utilized all of this material to the best of my ability in the present volume. In citing personal experiences, therefore, I am quoting from notes written at the time and not from memory. Such statements are statements of facts as I have seen them. If errors appear they are errors of judgment. If my experience has not agreed with others I have not hesitated to say so.

Under Treatment I have referred to the generally accepted old school methods first. Finally, I have added briefly the most frequently indicated homœopathic remedies, sometimes with and sometimes without indications. Only occasionally have I mentioned the potencies used, and then where some particular potency has specially impressed me. As a rule, I might add that unless otherwise stated my preference is for the third to the sixth. All medicines made from tinctures I use in dilutions. I believe them to be more satisfactory. Medicines from the minerals are the only ones I use in triturates, and some of those in the higher potencies I use in liquid form.

It is impossible to be a good homœopathic prescriber without a knowledge of the homœopathic materia medica. In a book on practice it is impossible to give indications for all the drugs that might be used. But I have endeavored to, at least, give efficient guides to the reader.

My main object has been to emphasize the clinical aspects of disease, the subjective and objective symptoms, and then to point the way to correct them.

I wish to thank the many internes who have worked with me at various times in the Metropolitan and in the Flower Hospitals. I am indebted to them for much valuable assistance. I am also under obligation to many nurses in hospital and private practice for carefully recorded observations.

WALTER SANDS MILLS.

New York City.
March, 1915.

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SECTION I.

Infectious Diseases.

SEPTIC POISONING.

(Greek σηπω, to putrefy.)

Introductory Note: Sepsis is one of the most important subjects that the medical man has to deal with and it is too often overlooked. Aside from its occurrence following injuries, it frequently complicates or follows many of the acute infectious diseases. Symptoms of sepsis are also often the first evidence we have of the formation of pus within the body, whether it be appendicitis or deep-seated abscess elsewhere, or that terrible condition, malignant endocarditis. It is essential, therefore, that considerable space be devoted to a consideration of it.

Forms: There are three forms of septic poisoning that have been differentiated experimentally. Clinically, they may resemble each other so closely, or one or more forms may be associated in such a way, as to be indistinguishable. For this reason they are considered under one heading.

Definitions: 1.—*Sapremia* (σαπρος, putrid; αιμα, blood) is a febrile condition due to the introduction into the blood of the chemical products of putrefaction.

2.—*Septicemia* is a febrile condition due to the introduction into the blood of the bacteria themselves.

3.—*Pyemia* (πυον, pus) is septicemia with the formation of secondary abscesses in the various tissues and organs of the body.

Historical Note: Septic conditions, especially puerperal fever and wound fever, have been recognized from the beginning of medical history. In 1843 Oliver Wendell Holmes wrote a paper

on "The Contagiousness of Puerperal Fever," which met with violent opposition. In 1847 Semmelweiss, of Vienna, announced his belief that the great mortality from puerperal fever in the Vienna Lying-in-Hospital was due to the fact that students went direct from the autopsy table to the delivery-room, thus infecting the women. Panum, in 1855, was the first, systematically, to inject putrefying substances into animals for experimental purposes. Koch, in 1876, was the first to detect pus organisms in the blood. Within the past few years various disease germs other than the pus organisms have also been found to cause general sepsis. In short, it is only since the development of the science of bacteriology, a scant half century, that the exact nature of septic conditions has come to be understood. Most of the exact knowledge on the subject has been gained during the last two or three decades.

Etiology: The cause of septic poisoning is the entrance, either of pathogenic bacteria or of their products, directly into the circulation, probably through some break in the skin or mucous membrane. In infected wounds or in puerperal fever the source of the poison is obvious. If all septic cases were of this character the subject would not be considered in a medical treatise. Osler states that of the septic cases that reach the post-mortem room at Johns Hopkins those from the medical wards outnumber those from the surgical wards twenty to one. It is these cases, the causes of which are not obvious, indeed, may not be discoverable, that come to the physician. It is probable that in many of them infection takes place through the respiratory or digestive tracts, possibly from ill-kept teeth.

In these days of rigid asepsis cases of sepsis following surgical operations are rare. The rise in temperature sometimes immediately following an operation is said to be a mild sapremia.

Chronic suppuration in any part of the body, as middle ear disease, may be the origin of a general septic condition. The worst case I ever saw originated in a neglected rectal fistula.

No age and neither sex is exempt.

Certain cases exhibiting symptoms of the different forms of septic poisoning occasionally follow the various infectious dis-

eases as sequelæ. One of the most fatal is systemic infection by the gonococcus, which will be spoken of more fully in another chapter. These end results are usually due to the toxemia of the original poison, sometimes to the pathogenic bacteria themselves. In other cases the original disease leaves an opening for the entrance of pus organisms.

Bacteriology: The bacteria most frequently found in true sepsis are the streptococci and staphylococci. Roger gives a list of fifteen other organisms that are sometimes found. In cases following the infectious diseases the bacteria causing the original condition may be found.

Morbid Anatomy and Pathology: Rigor mortis is slight. Putrefaction begins very early. The blood is found to be dark and disintegrated. The spleen, as a rule, is enlarged and soft. Parenchymatous changes are frequently found in the kidneys, spleen and liver. In other cases these changes may be absent. I have found a fatty heart and large pale kidneys and liver. In severe cases of septicemia there may be numerous ecchymoses found in the various organs and tissues. If so it may be possible to demonstrate that the pus organisms are generally distributed throughout the body.

The initial lesion may or may not be found. When not found after careful search, the respiratory or the digestive tract is probably the seat of an undiscovered lesion.

If the case has progressed to one of pyemia, then emboli and thrombi, with degeneration of the surrounding tissue, may be found. One or more abscesses are present. The lungs are the most frequent seat of pyemic abscesses; then the kidneys, spleen and liver in that order.

Symptoms: Septic conditions usually begin with a chill or chills. There is a more or less sudden rise of temperature to 100° F. or more and an increased pulse rate. There may be headache, less often nausea. In other cases again the onset may be very indefinite, simply a feeling of malaise and feverishness.

In *sapremia* the symptoms are the result of the introduction of the chemical products of putrefaction into the blood. In case of wound infection this starts with a localized tenderness, swelling

and redness in the neighborhood of the wound. If unchecked the trouble extends along the line of the lymphatics and veins. In such cases the point of infection is obvious, and prompt surgical treatment will stop the symptoms by removing the source of supply of the poison.

If the pathogenic bacteria themselves have actually entered the circulation the case is one of *septicemia.* This form may begin in an indefinite way or it may begin with a chill or chills. The temperature rises and remains comparatively high with slight remissions. The pulse gradually becomes rapid and weak. This is one of the most important of symptoms, the pulse being the surest single index of the condition of the patient. Sweating is a common symptom, not continuous, but as night sweats or at irregular times throughout the twenty-four hours. Rheumatic pains in various joints are sometimes found together with a petechial or purpuric eruption. The spleen is usually enlarged. Headache of a dull character is quite common. It is not often severe, but when it occurs is quite constant. In severe cases the patient may become apathetic, even delirious. Sometimes the mind remains clear to the end. There is loss of appetite, there may be nausea and vomiting. If the disease remains unchecked the patient goes into the so-called "typhoid condition." The patient wastes away; the facial expression becomes markedly changed, the eyes sunken, cheeks drawn, and of a bad dusky or ashen hue. The patient dies in a few days or weeks.

In *pyemia* abscesses, one or more, develop in various parts of the body. In this form of septic poisoning there are usually marked variations of temperature, with actual chill, fever and sweat simulating true malarial intermittent fever. In other cases the symptoms will not be like intermittent fever, but will closely simulate typhoid. For example, a case in my service at the Flower Hospital during the summer of 1913. The patient entered with a history of having been out of sorts for a few days. As the case developed the temperature curve ran like that of typhoid fever. The pulse remained about 80 to 90, not unlike typhoid, but very unusual for sepsis. The patient had night sweats and a cough. Tuberculosis was ruled out by lack of

physical signs, and because the pulse was too slow and the temperature too even. Typhoid fever was ruled out because repeated Widal tests were negative. The patient complained of pain in the left side below the ribs, which at times was very severe. Finally we were able to demonstrate an abscess at that point, and the diagnosis was cleared up.

In obscure cases where pus is eventually found in the abdomen, as in the above case, sometimes in appendicitis, I have noted a cough frequently enough to put it down as a symptom, provided examination of the lungs fails to reveal any reason for cough there. It is a true reflex cough apparently.

In some cases there will be a purpuric eruption, or a few petechial spots, or an indolent papular eruption on the legs or body.

The *blood* usually shows sepsis, and should always be examined in suspected cases. The hemoglobin is always diminished, and in rare cases may be as little as 20 per cent. The red cells are diminished in number, the whites increased. Ewing says the white cells may be diminished just before death. There is usually an increase in the percentage of polynuclear neutrophile leucocytes when pus is present. Pathogenic organisms may or may not be found.

Complications and Sequelæ: Septic conditions are themselves end results and do not tend to leave sequelæ, except where abscesses occur resulting in destruction of tissue. Sepsis may complicate or follow wounds or the acute infections. Very rarely it is a primary condition.

Diagnosis: Clinically, it is frequently difficult to differentiate between the various septic conditions. In cases of wounds the onset of pain, with local heat, redness and extension of the pain or tenderness along the line of the lymphatics, should always excite suspicion. Following wounds, child birth or cases of infectious diseases, the onset of chills, headache, nausea, increase in frequency or diminution in tension of the pulse, with rise of temperature and perhaps sweats, all are indications of septic involvement. Occasionally there may be development of abscesses without rise in temperature. Or the temperature may suddenly rise, then gradually subside notwithstanding the formation of pus.

A diagnosis of sepsis is usually easy following wounds or child birth. Even then it must not be forgotten that other conditions may cause confusion. One patient under my care, after an operation for appendicitis, suddenly had a rise of temperature to above 102° F. The bowels appeared to be functionating properly, and the wound seemed to be all right. A high enema relieved the bowel of an accumulation of fecal matter, and the temperature promptly dropped to normal.

Another case that caused much anxiety was that of a lying-in woman in my service at the Metropolitan Hospital some years ago. She developed a temperature the day after confinement which we naturally thought to be septic. She was treated locally and medicinally without effect on the temperature. Finally, after two or three days, she developed facial erysipelas which ran a normal course. The fever disappeared with the subsidence of that disease.

Pyemia is sometimes confounded with malaria. Lying-in women who have ever had malaria in the past will frequently have a malarial paroxysm during the puerperal period. This possibility must always be borne in mind, as it will frequently save much anxiety. In doubtful cases examination of the blood should be made for the malarial organism. Quinine will clear up malaria, it has no effect on true sepsis.

Septic poisoning following the infectious diseases can usually be diagnosed by the severity of the symptoms, for they are always very grave.

Typhoid fever can be differentiated by its course, and by the presence of the Widal reaction. The spleen may be enlarged in both sepsis and in typhoid.

Tuberculosis can be differentiated by the physical signs found in the chest, and by the presence of tubercle bacilli in the sputum.

Malignant endocarditis is a true septic condition. In many cases careful physical examination will reveal endocardial valvular changes that will give the clue to the diagnosis.

Acute articular rheumatism may be confounded with sepsis. Perhaps it would be more correct to say that some septic cases that present joint involvement may be mistaken for rheumatism

for a time. Acute articular rheumatism runs a more definite course, and the swellings of the joints come and go, but are more or less symmetrical.

In many cases there will be ample evidence in the general condition of the patient of pus somewhere, but it will be only after repeated careful physical examinations of the patient that the exact location of the pus will be found.

Prognosis: In sapremia the prognosis is good. The symptoms usually promptly subside under appropriate surgical treatment.

In septicemia and in sepsis following the acute infections, the prognosis is not good. When the condition has progressed far enough for the micro-organisms to enter the blood the case is practically hopeless. Death may occur in a few hours, or at most, a few days. The worst case I ever treated lived three weeks. In very rare cases a septic condition may become chronic. I have seen one that lasted nine months and then made a complete recovery.

In pyemia if the abscess or abscesses are so situated that they can be treated surgically the patient may recover. Otherwise the prognosis is bad.

Treatment: In the care of wounds and in child birth rigid asepsis, properly carried out, will prevent septic poisoning. Proper care in the treatment of the acute infectious diseases, especially in the care of the mouth, will do much to prevent septic complications following them. Sepsis resulting from localized diseases will also require surgical treatment when the pus is found.

When sepsis has taken place from wound infection or from puerperal infection local surgical treatment is indicated.

In cases where the cause is obscure or altogether unknown the patient should be put to bed and watched for developments. If pus appears it must be removed if possible.

A liquid diet of milk, koumiss, buttermilk, egg-nog, meat extracts and broths should be given. Lemonade or water may be used ad libitum.

Alcohol may be necessary. A patient suffering from septic poisoning can sometimes tolerate a great quantity. A definite

amount should be given every two to four hours when used at all, and it should be pushed to the limit. Whiskey or brandy diluted one-half with water is the best way to use alcohol.

Antistreptococcic serum has been credited with some cures.

Of homœopathic remedies probably ***arsenicum album*** ranks first. Arsenic has profound blood changes and adynamia in its symptomatology. It is good for great restlessness with excessive thirst for small quantities of water at frequent intervals. I saw one desperate case with multiple abscesses cured with this remedy while an interne at the Brooklyn Homœopathic Hospital. The patient was a nurse who had accidentally infected herself. She was in bed seven months.

Arnica is especially useful after traumatisms or after child birth. Cowperthwaite believes that *arnica* given in such cases tends to prevent the development of sepsis.

Hepar sulphuris calcareum may be of service used in the higher potencies. I have used it low when I was sure of a septic condition, but was unable to locate the pus, in order to bring the case to a focus.

Carbolic acid is recommended when the discharges are foul. Helmuth, Sr., used the nascent phenic acid night and morning by hypodermatic injection in desperate cases with good results.

Lachesis is useful in cases with profound blood changes, with purpuric spots and great prostration. The patient is worse after sleeping. *Lachesis* and *carbolic acid* are not compatible.

Baptisia is indicated when the patient is in a low typhoid condition, with great muscular soreness, bad smelling breath, and low delirium. Baptisia must be used low.

Crotalus horridus is useful in cases associated with a hemorrhagic tendency.

Muriatic acid is indicated in cases with great prostration, dry tongue, sordes on teeth, intermittent pulse.

Echinacea, a remedy introduced during the last few years, is a very serviceable remedy in septic conditions. It cured one remarkable case of puerperal sepsis that I was permitted to see in the service of Dr. Hamlin at the Flower Hospital. This remedy gives the best results used in the tincture. It may be used locally also.

Many other remedies may be indicated, as *apis, silicea, elaps, mercury,* and so on. Each case must be studied by itself.

ERYSIPELAS.

(Greek ερυθρος, red; πελλα, skin.)

Synonym: St. Anthony's Fire.

Definition: Erysipelas is an acute systemic febrile disease of sudden onset, characterized by a localized and sharply outlined inflammation of the skin that may extend over a considerable portion of the body. It is most often seen on the face, though it may attack any part.

Historical Note: The disease was written of by Hippocrates under the Greek name of ερυσιπελας. It has been well known ever since.

Etiology: In 1881 Fehleisen described a streptococcus as the cause of erysipelas. This has been found to be so like the streptococcus pyogenes that most observers now classify the two as identical. The best place to find the organism is just at the margin of the affected area.

Both sexes and all ages are susceptible. Cheyne, in Allbutt's System of Medicine, says women are most often affected; whereas Anders, who collected over 2,000 cases, says men are most often affected. His report is based largely on hospital statistics. In private practice most of my erysipelas patients have been women, the majority of them maid servants. At the Metropolitan Hospital the proportion is about three men to one woman, the same as the general ratio of all the patients. My youngest patient was eight months, my oldest 61 years old.

The majority of cases present themselves in the winter and spring. Thus, in the two years ending June 1, 1904, there were admitted to the erysipelas wards of the Metropolitan Hospital 627 patients. The admissions by months were: January, 73; February, 62; March, 70; April, 106; May, 59; June, 44; July, 47; August, 33; September, 27; October, 30; November, 31; December, 45.

It is supposed that all cases of erysipelas start from some abra-

sion of the skin or mucous membrane. This is probably so, but in the majority of cases it is impossible to prove it.

Surgical patients and lying-in women are especially susceptible because the abrasion is there. Physicians in attendance on erysipelas patients should not treat surgical or confinement cases because of danger of infection. Erysipelas is considered to be more or less contagious. Personally, I have never met with a case where I could say positively that it had developed from some preceding one. I have never known a nurse or doctor to become infected. The 627 cases enumerated above were treated by me while the erysipelas ward was under my supervision.

According to Rotch infants under six months are especially susceptible. In them erysipelas frequently starts at the genitals, or, in early infancy, at the navel. When it attacks the face of the infant it is very fatal. On the other hand, in adults the face is most often affected, and is very rarely fatal.

I have had one lying-in woman, at the Metropolitan Hospital, ill with erysipelas, and two others, private patients, who were nursing young infants at the breast. All three nursed their babies during the attack without apparent injury to mother or child. In such cases, however, the physician must be prepared at any moment to remove the child from the breast.

An attack is said to confer immunity for a time, but I have met with a number of patients who had a relapse or a recurrence immediately following apparent recovery. Some patients seem to have a special susceptibility to erysipelas, and have an attack every year. I have one private patient whom I have treated for three attacks in seven years. One hospital patient said he had had an attack every year for sixteen years.

Morbid Anatomy and Pathology: The redness of the skin disappears after death. If there has been much infiltration a considerable quantity of serous fluid may be found in the tissues. This may be clear, or it may be turbid, due to pus organisms. Swelling of the spleen and parenchymatous changes in the kidneys and liver may also be found. There are no characteristic lesions.

Symptoms: The period of incubation is set down at three to

seven days. In Fehleisen's experiments on patients suffering from incurable tumors, symptoms of erysipelas appeared in from fifteen to sixty-one hours after inoculation.

The disease usually begins with a chill or chills followed by a rapid rise of temperature; at the same time the skin at some point —in adults usually near the nose—becomes red, hot and swollen. The inflamed area gradually extends, the border always being sharply outlined against the normal skin. In mild cases the temperature may not go much above 100° F. There is slight coating of the tongue and loss of appetite. The infiltration in the skin extends until at the end of twenty-four hours the eye is nearly or quite closed, and the features quite unrecognizable. Then all the symptoms subside, and by the end of the third day the patient is well, or nearly so.

In other cases the temperature rises much higher—it may be as high as 106° F., while 105° F. is not at all uncommon. There is complete anorexia. The patient may be delirious, or may go into a typhoid condition. Albumin appears in the urine. The temperature tends to remain up as long as the skin symptoms progress, without marked remissions. Finally, when the skin symptoms cease advancing, rarely before, the temperature falls either by lysis or crisis. There is no typical eryspielas fever curve. As in other infectious diseases, the heart should be closely watched for evidence of endocarditis.

The extent of the skin lesions varies greatly. In the typical case the swelling begins at or near one side of the nose. The skin becomes red, shiny, and tense, from infiltration. By the end of twenty-four to forty-eight hours, the whole side of the face is swollen, the eye is closed, and the features unrecognizable. Very often, at the end of twenty-four hours or later, the disease will appear on the other side of the face and go through the same process, while the swelling is gradually disappearing on the original side. In some cases blebs are formed over the affected area, and in still fewer suppuration takes place. These latter are due to mixed infection.

The disease may begin at any point and extend to any part of the body. In one girl of 21 at the Metropolitan the disease ex-

tended down the front of the body to the waist line, and down the back as far as the buttocks. In another girl about the same age, in private practice, the disease started at the genitals, extended over the buttocks, up the body, front and back, and ended at the face. In another patient, a girl of 7, the eruption started at the site of an abrasion on the left knee, extended to the foot and up the thigh and body to the waist line, crossed over and traveled down the other side and leg to the foot.

It takes from twenty-four to forty-eight hours for the skin symptoms to reach their height at any given point, and about the same length of time for them to subside. When the disease spreads for a number of days the earlier lesions are disappearing as the later ones develop. Thus we may have a case where the site of the original trouble has returned to its normal condition, while the disease is still spreading elsewhere.

When the skin symptoms are at their height the tension causes considerable discomfort, and there is itching or burning. Disappearance of the infiltration is usually followed by desquamation. This may take place in fine scales, or, as in the child mentioned above, great thick pieces of epidermis may peel off. The new skin is frequently of finer texture than the old.

Examination of the blood in erysipelas usually, not always, shows a leucocytosis varying with the severity of the other symptoms. If suppuration takes place, leucocytosis is, of course, marked.

Sometimes the infiltration and inflammation extend to the mucous membranes. When this takes place the condition is always serious.

Complications and Sequelæ: Erysipelas, like the other acute infections, is sometimes associated with pneumonia. The onset of the latter would be indicated by increased frequency of respiration.

In alcoholics delirium tremens may supervene. Jarrot also mentions arthritis as a complication.

The most important sequelæ are infective endocarditis and nephritis. Neither happens often in cases under homœopathic treatment. According to Osler erysipelas is the most frequent cause of infective endocarditis. I have not found it so.

Meningitis may also occasionally follow erysipelas. Sometimes otitis media is a sequel.

In old people, and in the poorly nourished, extension of the inflammation to the deeper structures may occur causing cellulitis.

Diagnosis: The diagnosis of erysipelas is usually easy. If the disease starts at some place other than the face it may resemble acute dermatitis in appearance. The latter, however, is not ushered in by chills and high temperature, and the skin does not present the same advancing sharp outline.

Erysipelas with development of bullæ might under rare circumstances resemble some forms of eczema, but erysipelas is a self-limited disease, and eczema tends to be chronic.

Prognosis: The prognosis in uncomplicated erysipelas is good. Goodno says he has never seen a fatal case in his own practice, and I can say the same of mine. Of the 627 cases in my service at the Metropolitan Hospital 17 died. All the deaths were cases with complications, mostly alcoholics. The vast majority of the 627 were from the lowest walks of life.

Treatment: The patient should be isolated and put to bed. If a severe case liquid diet, preferably milk, is indicated.

Local treatment of any kind is useless. The ichthyol preparations of various kinds so much in vogue at present are not only useless, but nasty. Applications of ichthyol and collodion, which are frequently put on patients at Bellevue who are later sent to the Metropolitan, I believe to be harmful. The mixture forms a varnish-like covering that nothing but time will remove. It is liable to crack, and when it does abrasions of the skin result that are difficult to heal. If there is much burning or itching clean linen cloths wrung out in cold water are soothing and all that is necessary. A weak solution of bichloride of mercury, 1 to 5,000, may be substituted if desired.

The iron preparations used by the old school are of limited or no value. The indicated homœopathic remedy is of the utmost value.

First on the list I wish to place *graphites*. This is recommended by Goodno, who uses it when no other remedy is urgently called for. I can heartily endorse that recommendation. I use

the sixth centesimal potency, one tablet every one or two hours, as needed. I have seen many cases clear up under it, some of them very severe ones, in three or four days. I began its use while an interne at the old Ward's Island Homœopathic Hospital in 1889.

The next most useful remedy is *belladonna*. This is indicated if the fever is very high, the skin very red, tense and swollen, the pulse full and rapid, and the eyes congested. When these acute symptoms subside *graphites* is a good remedy to follow the *belladonna*.

If the swelling is the main symptom and the skin pale and puffy looking *apis mellifica* is indicated.

Rhus toxicodendron is the remedy if blebs are plentiful.

These few drugs, in the order named, are the ones most often indicated. Many other drugs may be called for if their special indications are present.

In cases where abscesses develop *hepar sulphuris calcareum* is serviceable, although even in these cases *graphites* may be the best drug.

TONSILITIS.

(Latin, *Tonsilla,* tonsil; *itis,* inflammation of.)

Synonym: Amygdalitis.

Definition: Tonsilitis or amygdalitis is an acute infectious systemic disease, characterized, locally, by an inflammation of the tonsil or tonsils. The inflammation may be limited to the mucous membrane covering the tonsil, or it may extend to the parenchyma of the gland. It begins as an acute catarrhal inflammation of the covering of the tonsil, *acute catarrhal tonsilitis.* If the inflammation follows the continuation of the pharyngeal mucous membrane into the follicles of the tonsil it is called *follicular tonsilitis.* When the parenchyma of the gland becomes affected and suppuration intervenes the disease is known as *acute suppurative tonsilitis* or *quinsy.*

At the onset of the disease there is no means of telling which form it will take. Frequently we have a mixture of the different varieties so that clinical differentiation is difficult. It simplifies matters to look on these varieties as successive steps in the same disease.

Historical Note: Tonsilitis has been recognized for a long time as being more or less communicable, although I believe I was the first to classify it with the acute infectious diseases. In a paper on "Tonsilitis" read before the Homœopathic Medical Society of the State of New York in September, 1898, published in the Transactions and also in the *Homœopathic Eye, Ear and Throat Journal* for April, 1899, I said:

"I believe the proper place for a consideration of idiopathic tonsilitis to be in the list of acute infectious diseases. I have frequently seen tonsilitis pass from one member of a household to another. If a number of a group of persons are affected we will find the catarrhal, the follicular, and the suppurative forms indifferently originating from the same source or from each other."

Again, as a protest against the attitude of the Health Department of the City of New York when I was Medical Inspector in not excluding tonsilitis from the schools, I published a paper in the *Medical News* for January 24, 1903, entitled "Tonsilitis Classified as an Infectious Disease."

The first text-book in which Tonsilitis has been so classified is the Eighth Edition of Osler published in 1913. In all his previous editions and in all other books at my disposal Tonsilitis is classified with the Diseases of the Digestive Organs.

In recent years Jonathan Wright and others of this country, and Cornil, Woodhead and others of Europe, have shown that the healthy tonsil is a protection against the invasion of various pathogenic bacteria, and that diseased tonsils serve as the port of entry for such bacteria. It is therefore of importance that the tonsil be kept in good condition.

Etiology: Many things are assigned as exciting causes of tonsilitis. Chief among them is cold or exposure. Many young people seem to have a diathetic tendency to tonsilitis; that is, very slight causes will develop the disease and they will have repeated attacks. This tendency I believe I have seen eradicated by homœopathic medication. This does not oppose the idea of the infectious nature of tonsilitis; we cannot have a crop until the seed is planted in proper soil. Homœopathic medication changes the soil in this case.

Excessive venery is said to act as an exciting cause, supposedly, by reflex action. It may be so sometimes, but why is it not more probable that a large proportion of such cases are really due to carelessness in getting overheated and then chilled, or to transmission by osculation?

Tonsilitis often occurs in those subject to rheumatic fever of which it is sometimes the precursor. I had such a case in the spring of 1913, and again in 1914.

Tonsilitis is quite common during epidemics of diphtheria and of scarlet fever. Some of these cases may be mild forms of the epidemic disease. It is not at all unusual for tonsilitis to attack persons caring for patients ill with scarlet fever, persons who have at some time in the past had scarlet fever themselves.

Tonsilitis may also be found during epidemics of influenza.

Tonsilitis may be caused by the inhalation of noxious gases, it may be caused by traumatism, it may be caused by digestive disturbances.

During the last few years a number of milk-borne epidemics of tonsilitis have been reported, notably one in Boston in 1911. Dr. Halsey J. Ball, of Cortland, New York, reported an epidemic of 609 cases in 1913. He read his paper before the New York State Homœopathic Medical Society.

Repeated attacks of acute tonsilitis may cause a chronic enlargement of the tonsils. This enlargement in turn conduces to renewed attacks of acute tonsilitis.

Sex has no influence, my cases were about equally divided between the sexes.

Age seems to be an important factor. Twenty per cent. were five years or less, forty per cent. were between ten and fifteen. The youngest patient was fifteen months old, and I have seen three over thirty-five years of age.

Bacteriology: There seems to be no specific tonsilitis germ, at least none is known as yet. The staphylococci, streptococci, and pneumococci have been found.

Morbid Anatomy and Pathology: In the acute catarrhal form of tonsilitis there is a catarrhal condition of the mucous membrane covering the tonsil. In the follicular form this becomes

more severe and a cheesy secretion forms in the lacunæ. In the suppurative form the parenchyma becomes involved and breaks down.

Symptoms: In my recorded cases of tonsilitis, where I have been able to obtain a history of exposure, I find that the disease presents a definite period of incubation varying from one to four weeks. The great majority of cases appear within fourteen days. One case developed four weeks after the first exposure, but as this patient was in attendance on another case for an entire week infection may have taken place at any time during that period.

Tonsilitis may be preceded for a few hours, perhaps for a day or two, by a feeling of malaise. As a rule, however, sore throat is the first symptom complained of. This is usually accompanied by more or less headache sometimes by backache, and a general tired and aching feeling all over. The face is flushed and the eyes congested or shining. All of these symptoms develop rapidly, and the patient feels prostrated out of all proportion to the local manifestations.

Fever accompanies the onset of the other symptoms. There may be merely an ill-defined chilliness, or there may be distinct chills. The temperature ranges from 101° F. to 105° F., usually reaching its highest point on the evening of the third day. One of my patients had three distinct chills at the beginning, with intense headache, backache, and a very painful throat. At the time of highest temperature in this case, 104 3/5° F., she complained of feeling cold.

Examination of the mouth shows a coated tongue. It may be very dry, there may be an excessive secretion of mucus. One or both tonsils will be very red and more or less swollen. This redness may extend to the soft palate and uvula; later the parts may become bluish. Frequently a yellowish exudate shows itself on the tonsils.

If the inflammation extends to the lining of the tonsilar follicles the disease is known as follicular tonsilitis. If it goes still deeper, into the parenchyma of the gland and causes suppuration, it becomes quinsy. In other words, simple catarrhal tonsilitis, follicular tonsilitis, and quinsy are successive stages of

the same disease. Fortunately proper treatment will control its advance and prevent the development of quinsy in the vast majority of cases.

Usually the external throat is tender. Sometimes the small lymphatic glands under the chin are enlarged and tender. When the swelling of the tonsils is very great the patient can open the mouth to but a limited extent. There is pain on swallowing and sometimes on talking. If the inflammation extends to the Eustachian tubes there may be earache. Much enlargement of the tonsils changes the character of the voice, the patient "talks through his nose." Sometimes he may be hoarse.

Complications and Sequelæ: Frequent attacks of acute tonsilitis sometimes cause chronic enlargement of the tonsils.

The acute condition may go on to suppuration and become quinsy.

As in the acute infections, earache and middle ear disease may develop.

Of recent years tonsilitis has come to be considered a rheumatic condition and acute articular rheumatism has been found to follow it quite often.

Diagnosis: As a rule the diagnosis of tonsilitis is easy. Occasionally it is a symptom of grip, nearly always it is a symptom of scarlet fever. The prevalence of either grip or scarlet fever should cause the physician to be on his guard. The unfolding of the case will make the diagnosis clear. Sometimes follicular tonsilitis will simulate diphtheria so closely that a diagnosis will be in doubt. The secretion of tonsilitis can be removed without injuring the mucous membrane; the secretion of diphtheria, on the contrary, leaves a raw bleeding surface. As a final test a bacteriological examination can be made. This should always be done when in doubt.

Prognosis: The prognosis of tonsilitis is good. In the suppurative form, quinsy, the abscess has occasionally ruptured into the larynx, causing death. Sudden edema of the glottis complicating tonsilitis has also caused death. Fortunately, both of these accidents are extremely rare.

Treatment: The first thing to do in a case of tonsilitis is to

isolate the patient. This is particularly necessary where there are children in the house. The patient should be put to bed. The diet should be liquid and is best restricted to milk. The severe pain on swallowing may be relieved by the method introduced by Grewocks; that is, the patient pulls down on the ear at the moment of deglutition. Cracked ice will relieve the thirst and will often relieve the pain.

Local treatment has seemed to me of little value. Hot or cold compresses to the external throat may be of some help. As a gargle I prefer a cheap claret wine, the cheaper the claret the better the gargle. The tannic acid in the wine acts as an astringent, and the patient usually feels much better after its use. It may be used clear, or diluted with water up to one part in three. The patient may gargle every two hours or oftener. Alcohol and water may be used as a gargle.

Unless I see some special indication for other remedies I always start a case of tonsilitis on *aconite* and *phytolacca* in alternation; I use the third dilution ten drops in half a glass of water and give in teaspoonful doses every two hours to half hour, according to the severity of the symptoms. I have tried each remedy alone, but in the majority of cases I believe the two in alternation produce better results.

If the disease assumes the follicular or suppurative forms *aconite* ceases to be of value. *Phytolacca* continues to be useful.

If the fever is high, pulse full and bounding, face flushed, eyes red, and evidences of intense congestion of the throat are present *belladonna* is the remedy. The prescriber, however, should know when to stop it. If the above symptoms are aggravated after a few doses, stop the remedy at once and give a placebo. A few hours sees the patient free from fever and fully convalescent.

In follicular tonsilitis *phytolacca* is the best remedy we have, and I cannot speak too highly of it. I use the third potency in water every one or two hours as needed. I have also found a course of *phytolacca* of value in chronic hypertrophy of the tonsil.

If suppuration has already begun when the patient applies for treatment, nothing will clear it up more quickly than *hepar sulphuris calcareum.*

Apis mellifica may be of service in cases with marked edema, and without thirst.

Rhus toxicodendron may be of service when the patient feels rheumatic pains during the attack. He can swallow hot things better than cold, cold always aggravates.

The *mercuries* I rarely use. The above remedies usually suffice, although a long list of others could be given.

DIPHTHERIA.

(Greek, διφθερα, a skin or membrane.)

Definition: Diphtheria is an acute infectious disease caused by the Klebs-Loeffler bacillus. Locally, it is characterized by the formation of false membrane over mucous membrane, most often of the pharynx, less often of the nose and larynx, very rarely of the vulva. There are severe systemic disturbances; fever, prostration, and weak heart due probably to absorption of toxines from the local lesions.

Historical Note: Diphtheria is one of the oldest of known diseases. Its symptoms were described by ancient writers. A number of epidemics occurred and were reported at various times during the last thousand years.

In 1811 Samuel Bard, an American, described the disease fairly well. In 1821 Brettonneau, of France, named the condition *Diphtheria.* In 1883 Klebs, and in 1884 Loeffler, isolated and described the same bacillus as the immediate cause of the disease. It now bears their joint names—the Klebs-Loeffler bacillus.

In 1890 Behring and others began experiments which in 1894 resulted in the introduction of the serum treatment of diphtheria, by means of the diphtheria antitoxin.

Etiology: Diphtheria is due to a specific bacillus, the Klebs-Loeffler bacillus. It is transmitted by direct contagion from another case, or by means of something that has been in actual

contact with some other case. Physicians and nurses frequently contract the disease while caring for patients sick with it. It is not probable that the disease is transmitted through the air, as is measles or small-pox. I have seen it stated that in some places diphtheria is treated in the general wards of the hospitals, as is typhoid fever, and without detriment to the other patients.

I had one case in a seaman on board a large steam yacht in May, 1908. The man had been ill two or three days when I was called to see him and was very sick. The membrane on the throat covered the tonsils and soft palate. He had been confined to his bunk in the forecastle where seventeen other members of the crew ate and slept. The bunks were three deep. The patient was removed the next morning after I saw him, and the forecastle fumigated. No secondary cases developed.

Diphtheria is most frequently met with in children, though adults are not exempt. In schools and asylums it may become epidemic because the children are not particular about not drinking out of the same vessel and eating with the same utensils.

A person may have several attacks of diphtheria at intervals.

Bacteriology: The Klebs-Loeffler bacilli vary in size and shape. They are best cultivated on blood serum. Stained with Loeffler's methylene blue solution they show as rods with deeply stained granules and points. The rods may be club shaped or may be constricted in the middle.

The bacilli are found in the exudate on the fauces. They may be found in abrasions of the skin, they may be found in the clothing, they may be found in fatal cases in the lungs and other internal organs.

Morbid Anatomy and Pathology: The Klebs-Loeffler bacilli produce a pseudo-membrane of a whitish or dirty yellow color. It is a fibrous exudate with a network embracing epithelial cells and leucocytes. This undergoes coagulation necrosis. The membrane is usually so attached to the underlying mucous membrane that its removal leaves a raw and bleeding surface.

The diphtheria toxemia may produce necrotic lesions in internal organs. Myocardial and renal degeneration frequently occur. Cardiac thrombosis is more common in diphtheria than

in any other disease. Sometimes degeneration of the peripheral nerves occurs.

Symptoms: The period of incubation in diphtheria is one to three days. McCollom says the specific bacillus enters by an abrasion in the mucous membrane and this acts like an infected wound, developing local and general symptoms from this focus.

The patient suffers from malaise, is feverish and chilly by turns. The temperature usually ranges around 100° F., it may be as high as 103°. The head aches. The throat feels a little sore and may be a purplish red in color. The superficial glands near by may become enlarged, painful and tender.

In about twenty-four hours a grayish membrane begins to show itself on the tonsils. During the next few hours this rapidly spreads over the tonsils, uvula, and soft palate. At the very beginning this membrane can be removed without trouble, but as it gets older it becomes tougher and thicker and at the end of twenty-four hours from its commencement it cannot be removed without leaving a raw and bleeding surface.

As the disease progresses the temperature goes up usually to about 103° F., rarely to 105°. The throat becomes more sore and there is pain on swallowing. The heart becomes weak and the pulse rapid. The neck is stiff and the glands swollen and tender. The prostration is marked. The patient may become delirious, apathetic, or even sink into coma. The breath becomes foul and has a septic odor.

The urine shows albumin, sometimes casts and blood. It may be much diminished in amount.

In very severe cases death closes the scene in three to five days. In milder cases the disease lasts a week or more—it has no definite limitation—the membrane is thrown off, and the patient gradually recovers.

Post-diphtheritic paralyses result in quite a large percentage of cases.

The membrane may appear in the larynx. This laryngeal diphtheria is usually secondary to, or an extension of, the ordinary pharyngeal type of the disease. If primary the first symptom is apt to be a harsh, croupy cough. The patient is hoarse, or

the voice may be lost altogether. There is stridulous breathing, and as the disease progresses dyspnea becomes marked. There is play of the alæ nasi, the sterno-cleido-mastoid muscles stand out, the supra-clavicular and substernal spaces retract. There is paroxysmal cough, during which shreds of membrane may be thrown off. Death may occur from suffocation.

Altogether the general symptoms of laryngeal diphtheria are more marked than in the usual form. The pulse becomes more rapid and feeble, the prostration more marked. The temperature may not be so high.

Sometimes the diphtheritic membrane extends upward into the nose. In these cases there is a very offensive discharge from the nose. The glandular involvement is severe. The constitutional symptoms are marked.

The diphtheritic membrane may attack the eye, it may appear on the genitals, it may develop at any point on the skin or mucous membrane where there happens to be an abrasion.

In places other than the site of the original lesion the membrane is usually the result of auto-infection.

Occasionally the eyes of the nurse or physician become infected from an unexpected cough of the patient. The eyes of the attendants should, therefore, always be protected while examining or treating the throat of the diphtheria patient.

Complications and Sequelæ: Nephritis is always present in severe cases, and frequently in mild cases. It usually appears during the second week.

Broncho-pneumonia is quite a frequent complication, less often lobar pneumonia occurs.

Erythema sometimes occurs. In fatal cases purpuric hemorrhages are found.

Cervical adenitis is quite frequent.

The most common sequel is diphtheritic paralysis, usually of the palate. This produces a change in the voice, and if extensive interferes with swallowing as well. McCollom says paralysis, more or less extensive, follows diphtheria in about 40% of the cases. Paralysis of any nerve may occur. The patellar reflexes are lost. Cases of post-diphtheritic paralysis usually recover.

Paralysis of the heart may occur, or there may be a true degeneration of heart muscle. The heart complication—irregular pulse, disturbed rhythm, paralysis—may occur in the fourth or fifth week when convalescence was supposed to be fully established. It is therefore important to watch the heart constantly until the patient is discharged. The heart complications in no way correspond with the severity of the case, they may follow the mildest as well as the severest forms of diphtheria.

Diagnosis: The clinical symptoms of diphtheria as a rule are quite clear as described above. The ordinary pharyngeal diphtheria may be mistaken for tonsilitis or vice versa. Tonsilitis has higher fever and the membrane does not travel from the tonsils. Moreover, removal of the membrane of diphtheria leaves a bleeding surface, removal of the membrane of tonsilitis does not. The toxemia of diphtheria produces a septic condition after a few days that is not present in tonsilitis. In doubtful cases we have the microscope to fall back on. Cultures should always be made when possible, as simple examination of the membrane itself may prove negative, even when examination of a culture shows the diphtheria bacilli to be present.

Scarlet fever may be differentiated from diphtheria by the history of the case. In scarlet fever the onset is abrupt, the fever is high, the throat is sore, but there is no membrane, and the eruption appears in a few hours. In diphtheria the onset is less severe, the throat is less sore, the membrane appears, and if there is any eruption it is petechial or purpuric.

Finally diphtheria may be associated with, or complicate, other diseases; notably, measles and scarlet fever.

Prognosis: The prognosis should always be guarded. Severe cases may end life in three or four days. Other cases may die of heart failure after four or five weeks. The sudden onset of vomiting late in the disease is always of bad omen. Development of a very rapid or a very slow pulse is bad. Complications are always serious. Diphtheria complicating other diseases is also of serious import. The prognosis is unfavorable in the very young or the very old.

Treatment: Prophylaxis consists in isolating the diphtheria

patient at once. He should be placed in a room by himself and none but the necessary attendants should see him. This is to protect the patient from excitement as well as to prevent infection of others. All unnecessary furnishings should be removed. The clothing and bedding should be changed as often as necessary, and soiled articles should be sterilized at once by being immersed in an antiseptic solution, or by boiling. Eating and drinking utensils should be treated in like manner.

The physicians and attendants should be careful, when examining or treating the throat, to wear a gown or apron in order to protect their own clothing from being coughed upon and so acting as carriers of infection to others. To protect themselves they should be careful not to inhale the breath of the patient. They had best wear large glasses to avoid infection of their eyes from the unexpected ejection of germs by the patient.

Water may be given ad libitum. Milk is the best diet until convalescence, when broths, soups, soft boiled eggs, custards and other concentrated foods may be used. After convalescence is well established a full diet is indicated to build up the patient.

Pineapple juice is a useful article of diet. It is pleasing to the taste and seems to be of some use in cutting the membrane.

Brandy is necessary if the heart goes bad.

Since 1895 diphtheria antitoxin has been the accepted remedy by the majority of physicians. Whether the antitoxin treatment gives better results than careful homœopathic prescribing is doubtful. The two may be used in conjunction. Dr. E. C. M. Hall, of New Haven, in 1896 collected 753 cases treated allopathically with a mortality of 34.45 per cent.; and 146 cases treated homœopathically with a mortality of 21.23 per cent. In the same year Dr. R. W. Tooker, of Chicago, reported 8,000 cases treated with antitoxin with a mortality of 22 per cent.; and 315 cases treated homœopathically with a mortality of 7.3 per cent. In the early days of antitoxin there was some opposition to its use because it was claimed to be injurious to the kidneys. The direct mortality in the Boston City Hospital before the days of antitoxin was 30 to 50 per cent. Since then it has been from 10 to 15 per cent., not as low as Tooker's homœopathic figures.

Diphtheria antitoxin is put up by Boards of Health and by manufacturing chemists in packages containing a certain number of "units." It is given by hyperdermatic injection. Mild cases, seen early, may begin with 4,000 units, to be repeated every four to six hours until the membrane has disappeared. If the case is severe and the membrane extensive when first seen, as much as 6,000 units may be given at a dose.

The beneficent effect shows itself, according to McCollom, in a "shrivelling of the membrane, diminution of the nasal discharge, a correction of the fetid odor, and general improvement."

The diphtheria antitoxin is also used for immunizing purposes in those exposed to diphtheria, apparently with satisfactory results.

Local applications are not of special value. If used they must be non-irritating to the normal mucous membranes else they will do more harm than good by giving the membrane a larger surface to work on. Placing the patient on his side and irrigating the mouth with a mild solution of boracic acid may be of service. An ordinary irrigating bag and tube may be used, the flow being regulated by the height of the bag.

Hydrogen dioxide one part to three or four of water may be used.

Alcohol and water as a spray or gargle is sometimes useful.

In laryngeal diphtheria a steam tent is sometimes of service. In this form of diphtheria, if choking is severe and the patient seems in imminent danger of suffocation, tracheotomy may be performed. It is best done by dissecting the outer tissues, tying bleeding vessels as they appear, and then cutting the trachea directly through its centre, longitudinally. The wound of course must be kept open. How long it should be kept open depends on the progress of the case.

In 1885 O'Dwyer, of New York, made intubation a prominent means of relief. The tubes can be procured in sets. The tube selected, according to the size of the patient, is passed into the larynx, thereby allowing of the entrance of air into the lungs. If it becomes clogged it may have to be removed—the patient then coughs up some membrane and the tube may be replaced.

The homœopathic drugs of most service in diphtheria are the various preparations of mercury. Of these *mercurius cyanatus* ranks high. It is indicated by the general septic condition, and especially in the laryngeal form of diphtheria. It should never be used below the sixth potency.

Ignatia is highly recommended by Raue, Sr., purely on subjective symptoms.

Carbolic acid is indicated when the patient is greatly prostrated, with weak pulse, pale face, and extensive membrane. Some physicians consider that the good effects of antitoxin originate in the carbolic acid used as a preservative.

Apis may be called for where there is excessive edema. C. E. Fisher has reported excellent results with the 200th potency.

Phytolacca may be called for when there is much glandular involvement.

Among other remedies that may be thought of are *arsenicum album, baptisia, lachesis, muriatic acid,* and *nitric acid.*

In post-diphtheritic paralysis *gelsemium* should always be thought of. It was highly recommended in my student days by Prof. Edgar V. Moffat.

Zinc phosphate is recommended by Dr. George W. McDowell,

GLANDULAR FEVER.

Definition: Osler defines glandular fever as "an infectious disease of children, developing, as a rule, without premonitory signs, and characterized by slight redness of the throat, high fever, swelling and tenderness of the lymph-glands of the neck, particularly those behind the sterno-cleido-mastoid muscles. The fever is of short duration, but the enlargement of the glands persists for from ten days to three weeks."

Historical Note: Glandular fever has been reported by many clinicians for a long time. Osler says Pfeiffer called special attention to it in 1889.

Etiology: The cause of glandular fever is not definitely known. It is well to remember that swollen lymph glands have been found often in children who later in life have developed tuberculosis. Infants and young children are most often affected.

Morbid Anatomy and Pathology: The liver and spleen are frequently enlarged in glandular fever.

Symptoms: The symptoms of glandular fever come on, as a rule, without premonitory signs. The patient is feverish and irritable, the appetite is poor, and the child seems sick without definite cause.

Examination shows the glands of the neck to be swollen, sometimes tender and painful. They may be swollen enough to be seen, or they may simply be palpable, like beads along the lines of the lymphatics.

The temperature may go to 102° or 103° F. As a rule the fever subsides in a few days, but the swelling of the glands may continue for some time.

Complications and Sequelæ: Very rarely glandular fever may end in suppuration of one or more glands.

Diagnosis: Simple glandular fever may be confounded with adenitis due to some serious infection. Any possible underlying cause must be searched for and eliminated before a diagnosis is made.

Prognosis: The prognosis of glandular fever is always good.

Treatment: The child should be put under the best possible hygienic conditions. The food must be carefully regulated.

The cases that I have seen have responded promptly to the Homœopathic remedy.

Belladonna is indicated when the swollen glands are sensitive and the fever considerable.

Calcarea carbonica is indicated if the child seems ill-nourished, or if large and fat and perspires easily. Some children will have recurring attacks. Calcarea given at infrequent intervals for several months will often give good results.

Phytolacca is useful if the fever goes down and leaves the glands swollen.

RHEUMATIC FEVER.

(Greek, ῥευμα, to flow.) The poison was supposed to flow from the brain.

Synonym. Acute Articular Rheumatism.

Definition: Rheumatic fever is a non-contagious, self-limited,

febrile disease. It is characterized by acute inflammation of one or more joints, the inflammation changing its location frequently during the progress of the disease. It sometimes affects the fibrous tissues of parts other than the joints.

Historical Note: There has been, and still is, considerable confusion regarding an exact definition of rheumatism. Arthritis due to gonorrhea, or to various other infective conditions, is sometimes classed as rheumatism. Multiple neuritis is also sometimes called rheumatism. But true rheumatic fever, or acute articular rheumatism, is a distinct disease entity. It was first described by Sydenham. In 1862 Austin Flint, Sr., after observing a series of cases at Bellevue Hospital that were taken care of but received no medicine, stated that rheumatic fever was a self-limited disease, lasting on the average about three weeks. Since that time his conclusions have been confirmed. In recent years rheumatic fever has been classified as an acute infection, on account of its general character, course and duration. The exact nature of the poison still remains unknown.

Etiology: Rheumatic fever is a disease found almost exclusively in temperate climates. The inhabitants of arctic regions and of the torrid zone are practically free from it. Certain places in Europe also seem to be exempt. The majority of cases in this country occur during the winter and spring when the weather is especially changeable, although cases may develop at any season. Anders and Osler and others say there is some evidence of contagion. It is known that rheumatism is sometimes epidemic. Cold is said to be an exciting cause. It has been found that those exposed to long continued cold and dampness are more liable to be attacked than those subjected to cold alone, or than those occasionally exposed. Many cases are preceded by an attack of tonsilitis.

The majority of patients are between ten and thirty-five years of age, though no age is exempt. Heredity seems to be an important etiological factor. The sexes are equally susceptible.

Many patients have more than one attack. It seems to be the rule that a person who has had one attack is more susceptible to the exciting causes than one who has never had it. One of my hospital cases reported that he had had seven attacks.

The disease is more prevalent among the working classes. It is quite common in public hospitals. In my service at the Flower Hospital in March, 1910, of 63 patients 16 had rheumatic fever.

Bacteriology: A number of observers have at different times announced the finding of a specific germ for rheumatic fever. Poynton and Paine in 1900 reported a series of cases in which they thought they had at last found the true bacillus. A few others confirmed their findings, more did not. At the present writing the exact cause is not known.

Morbid Anatomy and Pathology: The pathological changes that take place in the joints in rheumatic fever are not marked. There may be an increase in the amount of the synovial fluid, and the cartilages may be thickened. When the heart is involved there is first a thickening of the endocardium with an increase of fibrinous material. Later there may be granulation and formation of scar tissue. The pleural cavities may contain an excess of fluid. There may result from the endocarditis infarcts in the spleen and kidneys, and cloudy swelling of the parenchyma of other organs.

The amount of fibrin in the blood is increased.

Symptoms: The onset of rheumatic fever is usually abrupt. Often it follows an attack of tonsilitis. There is loss of appetite, the patient is uneasy, fever develops, and then one or more joints present all the characteristics of an acute inflammation.

In the affected joint there is swelling, sometimes very great. There is more or less redness and intense pain and tenderness. The patient cannot bear to have the affected joint touched or moved. The leading characteristic of rheumatic fever is its tendency to shift from one joint to another. The most acute symptoms may develop in one set of joints in a few hours, last for a day or two, and then disappear as rapidly as they came only to reappear in some other part.

Flint called rheumatic fever a symmetrical disease, because if the corresponding joints on both sides are not affected at the same time we are apt to have a knee and an elbow, or an ankle and a wrist, or a hip and a shoulder simultaneously affected.

Rheumatic fever never leaves the joints permanently impaired. It sometimes attacks immovable joints like the symphisis pubis.

Another characteristic of rheumatic fever is an acid and profuse sour-smelling sweat. A rash may result from it.

In a few cases I have seen purpuric spots on the legs.

The urine is scanty, of high specific gravity, has a high color, and is intensely acid. It frequently contains albumin.

The appetite is poor, the tongue is coated. Rarely are the digestive disturbances more marked than this.

The fever is irregular, rarely going higher than 103° F. The more intense the other symptoms the higher the fever.

The pulse rate is not high—90 to 100 or thereabouts. If the heart becomes involved the pulse may become fast or it may become irregular or it may be both.

The severe pain may keep the patient from sleep. Delirium is rare. Occasionally the disease attacks the meninges of the brain, a very serious complication.

In the *child* rheumatic fever presents some peculiarities that should be remembered. The cases are atypical. As a rule, in very young children, the acute and severe swellings of the joints as found in the youth or adult are lacking. The little patient may complain of indefinite pains here and there that are not very severe and so may be neglected. These indefinite pains call for careful investigation. Formerly called "growing pains," for want of a better name, it is now known that they frequently mean rheumatic fever. If neglected the heart may become involved. An attack of the rheumatic poison on the heart is in nowise related to the severity of the joint affection:—the mildest arthritis may be associated with the most dangerous heart complication.

Complications and sequelæ: The most common as well as the most serious complication of rheumatic fever is acute inflammation of the heart and its membranes. We may have endocarditis, myocarditis, or pericarditis. In fact, so common is the first that it may almost be classed as a symptom rather than a complication. When endocarditis develops it is usually during the first week of the rheumatic fever, rarely later, although it may

develop at any time. Sometimes it precedes the arthritis. Valvular murmurs, usually a soft blow with the first sound of the heart, may appear early and leave when the patient recovers from the rheumatic fever. The ordinary course, though, is for the endocarditis to produce a permanent impairment of the valves of the heart and leave the patient with permanent heart disease. In my experience the mitral valve is the one usually affected.

Pericarditis and myocarditis are much less common. Both may be of serious import.

Hyperpyrexia is another somewhat uncommon but serious complication. The joint pains suddenly subside and the fever goes up to an unusual height—107° or 108° F. or higher. The cause is not known.

Pleurisy, and less often pneumonia, appear as complications. Pneumonia makes the outcome exceedingly grave. I had one such case in my service at the Flower Hospital in 1910 whose life hung in the balance for days, but who finally recovered.

Diagnosis: The diagnosis of a typical case of rheumatic fever should not be difficult. It may be confounded with acute gout. The main points of difference are that acute gout usually occurs in those past middle life, whereas, rheumatic fever is most frequent in youths and young adults. In gout the typical beginning is an inflammation of the great toe; the smaller joints, too, are more apt to be affected, and the inflammation does not travel about. In rheumatic fever the knees, ankles, elbows, and wrists are the favorite seats of the disease. Sufferers from gout usually present a history of spirit or malt liquor drinking. Gout also lacks the sweats and the heart complications of rheumatic fever.

Acute synovitis is usually of traumatic origin and does not travel from joint to joint.

Arthritis may follow some of the other acute infections, as gonorrhœa, septicemia, or scarlet fever. In such cases the previous history is of importance. It is rare indeed for any of these forms of arthritis to present the characteristics of rheumatic fever—such joint affections do not shift from joint to joint, the characteristic rheumatic sweat is wanting, and the temperature curve is different.

Multiple neuritis is sometimes mistaken for rheumatic fever, but the wrist drop and foot drop should differentiate it.

Prognosis: The prognosis of rheumatic fever is good as to life in uncomplicated cases. Only about three per cent. of cases die during an attack, but a much larger per cent. die eventually of the resultant heart lesions. As the heart involvement may appear at any stage of the disease, we can never be sure just how the case will terminate.

Relapse in rheumatic fever happens occasionally. One relapse is not uncommon; I have seen as many as five, one following the other keeping the patient invalided for months.

Treatment: Exposure is an important factor in the causation of rheumatic fever. Avoidance of exposure is therefore a good prophylaxis.

The most important single factor in treatment is absolute rest in bed; all other things—hygiene, diet, drugs,—are of secondary consideration. Rest in bed must be maintained until all symptoms of pain and fever have been absent for at least a week. In complicated cases with heart involvement rest must be maintained until the heart has a chance to adjust itself. During the height of the disease the patient is willing to remain quiet because moving around causes pain. After the symptoms have subsided it may be difficult to impress him with the importance of remaining quiet. I regret to say, too, that the physician is not always sufficiently impressed with the need of continued rest for the patient. I have seen so many relapses brought on by too early getting up that I believe the stay in bed for at least a week after all fever and pain are gone to be imperative.

The bed should be flat and not too soft. On account of the profuse sweating the patient may find a woolen gown and blankets more comfortable next the skin than muslin or linen.

Considerable comfort may be afforded by wrapping the affected joints in cotton batting. Oil of wintergreen, locally, will give added relief if it is necessary to use external applications. I rarely find it necessary.

Milk is the best diet during the height of the disease. If not well borne it may be peptonized, or it may be diluted with water or vichy.

A very useful adjuvant in my experience has been the free use of water. An ordinary glassful of water should be drunk by the patient every hour from 8 a. m. to 7 p. m. This flushes the system and seems to lessen the pain, lower the fever, and lessen the odor from the perspiration. Occasionally, after three or four days, the water will cause nausea. In that case the intervals between its administration must be lengthened.

To relieve pain and allow sleep, codeine in ⅛ grain doses may be used. It will rarely be necessary where the water and the indicated homœopathic remedy are used.

The salicylates are used very extensively. Osler recommends salicin in 20 grain doses every hour or two, or salicylate of soda every two hours. When the pain is gone the interval should be lengthened to four or five hours till the fever goes. It must not be forgotten that large quantities of the salicylates may produce temporary deafness. If this occurs the salicylate must be stopped.

Oil of wintergreen in 20 drop doses may be given in milk every two hours.

Colchicine, recommended by Goodno has not proven satisfactory in my hands. Goodno recommends a preparation of one grain of colchicine to one ounce of alcohol. He uses three to five drops every two or four hours. If gastro-intestinal symptoms appear, stop the remedy.

My personal preference is for the indicated homœopathic remedy. I do not believe the salicylates are as useful.

Aconite is indicated in cases with acute pain, high fever, and other symptoms of active inflammation. It is the best remedy for acute endocarditis.

Ferrum phosphoricum is very similar.

Bryonia is useful all during the disease. The slightest motion causes pain. The patient is thirsty.

Rhus toxicodendron is indicated when the patient is restless, the pain may be severe, and is relieved by change of position.

Cimicifuga is of value especially in nervous patients.

Pulsatilla is indicated where the rheumatic pains are constantly shifting, and there is fever without thirst.

Other remedies are *arnica, belladonna, caulophyllum, dulcamara, sulphur.*

SCARLET FEVER.

(So called on account of the color of the rash.)

Synonym: Scarlatina.

Definition: An acute contagious disease ushered in by sore throat, high temperature, and within twenty-four hours by the appearance of a more or less diffuse rash, beginning first on the back and extending thence to all parts of the body. Desquamation begins about the seventh day.

Historical Note: Scarlet fever as a separate disease was first differentiated by Sydenham.

Etiology: The specific cause of scarlet fever is not known. It is highly contagious and may be transmitted either directly or by means of clothing or other things that have been in contact with a patient suffering from the disease. One case that I know of was carried by a clinical thermometer.

Scarlet fever is a disease of childhood, although adults are by no means exempt. Young children are more susceptible than older ones.

One attack usually protects for life, although a few persons have the disease a second time.

Morbid Anatomy and Pathology: There are no specific lesions found after death from scarlet fever. The rash disappears.

Symptoms: The incubation period varies from a few hours to a week or ten days. Most often symptoms will appear four or five days after exposure.

The invasion is usually somewhat abrupt. In very young children there is nausea and vomiting, sometimes stupor, delirium, or even convulsions. The temperature goes up quite suddenly to 104° F. or higher. At this time, the patient also complains of sore throat.

Within twenty-four hours, rarely longer, the rash appears on the chest and back, then further down on the trunk, and finally on the legs and arms. The face is rarely affected, although the scarlet fever patient often has a peculiar expression due to very

red lips, which by contrast cause the surrounding skin to appear to be abnormally white. The upper part of the back is the best place to study the rash in the beginning. On close examination it is seen to consist of reddish punctate spots set close together. The skin between is erythematous, so that the entire surface presents a nearly diffuse redness. A miliary eruption of minute vesicles is sometimes found in the groin and axilla. Moreover, McCollom lays stress on the fact that in all cases the rash is accentuated in these regions.

The throat is sore, and the fauces and uvula show the eruption. The tongue is quite characteristic. At first it is white with the papillæ showing through, looking like an unripe strawberry. Later it is red with the papillæ very prominent, the so-called strawberry tongue.

The pulse is unusually high, 120 to 160. The respirations are somewhat increased, going up in proportion to the rise in temperature. The temperature rises abruptly, reaching 103° to 105° F. It may fall by lysis or by crisis..

The lymphatic glands are often enlarged, particularly those of the groin, less often those in the neck and axilla.

There is loss of appetite. The nausea and vomiting sometimes found soon ceases. Occasionally there is diarrhea. Except in very severe cases the stupor or delirium of the onset does not last long. If either condition persists the outlook is not favorable.

The urine frequently contains albumin. Acute nephritis may supervene.

At the end of six or seven days desquamation sets in, beginning where the rash began on the chest and back, thence extending down the trunk and finally over the extremities. The skin flakes off in smaller or larger pieces, sometimes from the hands and feet in almost perfect moulds. As desquamation begins the other symptoms subside. In uncomplicated cases the patient is practically well except for the peeling, in two weeks. Desquamation persists for four or six weeks. Sometimes a second desquamation takes place.

Most of the books make no special mention of the appearance of scarlet fever in the negro. I have seen quite a number of such

cases. The intensity of the natural color is augmented where the rash is present. The sore throat, temperature, and general symptoms are of course the same as in white persons. The desquamation is also the same, but the exfoliated particles look very white, and where peeling has occurred, minute white spots are left at first that stand out very clearly against the dark or black skin. A colored girl, aged 14, was inadvertently brought into my ward at the Flower Hospital, March 14, 1910, in the desquamative stage. About the neck and upper part of the back desquamation in large white flakes was very pronounced. Lower down the back was pretty well covered with small white spots where the desquamation had taken place. The throat was sore, pulse 140, temperature 104° F. The patient was brought in by the ambulance while I was in the ward, and after examination was promptly isolated, and later sent to the Health Department Hospital.

Varieties: In epidemics of scarlet fever there will be cases presenting all the symptoms except the rash, the so-called *scarletina sine eruptione*. They will have sore throat, fever, rapid pulse, but no rash.

The *malignant* form. Occasionally the onset is so sudden, and the toxemia so profound, that the patient dies in a few hours.

Very rarely the eruption is accompanied by hemorrhages in the skin;—the *hemorrhagic* type.

Finally, there is the *diphtheritic* form, where a true diphtheritic membrane covers the palate and tonsils.

It is not at all uncommon for those in attendance on a case of scarlet fever, mothers or nurses, to develop sore throat with fever, but without a rash. This may happen to those who have already had scarlet fever. I always advise against the attendance of a nurse on a scarlet fever patient if she has never had it herself. Even physicians are not exempt. Dr. J. E. Ambler, of New York, died of scarlet fever contracted from his own child in 1913.

Complications and Sequelæ: Acute middle ear disease is one of the most common complications of scarlet fever. Its advent will be indicated by a rise in temperature, or by earache, or by

both. Sometimes the appearance of an otorrhea will be the first evidence of it.

Albuminuria is common; but if the urine suddenly becomes scanty and high colored it probably means nephritis. This is a serious complication, but not infrequent. The urine should be examined from day to day in order to guard against nephritis.

Endocarditis is not uncommon. In fact, scarlet fever is one of the most frequent causes of endocarditis in my experience. It is not mentioned by Caiger and Dudgeon in Allbutt's System of Medicine.

Arthritis sometimes develops late in the case. It is septic in character.

Osler declares pleurisy with pneumonia to be one of the most dangerous complications.

Adenitis sometimes supervenes.

Quinsy developed in one of my cases.

Diagnosis: The diagnosis of a typical case of scarlet fever is easy. Probably measles is the disease most often confounded with it. It should be remembered that measles is ushered in with a coryza accompanied by fever which subsides in a day or two, and that the rash appears first on the face with a second rise in temperature, and that there is no sore throat; whereas, scarlet fever has no initial coryza, but does have sore throat with fever, and a rash which appears promptly, first on the body and rarely on the face; if these symptoms are remembered there should be no confusion.

German measles, rötheln, has a more marked adenitis than scarlet fever, less sore throat, and almost no fever. The rash looks more like that of measles proper, minus the swelling.

Some drug rashes may simulate scarlet fever, but they do not exhibit the sore throat.

Prognosis: The prognosis of scarlet fever should always be guarded. Until desquamation has ceased and the patient is well the unexpected may happen. The urine should be frequently examined until the end, as nephritis may develop late in the desquamating stage. The heart should be frequently examined because of a possible endocarditis.

The treatment also has something to do with the prognosis. Under homœopathic treatment the mortality is about half what it is under old school methods. Dr. E. C. M. Hall, in 1896, gave me the following figures taken from the records at New Haven. Of 1,271 cases of scarlet fever reported by old school physicians, the mortality was 10 per cent. Of 209 cases, reported by homœopathic physicians, the mortality was 4.3 per cent.

Treatment: The scarlet fever patient should be isolated at once. He should be placed in a large sunny room from which all unnecessary furniture has been removed. The room should be well ventilated, but the patient must not be exposed to drafts. A sheet kept wet with some antiseptic solution should hang over the door.

Soft diet, milk, broths and eggs is the best, with plenty of water to drink.

After the rash begins to subside and desquamation begins, daily anointings with cocoa butter or carbolized vaseline will be comforting to the patient and prevent distribution of the exfoliations. Sponge baths are useful throughout, and two or three warm baths with ordinary soap and water should be given before the patient leaves his isolation quarters.

The nurse or attendant should wear a gown, and should thoroughly fumigate herself on coming out. The physician on his visits had best wear a gown while with the patient. I provide myself with an ordinary linen duster when visiting contagious cases.

All dishes should be washed and boiled after use. All soiled bed linen and clothing should be put in an antiseptic solution and then boiled as in other infectious diseases.

The discharges should likewise be sterilized before being thrown away.

If ear complications develop, the best wash is a mixture of boracic acid, alcohol and water in equal parts, a combination first suggested to me by Dr. George A. Shepard. This mixture should be kept in a bottle and allowed to stand until the excess boracic acid has settled to the bottom. The ear should be cleansed with cotton, and then a few drops of the solution poured

into the ear and allowed to stay for two or three minutes. This in turn should be wiped out. This process may be repeated every two hours to twice a day as necessary, until the discharge ceases.

If endocarditis develops with excessively rapid or irregular pulse, an ice-bag applied over the cardiac area may be of service. The old school relies solely on the hygienic treatment.

Many remedies may be indicated homœopathically. Probably the most frequently indicated is *belladonna.* This has the sore throat, the fever, and the erythematous rash. I use the third potency in water every one or two hours. When the fever begins to go down the interval between doses should be lengthened.

Phytolacca may be used when the throat symptoms are severe.

Mercurius corrosivus is a frequently indicated and valuable remedy, particularly if the kidneys become involved.

Aconite is sometimes of service in the very beginning. It is also indicated if endocarditis develops.

Apis mellifica is useful if there is nephritis or endocarditis with edema.

Arsenicum album is useful in adynamic cases, with great prostration and restlessness. Nephritis is an additional indication.

Hepar is good for retarded convalescence. It is also good for the ear complications.

Calcarea picrata is indicated in the beginning of middle ear involvement.

MEASLES.

(German, *maser,* a spot.)

Synonyms: Rubeola. Morbilli.

Definition: Measles is an acute infectious disease, involving the skin and mucous membranes. It has an initial coryza, a fine rash appearing on the face and thence extending over the entire body, which is followed in a few days by desquamation. The eyes and respiratory mucous membranes are also usually affected. In my cases the time from the beginning of invasion till the end of desquamation has varied between twelve and fourteen days.

Historical Note: Measles is said to have been described by

Rhazes in the tenth century; but up to the close of the eighteenth century it was supposed to be a form of small-pox. Even Sydenham, who differentiated scarlet fever from the other exanthems in 1676, apparently did not make measles out to be a distinct disease, but considered it to be a form of small-pox. For the last century and a half it has been recognized as a distinct disease entity.

Etiology: The specific cause of measles is not known. Whatever it may be the virus of measles is, next to that of small-pox, the most contagious that there is. Measles is communicable from the beginning of invasion to the end of desquamation. The contagion is less tenacious than that either of small-pox or of scarlet fever. It may be transmitted by formites. An epidemic in a girls' boarding school in Stamford started from a letter received by one of the girls from a distant measles patient. Physicians if not careful may carry it from one patient to another. It is my custom to wear a linen duster into the sick-room of a contagious case. The first cost of such a garment is little, and it is easily kept disinfected.

Children are most often affected, though infants in arms usually escape. My youngest patient was six months old, and I have had one ten months old; those were exceptionally young.

In great camps of soldiers measles has sometimes run epidemic and caused a large mortality. When taken into a new community measles spreads with great rapidity and with a high mortality.

Adults are sometimes affected, especially those who have escaped during childhood. It is always a serious disease late in life.

As a rule one attack confers immunity for all time. Numerous exceptions occur. I have had a number of patients who have had measles two and three times.

Morbid Anatomy and Pathology: Measles causes no specific anatomical lesions. The rash disappears after death.

Symptoms: The period of incubation averages about ten days. It may be but four days, very rarely it may be as much as thirty days. After a child has been exposed to measles, at least two weeks should pass without the exhibition of any symptoms be-

fore we can feel assured that the disease will not develop. One of my patients, a little girl, came down with rubeola exactly seven days after invasion in an older sister. The older child had been isolated immediately, and the younger one given *pulsatilla* as a prophylactic—but without success. There are no observable symptoms during the incubatory period.

The stage of invasion is ushered in with all the symptoms of an influenza. The patient has coryza, the eyes run and are sensitive to light; there is pharyngitis and laryngitis, with hoarseness; there is bronchitis, with a dry cough. The patient feels chilly, but has no distinct rigor. The temperature runs up to 102° or 103° F. at the outset, to drop a little on succeeding days, until the eruption appears, when the temperature rises abruptly to 103° to 104° F. The tongue becomes covered with a thick white fur, the tip and edges remaining red. Epistaxis sometimes occurs. Convulsions are rare.

Usually, about the third day, it may be the second to the sixth, the eruption begins to show itself. It appears first in the buccal cavity, on the soft palate, uvula, and mucous membrane of the cheeks, this is sometimes known as "Koplik's spots." One of my patients presented an acute pharyngitis when I first saw her, together with a slight coryza, but I failed to note any eruption until it appeared on the face, forty-eight hours later. Another patient, whom I had an opportunity to watch carefully from the beginning of incubation, showed the eruptive spots on the pharynx and roof of the mouth on the third day of invasion.

From twelve to twenty-four hours after the appearance of the eruption in the mouth it begins to show itself on the skin. It appears on the face, then on the chest, arms, and legs, in the order named. It is said to come first on the forehead, but in two of my cases the cheeks were first attacked. The eruption consists of minute red papules that can be felt before they can be seen. At first they are distinct, but soon they form themselves into groups or patches, with curved or crescentic outlines. The color of the skin is not uniform, but has a mottled appearance and is swollen. It feels hot and dry, and sometimes there is intense itching. The rash is dark red and disappears on pres-

sure. It takes thirty-six to forty-eight hours for the body to become completely covered. It comes down over the patient like a wave. The eruption at any given point remains at its maximum about twelve hours and then begins to fade in the order in which it appeared. This stage lasts four days to a week.

During the eruptive period lachrymation and photophobia are more marked. The bronchitis increases and the cough becomes more frequent and looser and with more or less expectoration. In one of my cases the respirations rose to 60 a minute, and the cough was incessant.

The temperature rises as the rash appears and reaches its maximum in a few hours. It may be as high as 105° F.

The patient is restless at night and drowsy by day. The appetite is lost and the patient is usually very thirsty. The tongue is coated with the enlarged red papillæ showing through. There is a bad taste in the mouth. Sometimes there is nausea and vomiting; more often there is diarrhea. Occasionally there is delirium. This has happened in several of my cases.

The bronchitis and diarrhea are supposed to be due to the rash extending to the affected mucous membranes.

The stage of desquamation lasts from four to eight days. It begins when the rash begins to fade. All the symptoms decline as the eruption disappears. The expectoration becomes nummular. The temperature falls by crisis. The skin does not peel off, like in scarlet fever, but is given off in fine bran like particles. Convalescence is rapid. In ten to fourteen days from the onset of invasion the patient is practically well. In rare cases desquamation does not take place. I had such a case, a girl of thirteen with a most profuse rash, in the spring of 1912.

Varieties: Measles may present itself in an anomalous form. The rash may be hemorrhagic in character, the so-called *"black measles."* During epidemics occasional cases are seen that exhibit all symptoms, excepting the rash. Such cases are usually very mild. We may also have the rash without the catarrhal symptoms. Again, cases may run the usual course excepting that all symptoms appear to be very much aggravated. True

relapse in measles is extremely rare. As a rule, measles occurs but once in the same person. Exceptions are numerous. I have myself taken one patient through three attacks, and some of my other patients had had the disease before I treated them for it.

Complications and sequelæ: The complications of measles of most importance are those of the respiratory tract. Catarrhal symptoms of the eyes, nose, throat and bronchial tubes always accompany measles and are a part of it. When especially severe these symptoms require special consideration.

Capillary bronchitis followed in two of my cases and the patients died.

Pneumonia occasionally occurs.

Miliary tuberculosis, especially in very young children, is said to follow measles with comparative frequency. The usual form of pulmonary tuberculosis may have its origin in measles. I have met with neither.

The associated bronchitis may become chronic.

Laryngitis, even to aphonia, may complicate measles. One boy, aged twelve, developed aphonia.

I have seen severe tonsilitis associated with measles.

Stomatitis is quite common. Sometimes diarrhea is a very troublesome condition. The eruption attacks the mouth we know, and probably the entire digestive tract.

Acute otitis media sometimes occurs with measles; usually it does not develop till the stage of desquamation. It can usually be cured. Sometimes it becomes chronic, although the chronic condition does not follow measles as often as it does scarlet fever. I have met a number of cases of it.

Diagnosis: The diagnosis of measles during invasion may be confounded with influenza. The subsequent course of the discase will leave no doubt as to its character. In the midst of an epidemic of rubeola the catarrhal symptoms will, of course, excite suspicion.

In measles the catarrhal symptoms and the temperature usually subside on the second day, and the patient feels comfortable. This has led, at times, to a confounding of measles with malaria.

Rötheln, or German measles, may be mistaken for rubeola. The German variety has no stage of invasion, no stage of desquamation. The eruption appears at once, and is paler than that of measles. As a rule, the spots are fewer, and do not tend to coalesce. There is apt to be sore throat. The glands in the neck are palpable.

In scarlatina the onset is more sudden than in measles, and the evidences of systemic poisoning are more marked. The pulse is rapid, there is usually vomiting, a true angina supervenes, the catarrhal symptoms are lacking, and the eruption comes on within twenty-four hours. The scarlet fever rash is more of a diffuse redness, the color is a bright scarlet. When in patches the outline is irregular, whereas in measles it is crescentic or curved.

Variola presents lumbar and sacral pains in the beginning. Unlike measles, the temperature drops on the appearance of the rash. The rash itself is vesicular and appears earlier in the disease. Later it becomes pustular.

Prognosis: The prognosis of measles is good. Uncomplicated cases usually recover. Enough deaths occur, however, to require the practitioner to exert his utmost skill in handling the disease. In New York State in 1909 there were 52,773 cases of measles reported with 1270 deaths.

I believe the prognosis is influenced by the treatment. In 1896 I published a paper on "Some Reasons for a Belief in Homœopathy" in the *New England Medical Gazette*. Included in that were some figures given me by Dr. E. C. M. Hall, of New Haven. During 1891-1895 there were 286 cases of measles reported in New Haven by old school physicians with a mortality of 17.83 per cent. There were 106 cases reported by homœopathic physicians with a mortality of 1.88 per cent.

The complications and sequelæ are responsible for more deaths than the disease itself. I have personally lost two cases where capillary bronchitis followed. After an extensive epidemic in any given locality it has been found that the mortality among children exceeds the average.

Treatment: The first thing to do when measles is suspected

is to isolate the patient. He should be put in a large, easily ventilated room. On account of the accompanying conjunctivitis the room will have to be somewhat darkened. Other children who have come in contact with the patient should be carefully watched for catarrhal symptoms.

In the eruptive stage, if there is much itching, carbolized vaseline will relieve it. During desquamation anointing with cocoa butter to prevent the scales from traveling through the atmosphere is a good plan. At the end of the illness, and before the patient is allowed to mix with other children, one or two warm baths with soap and water should be given. This is done to cleanse the skin of any desquamating particles that might otherwise serve as sources of infection.

During the fever the patient may be given sponge baths. Steam will often do much to relieve the respiratory symptoms. A sponge soaked in very hot water will relieve croupy symptoms.

There is usually more or less stomatitis. For that an antiseptic mouth wash of boracic acid, or of bensolyptus, may be used.

In case of otitis, the treatment recommended for this complication in the chapter on scarlet fever should be used.

In conjunctivitis, a saturated solution of boracic acid may be used as a lotion.

The diet should consist of milk, gruel, eggs, meat juice—in other words it should be liquid. The milk may be diluted with seltzer water or with lime water if necessary. Water should be given ad libitum.

Old school authorities say no drug treatment is necessary. The homœopathic physician will find his indicated remedy useful here as elsewhere.

I have found *pulsatilla* to be the most generally useful drug. It fits nearly all the symptoms. It is pre-eminently a catarrhal remedy and is called for in the stage of invasion and in the stage of eruption. It is also of great service when diarrhea supervenes.

Aconite may be used advantageously in the beginning with severe inflammatory symptoms accompanied by rapid pulse.

Gelsemium is useful where the coryza is marked and the patient in an apathetic or nervous condition.

Kali bichromicum is useful for the cough and bronchitis.

Antimonium tartaricum may be of service where the cough is loose and rattling.

Arsenicum album has served me well where the gastric symptoms were marked.

If otitis occurs, *ferrum phosphoricum* will usually stop it. If the inflammation goes on to an otorrhea *calcarea picrata* is useful.

Hepar may be used at any stage of the ear condition.

Other remedies of course may be indicated, but the above are those most frequently called for.

GERMAN MEASLES.

(So called because first described by German physicians.)

Synonyms: Rubella. Rötheln.

Definition: An acute contagious disease, characterized by an eruption on the face and body, slight glandular swelling, accompanied by very slight, if any, systemic symptoms, and lasting a week or less.

Historical Note: The first observers to declare rötheln to be a separate disease were De Bergen and Orlow, German physicians, who wrote about the middle of the eighteenth century. Maton, an Englishman, wrote of it in 1815. During the next few years an occasional case was reported in this country. In 1874, J. Lewis Smith, of New York, reported on an epidemic of some 50 cases, and gave a very full description of it in his book on Diseases of Children. He says, "The first case which I observed occurred in the middle of December, in Seventy-first street, being in the suburbs of New York, on the north." The city has grown to and many miles beyond Seventy-first street now, 1914.

Etiology: German measles is pre-eminently a disease of childhood. Smith's cases varied in age from eight months to thirty years. I have seen it in girls and boys up to nineteen years of age.

The specific cause is not known, although it is unquestionably communicable directly or by fomites.

In an epidemic in New York early in 1913, one of my patients, a girl of fourteen, awoke with the rash. The family telephoned the school the same day and were told there was but one other case there, and that in a much younger girl in a lower grade. The next day the school authorities notified my patient that seven of her classmates had German measles. Every patient I had during that epidemic was of school age, or had brothers and sisters who went to school. In every case there were many classmates affected; and in several instances the school member of the family brought it home and gave it to the others.

Morbid Anatomy and Pathology: There are no special pathological changes known.

Symptoms: The period of incubation varies from three to twenty-one days. One of my patients was exposed on Monday and had the rash on Friday.

Usually the earliest sign noted is the appearance of the eruption. In many cases questioning will elicit the fact that the glands at the back of the neck have been swollen for a day or two. In some of my cases the swollen glands and congested eyes have constituted a period of invasion, lasting two or three days, although the patients have felt well.

In the *Journal of the American Institute of Homœopathy* for August, 1913, I reported on the recent epidemic as follows:—

"Without any special premonitory symptoms, as in measles proper, the children have all at once shown a rash on the face. The spots have been very numerous, small, pinkish, and crowded together, giving a peculiar mottled appearance. The appearance has been very similar to that seen in ordinary measles with two important differences. The face has not been swollen as it is in ordinary measles with a similarly profuse eruption, and the eyes have not been as congested as they are in ordinary measles. The throat and tonsils have been somewhat reddened. The glands in the neck and under the chin have been distinctly palpable—a valuable diagnostic sign. Almost at the same time the rash has developed on the back and chest, much more profuse on the back. After a few hours the legs and arms have been covered with the eruption. Within twenty-four hours the rash

has been distinctly less, and in two or three days it has disappeared entirely. There has been no desquamation noticeable.

"A peculiar feature I have noticed in each of my cases that has lasted for a week or more has been this: after the rash has gone pressure of the fingers or of the clothing, or an emotion that would ordinarily cause blushing, any of these things has caused a transient reddening of the skin in irregular patches. I noticed this after palpating the swollen glands. I noticed it after using the stethoscope. I noticed it about the neck when the clothing was loosened that I might examine the patient. The skin also seemed to be a little roughened for a few days after the rash subsided.

"There have been practically no systemic symptoms. The highest temperature has been 100° F. and that only for one day. The appetite and digestion have been unimpaired and the patients have in no way felt sick. In ordinary measles, at the height of the eruption, a temperature of 104° or 105° F. is the rule. Like wise the digestion is impaired and the patients feel sick."

German measles is almost never serious, although once in a great while the fever may go to 103° F. or more, and the eruption cause swelling about the eyes.

Complications and Sequelæ: Complications are very rare. Smith reported two cases of diphtheria in his series, but as diphtheria also was epidemic at the time the combination was probably quite accidental. I have seen no complications.

Diagnosis: The differentiation of rötheln from measles and scarlet fever in typical cases is not difficult. In doubtful cases the following points are to be remembered:

Measles has an initial coryza with fever lasting for a day or two. This improves, then all at once the rash appears, first on the face, thence extending over the body and extremities. When the rash appears the fever goes up. Other points have been noted above.

Scarlet fever has a sore throat and swollen glands. The rash appears in twenty-four hours, on the body first—it rarely attacks the face. Fever and pulse rate are always high, indicating severe systemic disturbance.

Rötheln has slight prodromal symptoms—slight swelling of the cervical glands that may escape attention. The throat is slightly sore. The rash appears first on the face. The patient usually feels good throughout. Temperature and pulse are very little affected. The sore throat resembles scarlet fever, but the systemic affection is slight. The rash resembles measles, but there is no initial coryza, no respiratory symptoms.

Sometimes a simple roseola without other symptoms appears and is mistaken for German measles.

Prognosis: The prognosis is always good.

Treatment: Patients should be isolated as is the case in other exanthematous diseases. It is just as well to have the patient remain in bed for two or three days, and when the rash has gone to give him a warm bath. If desquamation should appear, as it does very rarely, keep the patient isolated till it is ended.

Aconite is useful at the very beginning.

Phytolacca is the best remedy for the glandular condition and if the sore throat is troublesome.

Pulsatilla I have had occasion to use if catarrhal symptoms were present.

SMALL-POX.

(*Pox*, spotted.) Syphilis was called *pox* or *great-pox;* this disease *small-pox* to distinguish it from syphilis.

Synonym: Variola (Lat., *varius*, spotted).

Definition: An acute infectious disease, characterized by an eruption which begins as a papule and passes through the stages of vesiculation, pustulation and crustation.

Historical Note: There is evidence that small-pox existed in China and India long before the Christian era. The first known description of the disease is by Rhazes, an Arabian physician, who wrote his account during the tenth century.

Etiology: A given case of small-pox always comes from contact—either direct or indirect—with some preceding case. It is the most contagious disease known. Before the era of vaccination small-pox attacked nearly every one, and was a children's disease, as are measles, whooping-cough, and other diseases to-day.

The exact nature of the virus is not known. Whatever it is, it is given off in the discharges of the body, and is present in the eruption from the stage of vesiculation on to the end. It may be transmitted not only directly, from person to person, but by a third person, by fomites, or may be air-borne for a considerable distance—a mile or more. It is supposed to enter the system by way of the respiratory tract.

No age or sex is exempt. There are very rarely persons who are not susceptible. The late Dr. Bryant, for several years in charge of the small-pox hospital of the New York City Department of Health, was never able to successfully vaccinate himself, nor did he ever contract the disease.

Morbid Anatomy and Pathology: The changes that take place in the skin in small-pox appear first in the epidermis. The spots begin as serous exudations, which later become cellular. As the exudations increase the reticulum in which they are contained ruptures and pustules result. If the exudate extends into the corium, scarring results. If it does not, scarring is avoided. As the pustules subside absorption of the liquid portion takes place and the outer part exfoliates. Complete evolution of a given lesion requires about two weeks.

Similar changes, in lesser degree, occur in the mucous membranes. Associated with the eruption are edema and redness of the surrounding skin.

Symptoms: Incubation lasts five to twenty days. Invasion occurs at the end of the incubation period. The disease is ushered in by a chill or chills, rise of temperature to 103° or 104° F., or higher, severe backache and aching all over. The pulse and respiration also increase. Headache and vomiting are common. In children convulsions are frequently present. The second day the temperature is somewhat higher. Prostration is marked.

At this time there is in some cases an initial eruption that may resemble the rash of scarlet fever or of measles. Osler found it in 13% of his cases.

Eruption: On the third day succeeding the abrupt onset the true small-pox eruption appears, first on the forehead and face and about the mouth. It is said that the forehead presents a

shotty feel to the finger before the eruption becomes visible. During the next twenty-four hours the eruption extends to the scalp, arms and hands, thence over the body and down the legs, reaching its height in two or three days.

The eruption appears first as small papules with a hard shotty feel. After three days the papules begin to change to vesicles, and by the fifth day all are vesicles. They contain a clear fluid, and are round with a depressed top—umbilication—which is characteristic and a diagnostic point.

During this stage the temperature, pulse and respiration go down and the patient feels comfortable.

About the sixth day the contents of the vesicles become pus-like, the change taking place in the same order as does the original appearance of the rash; namely, beginning on the forehead and face. The pustules become rounded up—that is, the top is no longer depressed. The skin adjacent to them becomes red and edematous, so much so that the features become almost obliterated in the general swelling.

At the same time, with pustulation, the fever returns, reaching 103° F. or higher. The patient may become delirious. At this stage the eruption also appears on the mucous membrane of the mouth and throat.

If the case is mild, after another day or two, the pustules begin to dry up and the general symptoms to subside. The eruptive development is usually ended in about two weeks.

From now on dessication takes place, a process lasting from two to four weeks. The pustules break and crusts are formed which dry up and disappear. There may be intense itching with this. The skin is left in a more or less mottled condition which eventually disappears. If the eruption has been deep enough to reach into the corium, pitting is left, otherwise the skin becomes smooth.

Varieties: The above describes the ordinary case of small-pox, *variola vera.*

The *confluent* form is more severe and the pustules run together.

In the *hemorrhagic* form the pustules contain blood.

In the *purpuric* form the eruption becomes purpuric, the patient becoming dark purple in color. Hemorrhages from the stomach and lungs are frequent.

Both the hemorrhagic and the purpuric forms of small-pox are almost invariably fatal.

Varioloid is the name given to very light attacks of the disease, where the papules are very few and scattered. It occurs mostly in those who have been vaccinated.

Complications and Sequelæ: Besides the pitting the convalescent from small-pox may develop boils and abscesses. Conjunctivitis may follow, likewise middle ear disease. Erysipelas may also be a sequel. In a few cases gangrene of the scrotum ensues, usually a fatal termination.

Diagnosis: Mild cases have been mistaken for chicken-pox. This is an unfortunate mistake to make, because the most severe hemorrhagic form of small-pox may develop from infection by a mild case. In chicken-pox there are usually no constitutional symptoms of note. The eruption is most numerous on the back and in the scalp. Spots appear in all stages of development at the same time. The papules are not hard. Dessication leaves reddish spots. The onset of small-pox is sudden, headache and backache are severe, there is vomiting. The papules have a shotty feel.

In measles we have Koplik's spots, the early appearance of a rash on the buccal mucous membrane, which are absent in small-pox. In measles the temperature rises with the onset of the rash, in small-pox it falls.

In scarlet fever the onset may be somewhat like small-pox, but it also has sore throat, which small-pox does not have. The temperature stays up in scarlet fever, whereas in small-pox it falls as the rash appears. The small-pox rash in its development is different from that of measles or scarlet fever.

A few other skin lesions may resemble small-pox at certain stages; for example, certain syphilitic rashes and impetigo contagiosa. The history of the case should serve to differentiate them. Impetigo is especially mentioned by the *Monthly Bulletin of the New York State Department of Health,* of May, 1912.

The prodromal headache and fever, and the appearance of the eruption on the face, forearms and hands, should always cause a suspicion of small-pox.

Prognosis: In the unvaccinated small-pox is a quite fatal disease. Statistics of different epidemics show a mortality of 15 to 50 per cent. in the unvaccinated; whereas, it is very much less in the vaccinated, from 2 to 10 per cent.

In a given case, the severity of the symptoms and the quality of the rash, especially on the head and face, are said by Welch to be the best indications of the outcome. Very severe cases are most apt to be fatal.

The very young and the very old are very liable to die.

Treatment: *Prophylactic*: Vaccination should be done on all persons supposed to have been exposed to small-pox. Even if done during the incubation period, it is said to either prevent or lighten the development of the disease. For the technique, the reader is referred to the next chapter on vaccinia. Vaccination is not an absolute preventive, as is shown by Mac Combie, (Allbutt's System of Medicine, 2d edition) in his article on small-pox. From 1889 to 1904 there were reported 10,997 cases in persons who had been vaccinated with a mortality of 7.6 per cent., and 3,223 cases in persons who had not been vaccinated with a mortality of 24.7 per cent. Mac Combie claims that the size of the vaccination scar indicates in a general way the prophylactic value of the vaccination, a large scar meaning that the bearer is better protected than he would be if the scar were smaller.

Some of the members of the homœopathic profession have used what is called "internal vaccination" with success. That is, the vaccine has been potentized and given internally, the 30th usually. This has been made a legal method in Iowa, fulfilling the requirement that all school children should be vaccinated. *Variolinum* has likewise been utilized in the same way. Raue speaks of the internal administration of ***melandrinum*** as a preventive of small-pox. It was introduced by Dr. Boskowitz, and was used successfully by Dr. Raue in 1880-81.

Small-pox is the most contagious disease known. It is trans-

missible from the beginning of the eruptive stage until the skin has returned to its normal condition—a period of four to six weeks. The exact contagion is not known, although forms of protozoa have been put forward by various observers as the specific cause. Whatever it may be, the contagium is in all the discharges of the body and in the skin lesions. It is therefore imperative that all discharges be promptly destroyed, and that all clothing used by the patient be promptly disinfected, and all dishes should be boiled after use.

The nurse must also be careful to disinfect her own clothing and to disinfect herself before coming in contact with other persons.

The care of the patient during the eruptive stage is especially important. It means attention to the skin in all cases. It means attention to the eyes. In the severe forms of the disease the mouth and throat are involved and require special care. If the patient becomes delirious he must be kept from injuring himself by tearing at the affected parts.

The best local application to the skin seems to be cloths, wet with boric acid solution. Other mild antiseptics may be used. This relieves the itching and irritation.

Boric acid solution should also be instilled into the eyes. Where the mouth is affected frequent swabbing with boric acid solution will keep it clean. If the mouth is dry, Mac Combie recommends a solution of boric acid in glycerine to moisten it after swabbing.

The odor from the patient is frequently disagreeable and the antiseptic applications relieve it somewhat.

The crusts on the head and face are best removed by linseed meal poultices (Mac Combie). A thin poultice is applied by means of a lint mask with openings for the eyes, nose and mouth. The surface of the poultice is covered with a little vaseline mixed with iodoform. The poultice should be changed every two hours. Other parts of the body must be kept moist with the boric acid solution. Warm baths may be given of half to one hour in duration.

If the patient becomes delirious it may be necessary to muffle the hands to keep him from scratching himself.

The diet should be liquid. Welch recommends two to three quarts of milk and three to six raw eggs daily during the height of the disease. Alcohol may be necessary as a stimulant.

Cracked ice is usually pleasing and soothing to the patient. Lemonade can be used for the thirst. If swallowing becomes painful, both Mac Combie and Welch recommend painting the fauces with a weak solution of cocaine a few minutes before feeding.

The dominant school of medicine uses tonics and stimulating remedies to carry the patient through.

Raue recommends homœopathically, *melandrinum* 30. He used it in an epidemic in 1880-81 and speaks from personal experience.

Vaccininum and *variolinum* are also recommended by him. He claims that each of these remedies has a specific action.

Antimonium tartaricum has the skin symptoms. Dr. J. H. Storer has used it successfully.

Apis is useful for the swelling if very pronounced.

Belladonna is indicated for cases with much sore throat.

Stramonium for the delirium when it occurs. Among other remedies *arsenicum album, crotalus* and *phosphorus* may be called for.

VACCINIA.

(Latin, *vacca,* cow. *Vaccinus,* from cows.)

Synonym: Cow-pox.

Definition: The symptoms produced by the accidental or intentional introduction into the body of vaccine virus.

Historical Note: In 1796 Jenner announced his discovery that persons who had suffered from cow-pox seemed to be immune to small-pox. He put his knowledge to practical use by artificially inoculating people. That was the origin of present-day vaccination. At one time vaccination was done from man to man. At present the preference is for glycerinated calf-lymph. The whole subject of vaccination is treated very fully in Allbutt's System of Medicine, second edition, by Ackland, Copeman, and McVail.

Internal vaccination, that is, the taking by the mouth of the

potentized vaccine virus, *variolinum*, or *melandrinum*, has already been mentioned in the chapter on small-pox. The thirtieth potency of each of these substances has been used successfuly by some homœopathic physicians as preventives of small-pox. This method of vaccination has not been generally accepted. It is legal in Iowa.

The Usual Method of Inoculation: Vaccine virus in my early days was dried on ivory points. The part to be inoculated, usually the arm, sometimes the leg, was cleansed, then it was scarified with an instrument made for the purpose, and the vaccine-covered point rubbed thoroughly in the bloody scarified surface.

The most satisfactory method now is the use of a common needle as a scarifier, and an ordinary wooden toothpick with which to rub in the glycerinated lymph.

The part to be inoculated should be cleansed first with soap and water, then with alcohol. A few scratches should be made crosswise over a quarter to a half square inch of surface, and deep enough to draw bloody serum. Then the lymph should be dropped on and rubbed thoroughly in with the tooth pick. Needle and toothpick should then be thrown away and not used again. The part should be allowed to dry thoroughly before being covered. A gauze covering may be worn over the inoculation.

The inoculation should be done with aseptic precautions. A simple gauze covering, loosely applied, is the best protection. Most of the commercial "shields" are unsatisfactory because they have stiff rims with a small opening, and do not allow sufficient room for the swelling that frequently appears in a successful vaccination.

The method outlined above was the one used by the Health Department of the City of New York while I was Medical Inspector, 1897-1901, and was very satisfactory.

Symptoms: Very rarely a person is found who is not susceptible to the vaccine virus and no symptoms develop after inoculation. The late Dr. Bryant, at one time in charge of the New York City small-pox hospital, and whom I personally knew,

was one. He took personal charge of all the small-pox cases at that hospital for several years, and he had done the same thing in Boston before he came to New York. He never contracted small-pox, and he never was successfully vaccinated.

As a rule, after inoculation, no symptoms develop for several days. Then one or more small red papules appear at the site of the inoculation. About the sixth day these become vesicles. If more than one they may remain separate or they may unite as one multilocular vesicle. These vesicles are usually umbilicated. By the tenth day the vesicles have become pustules. These in turn may be umbilicated.

In some cases, beginning with vesiculation, a reddish areola of indurated tissue appears around the site of inoculation of varying width. This and the pock itself may be sensitive and painful.

At the end of the third week, sometimes later, the scab tends to come off. This may not take place for six weeks, as in one of my patients. After removal of the scab the skin underneath is found to be pinkish in color. After a few months this scar has become white with a few pits—the well-known vaccination scar.

In many cases there is some malaise, the patient is feverish, and the appetite disturbed.

Complications and Sequelæ: The above describes the typical case. In a few instances the general symptoms are more marked, and the site of inoculation may become erysipelatous or even slough out. When either happens it is probably due to infection with elements other than the vaccine virus. I had one case where an erythema appeared on the arm and forearm and the axillary glands were swollen for a few days. Happily, with aseptic precautions and the use of glycerinated lymph, such accidents are becoming very infrequent.

Very young infants, or very ill patients, or persons in a badly run down condition, should not be vaccinated.

Vaccinal Rashes: In a small proportion of cases various rashes complicate vaccination. Personally, I have never seen any, but considerable space is devoted to their consideration in the various extended articles on vaccination.

First, there may be accidental inoculation in parts other than the original vaccination site. In such cases the virus is usually transferred by scratching. In other cases it seems to become general through absorption.

Second, various rashes may follow vaccination, similar to those that sometimes follow inoculation of the antitoxines;—urticaria, erythema, papular and other eruptions.

Third, vaccination may be followed by impetigo, pemphigus, erysipelas, or purpura.

Jacob Sobel reported 2 per cent. of various general eruptions in a series of 4,160 children vaccinated at the Good Samaritan Dispensary in New York in 1897-99. He considered only those cases that developed before the vaccine inoculation was healed. The urticarial and erythematous rashes appeared early as a rule, —within five days of inoculation. The other forms later, up to five weeks.

Finally it must be remembered that the various skin lesions and rashes may coexist in vaccinated children, and yet not be caused by the inoculation.

Diagnosis: A few words must be said about this because the physician may be called on to express an opinion about an isolated eruptive spot that has been due to accident. The vaccine eruption like the small-pox eruption runs through the various stages of papule, vesicle which becomes umbilicated, pustule, and formation of scab which dries up and falls off.

Impetigo contagiosa, when the crust is present, resembles somewhat the vaccination mark at the same stage. It occurs mostly in children and a number of cases in the same school or among playmates is the rule.

One physician of my acquaintance was much alarmed at a spot on his forearm because he feared the eruptive spot was a chancre at first. Its evolution proved it to be vaccinia. He had accidentally inoculated himself.

Treatment: The site of vaccination should be protected until the scab has come off. As noted above, gauze is the best covering, loosely applied. If there is much oozing of matter, as there is sometimes, peroxide of hydrogen may be used as a cleanser.

If the wound becomes infected, it must be made clean and kept clean.

Any complications that may arise will have to be treated according to circumstances.

CHICKEN-POX.

Synonym: Varicella. (Diminutive of *variola,* small-pox.)

Definition: An acute contagious disease occurring usually in children, and presenting a discrete vesicular eruption that appears in successive crops. Systemic symptoms are slight or absent. The vesicles burst in four or five days, sometimes leaving slight pocks.

Historical Note: Chicken-pox and small-pox were formerly considered as one disease. It was not until 1767 that Heberden differentiated them. Since then they have been considered as distinct. An attack of one does not protect from the other. Cases of a second attack are exceedingly rare in either.

Etiology: Chicken-pox is almost exclusively a disease of childhood. It attacks both sexes alike. It is very contagious, exposure to a preceding case is always necessary. The specific virus is not known, though several protozoa have been described by different observers as being the causative factor.

Symptoms: The incubation period varies from ten to twenty days, usually about fourteen. In February, 1910, a colored boy in my service at the Metropolitan Hospital broke out with chicken-pox a few days after admission. He was promptly isolated. In just two weeks two other colored children who were in the ward came down with it.

The eruptive stage rarely has any prodromal symptoms. Occasionally there may be slight febrile symptoms. The appearance of the rash is usually the first symptom. It consists of small vesicles scattered here and there, principally on the trunk, sometimes a few on the face and always, in my experience, a few on the scalp. Macules appear first, which become vesicles in an hour or two. The vesicles may be very few, or there may be a hundred or more. A given vesicle appears, lasts two or three days, and then dries up. At the height of the disease vesicles

in all stages may be seen near together. Some of the vesicles may suppurate, most of them merely dry up. Scabs are found which come off after ten or twelve days, leaving a smooth red surface which rapidly becomes normal in color. A few of the spots may be pocked.

The disease lasts about ten days. General symptoms, fever, anorexia, etc., rarely occur.

Complications and Sequelæ: Rarely ulceration or gangrene may supervene. A few cases have been reported with an associated pneumonia or bronchitis. These complications add to the gravity of the case.

A swelling of the legs followed in one of my cases.

Diagnosis: Chicken-pox is rarely difficult to diagnose, although occasionally it has been mistaken for small-pox, and mild cases of small-pox have been mistaken for it. Small-pox has usually severe prodromata, fever, headache, backache, and so on. Chicken-pox has few or no prodromata. The eruptive stage of small-pox has a lowering of temperature which returns as the vesicles maturate. Chicken-pox rarely has fever. The vesicles of small-pox become umbilicated and purulent, the vesicles of chicken-pox are clear, may become cloudy, rarely umbilicate and rarely become purulent. In small-pox the face is a favorite location for the eruption, in chicken-pox the trunk and scalp. In small-pox the vesicles frequently run together, in chicken-pox almost never.

Prognosis: The prognosis is always good, unless in the rare cases of complications.

Treatment: The child should be isolated to prevent further spread of the disease. Small children are best kept in bed. The diet should be light and nutritious.

Remedies are rarely necessary. *Antimonium crudum, antimonium tartaricum, apis, phytolacca,* and *rhus toxicodendron* may all be thought of.

MUMPS.

(*Mumps* also means sullen or sulky; and *mum,* speaks through a mask.) The disease is therefore well named.

Synonym: Parotitis.

Definition: Mumps is a contagious disease, consisting of swelling of the parotid, sometimes of the other salivary glands, and accompanied by fever. Occasionally metastasis to other glands occurs, particularly to the testicles, mammæ and ovaries.

Historical Note: Mumps is said to have been described by Hippocrates. According to Ruhräh, Mangor was the first to assert the contagiousness of mumps in 1773.

Etiology: Mumps is very contagious although the specific poison is unknown. It may be conveyed directly or indirectly. It is usually a disease of childhood, although adults may be attacked. My oldest case was a married woman, aged 27. In an epidemic in the apartment-hotel where she lived the disease apparently was brought in by a maid. From this maid others sleeping in the servants' quarters contracted the disease. These other maids in turn spread the infection to the guests in the various apartments of which they had charge.

Morbid Anatomy and Pathology: There is much swelling of the affected gland for a few days, but no permanent changes are left.

Symptoms: The incubation period is, on the average, about three weeks. It may be less, rarely more.

Usually the disease is ushered in with a certain amount of febrile disturbance—101° F. or more. There may be headache and vomiting. Sometimes the swelling is the first symptom noticeable and the systemic disturbance is co-incident with it.

First one parotid gland becomes swollen, tender, and painful. After a day or two the other one becomes swollen. Sometimes the second side does not swell till the first is well. Frequently the swelling extends down to the sub-maxillary glands as well. It may take four or five days for the swelling to reach its height. It begins under the ear, filling out at the hollow, then extends forwards and downwards, and backwards. The facial expression is lost and the patient is unrecognizable.

The patient cannot open the mouth very far, mastication is painful. Even swallowing may hurt. There is fever,—101° F. or more. The neck feels stiff. The patient is very uncomfortable.

The skin over the affected part may be slightly reddened, otherwise it is normal. Suppuration is very rare.

After the swelling has been at its height a day or two, it begins to subside, and in ten to fourteen days the condition is normal once more.

Complications and Sequelæ: The most frequent complication is a metastasis to the testicles in the male, or to the ovary or mammæ in the female. This is more or less serious; it may even result in atrophy of the gland affected.

Suppuration is very rare, that is, of the parotids.

Occasionally a deafness of one ear results.

Diagnosis: The diagnosis can usually be made from the appearance of the patient. Any other condition causing a similar appearance would have an entirely different history. Sometimes the parotid becomes inflamed and swollen as an end result of some infection.

Prognosis: The prognosis in uncomplicated cases is always good.

Treatment: The patient should be promptly isolated.

The diet should be liquid.

Of remedies, *belladonna* in the beginning may be of service for the swelling and for the temperature.

I have also found *phytolacca* to be extremely useful at all stages of the disease.

Mercurius is sometimes indicated where there is excessive salivation.

I have found *aconite* useful occasionally in the very beginning.

WHOOPING COUGH.

(So called on account of the peculiar whoop sometimes heard.)

Synonym: Pertussis. (*Tussis,* cough.)

Definition: A contagious respiratory catarrhal disease characterized by spasmodic coughing attacks ending in a so-called "whoop."

Historical Note: Whooping cough is an old disease, a description of which can be found (Keating) in the writings of Hippocrates, Galen and others of the early medical writers. It has been clearly defined and recognized as a distinct disease since the middle of the 17th century.

Etiology: The specific cause is the bacillus pertussis, recently described by Bordet and Gengon. The disease is contagious, and is supposed to be most so during the early catarrhal stage before the whoop becomes well-marked. The communicability persists until the end, but in a constantly lessening degree.

The virus is given off in the catarrhal secretions from the nose and throat. It may be carried by persons in contact with the disease, but who do not themselves have it.

Whooping-cough is essentially a disease of childhood. Younger children are more seriously affected than older ones. It sometimes occurs in adults. In the aged it is very serious and may cause death. My cases have ranged from 7 months up.

Morbid Anatomy and Pathology: In uncomplicated cases of whooping-cough the only pathological change is a catarrhal condition of the larynx, trachea, and perhaps of the bronchi.

Symptoms: The period of incubation is probably one to two weeks.

At first the child seems to be suffering from an ordinary catarrhal cold, with a cough and slight fever. The patient sneezes. The eyes and nose run. After a few days to a week or two the characteristic spasmodic cough begins. This consists of a number of expiratory coughs, with at the end a quick long drawn inspiratory sound which produces the so-called "whoop." This process may be repeated several times in succession. At the end of the paroxysm there is usually some expectoration. When the patient feels the attack coming on, if old enough, he will seize any nearby object for support, or run to the mother or other attendant. There is a look of fear and distress. With the expiratory coughs the air is forced out of the lungs to such an extent, and inspiration is held back so long, that the face becomes bluish or livid from excess of carbonic acid in the blood. The eyes become puffy. There may be hemorrhage from the nose and throat, rarely from the eyes and ears.

These paroxysms last from one to fifteen minutes, and may vary in number from one to a hundred in twenty-four hours. In children who are kept very quiet they are not apt to be frequent. Emotion and excitement, as well as physical exertion, or

even the act of swallowing may bring them on. Vomiting immediately after a paroxysm is frequent.

After persisting for a few days or weeks, the paroxysms of coughing begin to occur less frequently and to become less severe, till the characteristic "whoop" is gone.

An ordinary bronchial cough may follow and persist for weeks or months. I have seen such a cough last four months.

During the quiescent periods there may be no special physical signs in the lungs. During the paroxsyms and immediately following all sorts of noisy rales can be heard in the chest.

While in great distress during the paroxyms, between times the patient feels well and comfortable. The patient is apt to be worse at night.

Occasionally, more often in very young children, convulsions may occur.

The books speak of an ulcer under the tongue as being more or less constant. It occurs only in small children with one or two teeth and is due to protruding the tongue during the paroxsym. As a rule, there is not much systemic disturbance.

Complications and Sequelæ: Respiratory complications or sequelæ are the most frequent. A bronchial cough may persist for months. Sometimes pulmonary tuberculosis supervenes. Broncho-pneumonia is quite common and the chest should be frequently explored to detect signs of it. Lobar pneumonia sometimes occurs.

There are certain accidents that may occur as a result of the severe circulatory strain during the paroxysms. (1) Is collapse of the lung. (2) Hemorrhage from various parts, nose, throat, and so on. (3) Hemorrhage into the brain or cord causing any of the manifold symptoms—hemiplegia, aphasia, various paralyses—that may follow similar hemorrhage due to any other cause.

Middle ear disease sometimes occurs.

Finally we have the complications of the digestive tract—vomiting, sometimes diarrhea. The vomiting may be mechanical, or the vomiting and diarrhea may be caused by a true catarrhal condition due to swallowing discharges from the nose and throat.

Diagnosis: The diagnosis of a typical case of whooping-cough

is not difficult. Mild cases may be mistaken for ordinary simple bronchitis. The fact that the cough persists without marked physical signs may lead one to suspect whooping-cough.

Prognosis: In simple cases the prognosis is usually good, except in the very young or the very old. Complicated cases are more serious. Osler says that with its complications it is very fatal. There were 782 deaths from whooping-cough in New York State in 1909. It ranked eighth in the mortality list.

Treatment: The whooping-cough patient should be kept away from other children. If there is any fever the child should be put to bed. If the case is very mild this is not necessary. In any case, if the weather permits, the child should be kept in the open air as much as possible.

Mental and physical quiet are essential, because emotional or physical exertion will make the paroxysms of coughing more frequent.

The expectoration should be destroyed. Likewise any vomited matter should be promptly sterilized before throwing away. The contagion is in the respiratory secretion, and if vomiting occurs some of it is in that also, because the patient swallows it first.

Simple nourishing food and plenty of it is necessary to keep up the strength of the patient.

Application of an abdominal band has been recommended as a relief for the vomiting and for the paroxysms of coughing.

Bromoform 2 minims every few hours is recommended as a sedative.

My own observation leads me to believe that much can be done to lessen the severity of the symptoms by the use of the indicated homœopathic remedy. Many may be indicated. My preference is for *hepar* if the catarrhal symptoms are prominent.

Ipecac is the best remedy if there is much vomiting, or if broncho-pneumonia sets in.

Antimonium tartaricum where the cough is loose and rattling. The child chokes when he coughs and it makes him angry. The patient is worse at night.

Kali bichromicum is the remedy if the mucus raised is tough and stringy.

Belladonna may be called for if the cough is hard.

Pulsatilla is indicated if the catarrhal condition of the nose is prominent.

Sambucus is useful in the very beginning, with sneezing, much running of the eyes and nose, and cough.

INFLUENZA.

(Latin, *Influence,* thought to be due to the stars.)

Synonyms: Russian Fever. La Grippe of the French, so nicknamed on account of the severe hold it takes of the victim.

Definition: A severe form of catarrhal disease most often respiratory, characterized by prostration out of all proportion to its other symptoms.

Historical Note: True influenza is a pandemic disease which has swept over the civilized world at irregular intervals for the last four hundred years. According to Wilson (Pepper's System of Medicine) there are records of a few epidemics now supposed to have been influenza dating back to the ninth century. Most of the epidemics about which there can be no question have started in Russia or the far East, and have spread rapidly throughout eastern and western Europe, and thence to America. The last great epidemic reached the United States during the winter of 1889-90. This was followed by a milder outbreak the next winter. Since then there have been sporadic cases every year, with a few mild general epidemics.

Etiology: In the great waves of influenza that sweep throughout the country no age or sex is exempt. Nearly all are attacked without regard to condition or circumstances. It is said to be transmissible only through direct contact with the discharges.

Bacteriology: In 1892, R. Pfeiffer isolated a specific bacillus which has since been known as the bacillus influenza, or the bacillus of Pfeiffer. These are very small, with rounded ends and of varying length, sometimes growing into long forms, more or less bent or curved. (Wright and Mallory.) They stain more deeply at the ends than in the middle.

Morbid Anatomy and Pathology: There appear to be no char-

acteristic pathological changes in uncomplicated influenza, unless, indeed, in the respiratory forms a congestion of the bronchial tubes. In death from complications, of which various types of pneumonia are most frequent, the changes characteristic of the complication are found.

Symptoms: The period of incubation is probably not more than a day or two. The disease frequently runs through a household, passing from one member to another so rapidly that all who are attacked are laid up at once. In the epidemic of 1889-90 there were thousands of cases in New York city within two or three weeks of the appearance of the disease.

Influenza may be preceded by a day or two of malaise or a general tired feeling. More often it sets in somewhat suddenly. The patient may have a chill or chills accompanied by a rise of temperature to 100° to 103° F. or higher. The patient is completely prostrated. He is hot and feverish, and yet if he moves it makes him chilly. The pulse usually ranges from 120 to 140. The tongue is coated, there may be nausea, even vomiting.

In the *respiratory* type of the disease, the type most commonly met with, there may be sneezing and some coryza. The throat is apt to feel sore, and occasionally there is aphonia. After a day or two a cough develops, possibly accompanied by some expectoration. There is a bronchitis with a feeling of oppression in the chest. The patient may even feel some shortness of breath. Physical examination of the chest, however, reveals nothing more serious than a bronchial catarrh. In many cases this extends to a bronchial pneumonia.

In other words, this type of case is like a severe catarrhal cold with all the symptoms exaggerated, and the patient much more prostrated than the symptoms would seem to warrant.

The patient is apt to be drowsy, the sleep restless. He is blue and despondent, almost hypochondriacal. He is nervous and apprehensive. Occasionally there is mild delirium.

The temperature is typical of nothing. It runs along from 100° to 103° F., sometimes in severe cases to 104° or 105°, the pulse rate increasing with the height of the temperature.

The patient aches all over. His muscles are sore. This may

be so severe as to lead to a diagnosis of rheumatism. There may be neuralgic pains at different parts of the body. Occasionally there is stiff neck.

In a certain number of cases the respiratory symptoms are not so severe, but the disease seems to spend its force on the digestive tract, the *gastro-intestinal* type. In this type we always have nausea and vomiting. The tongue is badly coated. The abdomen is sensitive. There is diarrhea.

In still other cases the *nervous* system is most profoundly affected. In all epidemics cases of the *nervous* type are met with. Here the melancholy is profound. All the other symptoms may be slight or absent. The temperature may be normal, but the patient is apprehensive. He is tormented between a fear of death at one moment, and a desire to end the mental torture by committing suicide the next. Associated with this are very apt to be attacks of palpitation of the heart during which the patient is sure he is going to die.

The urine in all of the cases may be increased or diminished. Frequently there is an albuminuria.

In very young children and in very old people influenza is a most serious disease. Otherwise there is nothing characteristic about the symptoms.

Complications and Sequelæ: The most frequent complications and sequelæ are diseases of the respiratory system. Of these the various forms of pneumonia head the list. All epidemics of influenza show a marked increase in the pneumonic death-rate. The very young and the very old seem to be the greatest sufferers.

There is also an increase in the incidence of pulmonary tuberculosis following influenza. The histories of the cases at the Tuberculosis Infirmary of the Metropolitan Hospital show that a large proportion of the patients date their lung trouble from an attack of influenza, and conversely, a large number of cases of tuberculosis are killed by an attack of influenza. This was true at the old Ward's Island Homœopathic Hospital, where the writer was an interne during the epidemic of 1889-1890.

Another serious effect of influenza is its action on the heart.

As a cause of endocarditis influenza ranks with acute articular rheumatism and scarlet fever. I well remember the many cases of sudden death due to this cause at Ward's Island.

Still another frequent complication or sequel of influenza is middle ear disease. In the mild epidemic occurring in 1900 a vast number of the cases were followed by this troublesome condition.

Diagnosis: The diagnosis of a typical case of influenza is not particularly difficult if one bears in mind the aching and prostration and the irregular fever, the catarrhal symptoms, and so on.

The most frequent mistake will be in differentiating the common respiratory type from catarrhal pneumonia. It should not be forgotten that the two are very often associated. The physician should be on the constant lookout and carefully examine the lungs in either case.

Cases of influenza are sometimes confused with typhoid fever. In the latter there are the mode of onset, the typical temperature curve, the peculiar eruption; in influenza the onset is more rapid, the temperature curve is not typical, there is no eruption. In doubtful cases the Widal reaction should be looked for. Bacteriological examination will reveal the influenza bacillus in one case, the typhoid bacillus in the other.

Acute muscular rheumatism is a diagnosis that I made once, when I simply had a severe case of influenza to deal with.

In the nervous type the diagnosis must sometimes be made by exclusion.

Prognosis: The prognosis of influenza should be guarded. Uncomplicated cases nearly all recover, except, as noted above, in the young or the very old. The greatest danger comes from the complications. The physician should be on the constant watch against accidents arising from his neglect or carelessness. The chest should constantly be gone over during the course of the disease with a view to detecting any symptoms of pneumonic involvement, or of a threatening endocarditis. If any of these complications supervene the prognosis is more grave than it otherwise would be.

Treatment: Isolation is necessary to stop the spread of the

disease. It is supposed to be communicated by means of the nasal secretions mostly, so these should be destroyed and handkerchiefs handled in a careful manner. Clothes that could be destroyed as soon as used would be the ideal sort to use. Any sputa should be as carefully handled as in a case of tuberculosis.

The diet must be light. In the gastro-intestinal type of influenza, with nausea, vomiting and diarrhea, the nourishment of the patient may become quite a problem. Milk diluted with water or vichy may be tried. It may be necessary to resort to nutrient enemata.

The old school use phenacetin or some similar acting remedy to subdue the headache and other pains. Sometimes morphine is resorted to.

The homœopath has his indicated remedy which he can rely on with confidence.

Gelsemium heads the list for the ordinary type of influenza, the flushed face, the chilly sensations on the slightest movement even with high temperature. The aches and pains also call for it. Headache, dull and heavy is another symptom.

Bryonia is useful in the respiratory type with cough and stiff neck.

Ipecac for the gastro-intestinal type with nausea, vomiting and diarrhea. *Ipecac* is also a good remedy if catarrhal pneumonia complicates the case.

After a day or two of *ipecac* the remedy may frequently be changed with advantage to *pulsatilla,* especially if the patient is restless and cannot sleep.

Aconite will give relief in the nervous type, with great fear of death and melancholy.

Arsenic is of use later if the symptoms run an undue length of time.

Hepar will be of use in the middle ear complication.

Belladonna is called for if there is a congestive headache, the eyes red, and there is backache with chills.

Cimicifuga has headache at the back of the head and neck as a prominent symptom.

Kali bichromicum has much cough, with a thick, stringy mucus.

Nux vomica, the patient cannot sleep because his brain is too active. There is frontal headache, loss of appetite, sometimes nausea.

Many other remedies may be indicated.

LOBAR PNEUMONIA.

(Greek, πνευμων—lung.)

Synonyms: Croupous Pneumonia. Pneumonitis. Inflammation of the Lung. Lung Fever.

Definition: An acute inflammation of one or more lobes of the lung and supposed to be caused by the pneumococcus.

Historical Note: From the symptoms given in many of the descriptions of early epidemics, Loomis concludes that pneumonia was known in early historic times. It was frequently confounded with pleurisy. As pleurisy often accompanies pneumonia the two were not distinguished. Early in the 19th century, Laennec gave the first accurate description of pneumonia as such. He developed the science of auscultation, and taught how to differentiate lung diseases by the physical signs.

Fraenkel was the first to discover and describe the pneumonia diplococcus in 1883.

Etiology: The specific cause of pneumonia is supposed to be the pneumonia bacillus. The disease is therefore infectious and may be transmitted from one person to another. It is not at all unusual to find two or more cases in the same household, nor is it unusual for nurses to contract it.

The telephone as a possible source of infection in this as well as other infectious diseases should not be forgotten. A few years ago one of the officials of a large corporation in New York city was taken ill with pneumonia. A second official took up the duties of the first, moved to his desk and made frequent use of his telephone. Within a month the second man came down with pneumonia. A third man took up the duties at the same desk and used the same telephone. Within a few weeks he also developed pneumonia. Careful investigation failed to find any common cause applicable to the three other than the use of the same

telephone. A new instrument was gotten and the trouble ceased.

Exposure undoubtedly acts as a predisposing cause of pneumonia. The initial symptom is frequently a chill, and chilling of the body by exposure to cold is frequently followed by pneumonia. Exhaustion, either from exposure, or dissipation, or overwork, seems to make persons more susceptible to pneumonia. This is especially true of alcoholism.

Pneumonia is also a frequent complication during the course of certain other acute infections, as typhoid, influenza, measles and tuberculosis.

Pneumonia is found in both sexes and at all ages. Men suffer from it more often than women. Osler says it is the most fatal acute disease of the present day.

One attack seems to predispose to another. I have had many patients who have had pneumonia several times.

Bacteriology: The pneumococcus of Fraenkel is sometimes called the diplococcus pneumoniæ or the micrococcus lanceolatus. It gets these various names because it is found in practically all cases of croupous pneumonia, because it is found in pairs, and because each coccus is oval, conical or lancet-shaped. The broader ends of each pair are found in apposition. Frequently one is smaller than the other. Sometimes they form in chains, but are different in shape from the streptococcus. In pus, blood, and other media, a thin capsule seems to enclose each pair. In pure cultures this is absent. The pneumococcus is found in the sputa of pneumonia patients. At autopsy it is found abundantly in the lungs and may be found in the blood and in all the organs of the body. It is stained by Gram's method.

Morbid Anatomy and Pathology: The pathology of pneumonia is distinguished by four stages which run into each other or overlap. That is, if the patient dies at the height of the disease, different portions of the affected lung may be at different stages of pathological development.

Lobar pneumonia usually affects but one lobe of a lung, sometimes more.

1st stage—congestion. The affected part is congested and more red than normal. A cut section exudes blood and serum.

Crepitation is lessened. There is an exudation of leucocytes, red blood cells, fibrin and epithelial cells. The capillaries are swollen.

2nd stage—red hepatization. The air cells now have become filled with an exudate containing red cells, fibrin, and other materials so that the affected lobe is solidified. The adjacent pleura is usually coated with fibrin. The hepatized lung is swollen and increased in size. Sections of it are heavy and sink in water. The color is reddish.

3rd stage—gray hepatization. The exudate gradually becomes decolorized, changing from red to gray. The exudate begins to degenerate and soften.

4th stage—resolution. The softened exudate gradually liquifies and disappears, some of it by way of the expectoration—most of it by the lymphatics. The lung finally regains its normal condition.

The normal weight of the right lung is about 22 ounces, of the left 20 ounces. The pneumonic lung is much heavier, depending on the amount of tissue involved. One of my patients at the Metropolitan Hospital, an alcoholic, died of pneumonia. The entire right lung was consolidated, weighed 74 ounces; the lower lobe on the left side was consolidated, that lung weighed 32 ounces. The normal lung floats when put in water, the pneumonic lung sinks.

The other organs may be normal.

If any complications are present the characteristic lesion of the complication will, of course, be found.

Symptoms: The onset of pneumonia varies somewhat with the character of the case. The ordinary case in a vigorous person usually begins with a chill, followed by high temperature and flushed face. The patient is prostrated. The breathing becomes rapid and shallow. Sometimes there is acute pain right at the start. One patient of mine, a man, aged 65, was in his usual health apparently at 9 p. m. At 11 he had acute pain in the left side of the chest, breathing short and shallow, pulse hard and full. A few hours later physical signs of pneumonia were fully developed.

In other cases there is little or no pain, or if pain develops it is

later in the disease. Pain is supposed to indicate involvement of the pleura. Occasionally a patient will not give a history of chill, but will all at once be too sick to get out of bed. There may be no cough at first and no pain. The patient will have a temperature and the respirations will be markedly increased. I have seen a number of cases of this kind in persons previously of good health and correct habits. The rapid respirations called attention to the chest and physical examination revealed the condition.

In cases of delirium tremens the onset of pneumonia is usually insidious. The complication is so frequent that its development should always be borne in mind and frequent examinations of the lungs made in such patients.

After the initial chill the temperature rises abruptly to 103° to 105° F., sometimes higher. It is said to remain up for from five to seven days, when it drops by crisis. This is probably true under old school treatment as all the old school text-books give it that way. But under homœopathic treatment this is not the rule, at least in my experience. Many cases will have a fall of temperature by lysis. As stated at a meeting of the New York Homœopathic Materia Medica Society, under homœopathic treatment the temperature in pneumonia seems to melt away. The majority of cases that I have seen have had a diminishing temperature for two or three days. I have also seen the temperature fall by crisis. One patient, a girl of 14 years, taken ill Christmas Day, 1912. The temperature ranged from 104° to 105° F., and was at 105° at 11 p. m., December 31. At 9 a. m., January 1, the temperature was 97° and never rose above normal again.

An occasional case will have a pseudo crisis; that is, the temperature will drop to normal or nearly so, and then rise again within a few hours to stay up for several days longer.

The respirations are increased to 30 or 40 or more per minute. In severe cases the respirations may be as frequent as 60 to 70. In infants they may be still higher. I have seen 80 per minute with recovery. In one woman the respirations were 65 to 70 for six days; the patient recovered. As a rule respirations beyond 50 per minute are rare, and then only for a day or two. They may or may not be accompanied by pain.

Even with the rapid respirations dyspnea is unusual in pneumonia. The patient is not distressed for breath, he simply breathes rapidly.

The pulse rate does not increase to the same degree as the respirations, a pulse of 100 to 120 in pneumonia being the average. In the infant with respirations of 80, the pulse was only 120. In the woman with a respiration of 65 to 70 for six days, the pulse was at no time over 130.

One of the diagnostic signs of pneumonia is a change in the ratio between the respiration and pulse. In health this is as 1 to 4, or 1 to 4½. In pneumonia the ratio changes and approaches 1 to 2, sometimes 1 to 1½. In other words, in health, with a respiration of 18 the pulse would be 72. In pneumonia with a respiration of 40 the pulse probably would not be over 100; with a respiration of 60 the pulse would be 120, once I saw it as high as 160. In but one other condition does this marked change in ratio between respiration and pulse occur; and that is in some cases of hysteria during an attack. The other symptoms and physical signs should of course serve to differentiate the two.

After the crisis in pneumonia, or the lysis as the case may be, the pulse usually becomes slow, 60 or even 50 beats per minute for a few days.

Recently it has been stated that if the blood pressure in millimetres falls below the number of pulse beats per minute, during the height of the disease, the outlook is unfavorable.

If the endocardium becomes involved the pulse becomes irregular and the heart sounds become vague and obscure. The rhythm is disturbed. These are dangerous symptoms.

The cough of pneumonia is not characteristic, although always present at some stage of the disease. It may not begin until a day or two after the diagnosis is certain. It may precede other symptoms. The cough may be a slight hack, or it may be severe and frequent. It may be almost constant, it may come only in paroxysms. It may not become fully developed until resolution begins. Only once have I known it to be absent altogether.

On August 11, 1913, a man of 24 years was brought into my ward at the Flower Hospital. I made a diagnosis of pneumonia

of the left lower lobe on the physical signs. The temperature was 105° F. On the 15th the temperature dropped to normal to return the same day to 105°—a pseudo crisis. On the 20th a true crisis occurred. During most of this time there was delirium. The patient made an uninterrupted recovery and was discharged cured September 5. At no time was there any cough or expectoration. After the crisis the physical signs of pneumonia disappeared.

The expectoration in pneumonia also varies. Usually it is considerable, an ounce or two a day. At first whitish, it later becomes blood-streaked—the so-called rusty sputa. Occasionally the blood is considerable throughout the disease. I have seen one or two cases ushered in with quite a hemorrhage.

The blood presents a marked leucocytosis in pneumonia as a rule. The red cells may remain at normal or nearly so. Anemia is not marked. There is an excess of fibrin in the blood. After the crisis the leucocyte count promptly returns to normal and the red cells may be diminished.

Very rarely is there any rash. I had one case at the Metropolitan Hospital with a profuse typhoid-like eruption.

The mental condition of the pneumonia patient may be clear throughout. In some cases there may be stupor, in others a more or less active delirium. The patient may be restless, or there may be twitching. A few cases have such marked mental and nervous disturbance that it would seem as though the meninges were involved. Delirium tremens may develop in an alcoholic.

As in all severe diseases the digestion is impaired. The tongue is coated. The appetite is lost. The patient cannot eat solid food. The bowels may or may not be disturbed.

The urine is usually diminished in quantity. The color, acidity, and specific gravity increased. Urea is usually markedly increased, the chlorides diminished or even absent. Albumin may be present and the diazo reaction positive.

Pneumonia usually affects one of the lower lobes of the lungs, the right more often than the left. Sometimes it attacks an upper lobe. When this happens it is claimed that the case is apt to be more severe. I have not found it so. As a rule but one lobe is affected. Sometimes both lower lobes are affected. Sometimes

more than one lobe on the same side becomes involved. If more than one lobe is affected it adds to the gravity of the case.

Occasionally the pneumonic process is so deep-seated that at first it is difficult to get the physical signs. This is more apt to occur in the very young or the very old. In such cases the diagnosis must be based on the other symptoms. Rapid respirations, changed ratio between the respiration and pulse, and fever should put the physician on his guard, especially if these symptoms are associated with cough.

The patient is apt to lie on the affected side, although he may prefer to lie on the sound side. He is not apt to lie on the back.

Physical Signs: *Inspection* reveals increased frequency of respirations. These are shallow. There is diminished motion on the affected side.

Palpation also reveals diminished motion on the affected side. There is increased vocal fremitus over the affected area after consolidation takes place.

Percussion gives diminished resonance over the consolidated portion of the lung, and sometimes an increased resonance elsewhere.

Auscultation in the beginning of the disease discloses a fine crackling sound—crepitant rales—over the affected area during respiration. Later, as the lung tissue becomes solidified, the finer crepitation is lost, and the breathing sounds become coarser—bronchial breathing. Still later breathing sounds may disappear altogether.

The physical signs disappear in the reverse order as resolution progresses and the lung clears up.

Complications and Sequelæ: Pneumonia is itself a common complication in typhoid fever, measles and other acute infections. It is a frequent termination of tuberculosis.

Pleurisy is an almost invariable accompaniment of pneumonia. In fact the pain in pneumonia is supposed to be due to involvement of the overlying pleura. Sometimes the pleurisy goes on to effusion and thence to empyema.

Acute bronchitis, although not common in my experience, is a very annoying complication. The excessive coughing tends to wear out the patient.

Some cases undoubtedly run into tuberculosis.

Meningitis is an occasional complication.

In alcoholics delirium tremens is apt to develop. All cases of delirium in pneumonia are not delirium tremens, however.

The endocardium is not often involved, but when it is the condition is very serious.

Diagnosis: The distinctive features of pneumonia are the sudden onset, beginning with a chill; the high temperature; the rapid breathing, with the changed respiration-pulse ratio. The physical signs are pathognomonic, when well marked. The finding of the bacillus of Fraenkel in the sputum also makes the diagnosis sure.

Pleurisy starts with chilliness, rather than a distinct chill. The temperature does not go so high. The respiration-pulse ratio is not changed. Vocal fremitus is diminished instead of increased in case of effusion.

Pulmonary tuberculosis is of gradual onset. As a rule the trouble is in an upper lobe rather than in a lower. The temperature is not so high. The pulse rate increases rapidly as the respirations increase. The tubercle bacillus will be found in the sputum.

There is a condition described by Osler as "acute pneumonic phthisis." I have seen two cases of this disease, each of which ran the course of the disease and died in three weeks. These patients presented all the physical signs of pneumonia. But autopsy showed them to be tuberculosis.

Catarrhal pneumonia may act much like lobar pneumonia. As a rule in the catarrhal form the onset is less abrupt, there is chilliness rather than a distinct chill, the fever is not so high, the pulse is more rapid, the respirations may be fast. The physical signs will be the same, but the areas presenting them will be isolated spots and not entire lobes. Catarrhal pneumonia may travel from one place to another and is not a self-limited disease, it may run for two or three weeks.

Empyema will sometimes simulate pneumonia, clinically. I think it is more apt to be a sequel of pneumonia. That is, the patient has a true pneumonia which does not clear up, and after the

temperature has run along for two or three weeks, the aspirating needle will show pus.

Strange as it may seem, appendicitis and pneumonia are sometimes mistaken for each other for a day or two. This is true of right-sided pneumonia more particularly. Both may have chill, rise of temperature, and increased respirations. But the appendicitis case will have rigidity of the rectus muscle and possibly develop localized tenderness. The pneumonia case will develop the physical signs in the lungs.

Prognosis: About 10% of the cases of pneumonia die. It is more fatal in the very old and the very young. The vast majority of patients get entirely well eventually. One attack seems to predispose to another.

The great danger in pneumonia is the heart. Patients must not be allowed to get up too soon. They should not be allowed to sit up until the temperature has been normal for several days. Then if sitting up sends the pulse rate up materially they should be put back in the recumbent position for two or three days before trying to sit up again. It sometimes happens that when the temperature is normal and the patient seems well on towards convalescence, some extra exertion, like walking about the room, will cause sudden death, probably by embolism. It is necessary therefore to use the utmost care.

If endocarditis develops during pneumonia the patient is in a very grave condition, although it is not necessarily fatal. The heart should be carefully watched until the patient is entirely well.

Occasionally resolution is delayed, the fever continues at 101° F. or thereabouts, and it seems as though the patient had tuberculosis. These cases usually recover after some months.

Pulmonary tuberculosis sometimes supervenes.

In straight uncomplicated cases of pneumonia the patient is fully convalescent at the end of two weeks, and well in three.

Treatment: The best preventive treatment is to avoid doing anything that causes sudden chilling of the body, and to avoid exposure.

The discharges from a pneumonia patient should be destroyed

to avoid infecting others. It is not at all uncommon to have secondary cases due to lack of care in handling those already sick.

The pneumonia patient should be put to bed in a well ventilated room, or in suitable weather he can be put to bed out of doors, on a porch, or on a roof. If in a room there should be an ample supply of fresh air. If out of doors the head should be protected by a hood, and some sort of covering should be arranged to protect the patient's head from direct sunlight.

There should be a sufficient supply of bed clothes to keep the patient comfortable, without having enough to make him uncomfortable, either as to warmth or as to weight. The most suitable garment to wear next the skin is an ordinary woolen undershirt or under vest. This should be open its entire length so that the chest can be readily examined. It should also have sleeves that the patient may keep his arms out from under the covers without getting cold. Outside of this a night gown, also open, may be worn.

The so-called pneumonia jacket I only mention to condemn. Under no circumstances is it advisable. It is clumsy, it is unclean, it keeps the patient super-heated. On account of its super-heating qualities it is dangerous to the patient, because it cannot be removed to allow of examination of the chest or to change without producing a chill.

Antiphlogistine may be applied to relieve the pain. Care should be used to keep it from crumbling.

The patient should be given sponge baths two or three times a day to make him comfortable and to keep the temperature from getting too high. It has a good effect also in making the patient more quiet.

An ice cap may be used on the head in case of delirium. I do not like the ice water compresses sometimes recommended, and never use them. I believe them to be detrimental to the patients.

The diet should be liquid. Milk is the most suitable food. Koumyss, broths and soups may be of service. They may be given to avoid the monotony of milk, or they may be used where milk is not acceptable.

Water should be used freely. It should be given at regular intervals, even if the patient does not ask for it.

The old school authorities now use no medicines, except as stimulants. I am opposed to alcohol in this disease as a routine. I use it only in case of impending heart failure, and am in doubt as to its value even then.

Strychnine is also recommended as a stimulant. This is a drug that must be used with discretion—never in larger doses than 1/50 of a grain, and not oftener than once in three hours.

As a heart stimulant, for emergency use, camphor is the most satisfactory remedy that I know of. It may be given in drop doses of the ordinary spirits of camphor, on sugar, as often as every five minutes. Or camphor in olive oil may be used hypodermatically. Its action is evanescent, and it has to be given frequently to sustain the effect. After a few doses the emergency is past and something else may be used.

Digitalis is sometimes useful for more prolonged use—for days or sometimes weeks. Five to ten drops of the tincture may be given.

A most important part of the treatment is the indicated homœopathic remedy.

For the pneumonic process itself *ferrum phosphoricum* 6x is my first choice at the beginning. I think I have seen three cases aborted by this remedy when its administration was begun within a few hours after the initial chill. In each of these cases my diagnosis was confirmed by other physicians. The remedy was repeated every hour. It may be continued, beyond the first day or two, if there is much blood in the sputa.

Aconite in dilution may be given every hour, also in the beginning. I use the 3x potency. Sometimes I have alternated one or the other of the above with *bryonia*.

After the first twenty-four hours *bryonia* in dilution is the most useful remedy we have. It may be continued till the case is convalescent. I use the 3x, the dose repeated every two hours.

Phosphorus is recommended, but my experience with it is limited. It cured one case where aphonia was a symptom.

Ipecac is useful in children and in old people when the respira-

tions are very rapid, the crepitation very fine, and usually with some nausea.

Antimonium tartaricum cured one case where the cough was constant, there were large quantities of foul smelling expectoration, the respiration was rattling as though the patient was dying, the facial expression was bad, there was delirium, there was hot perspiration.

Ammonium carbonicum is useful when the pulse is weak, slow and irregular, and the patient suffers from dyspnea.

Kali bichromicum is indicated for an associated bronchitis with much cough and stringy or lumpy, tough expectoration.

Belladonna may be indicated in the beginning with delirium and hot, dry skin.

Natrum sulphuricum is indicated in cases with delayed resolution.

Hyoscyamus may be needed if the patient is delirious.

Sulphur 30 has cleared up some cases of unresolved pneumonia very satisfactorily.

After the pneumonic process has ceased and the patient is convalescent, *arsenicum album* will be found satisfactory as a support to the heart until the patient is entirely well. I use the 6x trituration every two to four hours.

Nux vomica may also be used during the period of convalescence as a mild tonic.

Many other remedies may be called for.

TYPHOID FEVER.

(Greek, τυφος, stupor.) So named because of the mental condition usual at the height of the disease.

Synonyms: Enteric Fever. Ileotyphus. The Typhus Abdominalis of the Germans.

Definition: Typhoid fever is a systemic disease due to the ingestion of the bacillus of Eberth. The typhoid pathological lesion is an inflammation of Peyer's patches in the intestines. The disease is of gradual onset. The fever is characteristic; there is a gradual rise for about a week, a continued high temperature

for two weeks or more, and a gradual decline for another week. There is usually diarrhœa, sensitiveness and gurgling over the ileo-cecal region, and an eruption on the abdomen. The spleen is enlarged. There is nearly always more or less mental hebetude.

Historical Note: Typhoid fever has probably existed since the beginning of medical history. Nevertheless, it was not accurately described as a separate and distinct disease until early in the nineteenth century. The name "typhoid" was first proposed by Louis, of France, in 1829. It was not much later that Gerhard, of Philadelphia, first differentiated between typhoid and typhus fever. Shattuck, of Boston, in studies abroad, the two Jacksons and Hale, of Boston, at the Massachusetts General Hospital, and Flint, Sr., of New York, did much to advance an accurate knowledge of the disease. In 1880 Eberth discovered the bacillus of typhoid. About 1895 the agglutination method of diagnosis, known as the Widal test, was first described. Late in the nineteenth and early in the twentieth centuries immunization against typhoid by the injection of antityphoid serum was tried extensively on troops in the field. Since 1911 this has been compulsory in the United States Army.

Etiology: Typhoid fever is caused by the ingestion of the typhosus bacillus of Eberth. It is supposed that the germ is always introduced into the body by way of the digestive tract. By far the commonest means of transmission is contaminated water. Certain epidemics have been traced to milk. Invariably further examination has shown the milk to have been contaminated from some water supply. Either the water was used to dilute the milk, or the water was used to wash the receptacles in which the milk was afterwards put. In the epidemic occurring in Stamford, Connecticut, in 1895, 406 cases developed, all of which were traced to milk from one dealer.

Infected water is always contaminated by excreta from a typhoid patient. For example, the famous epidemic at Plymouth, Pennsylvania, in 1885, which was traced to the water supply. In the preceding winter typhoid had appeared in a patient living on the banks of the stream from which the town was supplied. The excreta were thrown out on the frozen ground. In the

spring a sudden thaw came and these discharges were washed into the river. The epidemic promptly followed.

Another epidemic occurred in 1894 among some students of Wesleyan University and their guests at a fraternity dinner. The infection was traced back to some raw oysters that had been served at the banquet. These oysters had been fattened in water near the opening of a drain from a house where there were two typhoid patients.

Various insects may be the carriers of the contagion. During the Spanish-American War many cases of typhoid fever developed in the various camps. It was demonstrated that flies were the means of the spread of the disease in many cases. The flies swarmed about the sinks where they lived on human excreta, then they flew to the food and drink of the men where they deposited the germs.

Direct transmission of the typhoid germ through the air has not been demonstrated. Nurses and attendants who contract the disease probably do so by neglecting to take proper precautions with the discharges. They may inadvertently carry the infection on soiled hands, or on hankerchiefs, napkins, or what not, to the mouth.

The typhoid bacillus may persist in the soil or in filth for many months. It is probable that some untraceable cases may originate from bacilli-laden dust that gets into the food a long time after excretion, from some preceding case. Barringer, of North Carolina, called attention to a possible source of cases of obscure origin in 1904. He said many typhoid patients travel on the railroads throughout the country, some before going to bed, some after getting up, a few during the height of the disease. While traveling these persons are liable to have movements from the bowels of infected feces, which are distributed along the roadbed. He showed that trackmen were prone to typhoid, and believed they became infected from dust while in the performance of their duties. He was of the opinion that occasionally travelers were infected by the dust from the roadbeds in traveling.

Creel, of the Public Health Service, in 1912 showed experi-

mentally "that plants cultivated in contaminated soil will take upon the leaves and stems, as they grow through the soils, organisms existing therein." He found the typhoid bacillus on lettuce and radishes grown from infected soil where the plants appeared clean to the eye. Rainfall did not free the vegetables from the infected material. Under the most unfavorable conditions the bacilli remained alive after thirty-one days.

Another obscure source of contagion recently brought to the attention of the profession is the so-called "bacillus carrier." It has been shown that certain persons harbor the bacillus typhosus for many years after they have entirely recovered from the typhoid fever itself. The case of "Typhoid Mary" who was kept under observation by the Health Department of the City of New York for several years is a case in point. I had personal knowledge of the series of cases that finally caused her detention. Mary was a good cook. In the spring of 1907 she was employed by a well-to-do family, and a few weeks later accompanied her employers to their country home. Before the summer was over the mother and two children of the houeshold and four servants developed typhoid fever, all seven being ill at one time. The head of the family spent a large sum of money to trace the source of infection. Finally, the record of each of the servants was investigated, and it was found that the cook, although apparently in good health herself, for years had left a trail of typhoid fever wherever she had been employed. The microscope showed that her dejecta constantly carried typhoid bacilli. I might add that, although kept under supervision for several years by the Department of Health, the bacilli were never entirely got rid of.

Typhoid is most prevalent in the late summer and autumn. The majority of my cases, eighty per cent., were taken ill in the second half of the year, the other twenty per cent. in March, April or May. The sexes were about equally divided. My youngest patient was one year old, my oldest thirty-five. More than fifty per cent. were in the twenties. These percentages agree with the great mass of figures that have been recorded by numerous observers. The disease most often attacks youths and young adults,

though no age is exempt. The disease is not common in infants, and is very rare after sixty. At the extremes of life typhoid is apt to run an atypical course. As a rule, the same person has but one attack. Except, that quite a number of patients have a relapse immediately after the beginning of convalescence, going through the whole cycle of symptoms, a second, more rarely a third time.

Bacteriology: The immediate cause of typhoid fever is supposed to be the ingestion of the bacillus typhosus discovered by Eberth in 1880. This bacillus is about one-third as long as the diameter of a red blood corpuscle. It is about three times as long as it is wide, and with rounded ends. Occasionally it may be much longer, thread-like. Very fine fibrillæ have been discovered at the ends, and are supposed to be motile organs. The bacilli are very active in their movements. They frequently present marked areas in their protoplasm. They grow on gelatin in a broad translucent streak with wavy margins. The gelatin is not liquified. On potato the growth grows quite rapidly, covering the surface. The growth is invisible, but can be felt by drawing a needle across the cut surface.

The bacillus typhosus may be found in the blood early in the disease. Coleman and Buxton presented an analysis of six hundred and four cases in which the blood was examined for the typhoid bacillus. It was present in 93 per cent. in the first week, 76 per cent. in the second week, 56 per cent. in the third week, and 32 per cent. in the fourth week. The bacilli were more numerous at first, and gradually diminished as the disease progressed.

The *Widal reaction*, on the contrary, develops later, rarely before the end of the first week. This is obtained by taking a drop of blood serum from a typhoid patient, and adding ten drops of a fresh bouillon culture of the typhoid bacillus. Examined with a high power lens, the bacilli are seen to lose their motility and congregate in clumps. If this reaction does not take place in twenty minutes, it is said not to be present.

The typhoid bacillus may be found in any part of the body. It is especially numerous in the spleen, intestinal lesions, feces,

more than one lobe on the same side becomes involved. If more than one lobe is affected it adds to the gravity of the case.

Occasionally the pneumonic process is so deep-seated that at first it is difficult to get the physical signs. This is more apt to occur in the very young or the very old. In such cases the diagnosis must be based on the other symptoms. Rapid respirations, changed ratio between the respiration and pulse, and fever should put the physician on his guard, especially if these symptoms are associated with cough.

The patient is apt to lie on the affected side, although he may prefer to lie on the sound side. He is not apt to lie on the back.

Physical Signs: *Inspection* reveals increased frequency of respirations. These are shallow. There is diminished motion on the affected side.

Palpation also reveals diminished motion on the affected side. There is increased vocal fremitus over the affected area after consolidation takes place.

Percussion gives diminished resonance over the consolidated portion of the lung, and sometimes an increased resonance elsewhere.

Auscultation in the beginning of the disease discloses a fine crackling sound—crepitant rales—over the affected area during respiration. Later, as the lung tissue becomes solidified, the finer crepitation is lost, and the breathing sounds become coarser—bronchial breathing. Still later breathing sounds may disappear altogether.

The physical signs disappear in the reverse order as resolution progresses and the lung clears up.

Complications and Sequelæ: Pneumonia is itself a common complication in typhoid fever, measles and other acute infections. It is a frequent termination of tuberculosis.

Pleurisy is an almost invariable accompaniment of pneumonia. In fact the pain in pneumonia is supposed to be due to involvement of the overlying pleura. Sometimes the pleurisy goes on to effusion and thence to empyema.

Acute bronchitis, although not common in my experience, is a very annoying complication. The excessive coughing tends to wear out the patient.

Some cases undoubtedly run into tuberculosis.

Meningitis is an occasional complication.

In alcoholics delirium tremens is apt to develop. All cases of delirium in pneumonia are not delirium tremens, however.

The endocardium is not often involved, but when it is the condition is very serious.

Diagnosis: The distinctive features of pneumonia are the sudden onset, beginning with a chill; the high temperature; the rapid breathing, with the changed respiration-pulse ratio. The physical signs are pathognomonic, when well marked. The finding of the bacillus of Fraenkel in the sputum also makes the diagnosis sure.

Pleurisy starts with chilliness, rather than a distinct chill. The temperature does not go so high. The respiration-pulse ratio is not changed. Vocal fremitus is diminished instead of increased in case of effusion.

Pulmonary tuberculosis is of gradual onset. As a rule the trouble is in an upper lobe rather than in a lower. The temperature is not so high. The pulse rate increases rapidly as the respirations increase. The tubercle bacillus will be found in the sputum.

There is a condition described by Osler as "acute pneumonic phthisis." I have seen two cases of this disease, each of which ran the course of the disease and died in three weeks. These patients presented all the physical signs of pneumonia. But autopsy showed them to be tuberculosis.

Catarrhal pneumonia may act much like lobar pneumonia. As a rule in the catarrhal form the onset is less abrupt, there is chilliness rather than a distinct chill, the fever is not so high, the pulse is more rapid, the respirations may be fast. The physical signs will be the same, but the areas presenting them will be isolated spots and not entire lobes. Catarrhal pneumonia may travel from one place to another and is not a self-limited disease, it may run for two or three weeks.

Empyema will sometimes simulate pneumonia, clinically. I think it is more apt to be a sequel of pneumonia. That is, the patient has a true pneumonia which does not clear up, and after the

The classical temperature curve shows a daily fluctuation of two or three degrees for the first week. Each morning the temperature is a degree or so higher than it was the preceding morning; each evening a degree or so higher than on the preceding evening. This part of the temperature curve is rarely seen by the physician, because during the first few days the patient does not feel sick enough to call in the doctor, or, if he does, the taking of the temperature is often neglected. This typical "step-ladder" rise also takes place when the patient has a relapse.

By the end of the first week the temperature has attained a height of 103° to 105° F., and with a daily fluctuation of one or two degrees remains there during the next ten days to two weeks.

Then it begins to fall, going a little lower each morning, and nearly to the maximum each evening. During this phase of the disease the difference between the morning and evening temperatures is considerable, and may be even as much as six degrees. This period of wide fluctuation lasts several days to a week, the variation becoming less and less, until at the end the temperature is normal. In other cases the temperature is lower each morning than it was the preceding morning, and lower each evening than it was the preceding evening until the normal is reached.

In case of relapse—which usually sets in within two weeks after the normal is reached, if at all—the entire cycle is repeated. A rise of temperature is indeed the first indication of relapse, so that it should continue to be recorded after it has dropped to normal, and until the patient is fully convalescent.

The pulse presents nothing characteristic. It usually runs 100 to 110, not very rapid. Sometimes it is dicrotic. This used to be set down as a diagnostic point, but I have not seen it often enough to consider it of very much importance. Late in the disease the pulse may become very weak. This is due to weakness of the heart muscle.

As in all of the acute infections, the heart must be carefully watched from day to day. Endocarditis is not common in typhoid, but a degeneration of the heart muscle is. When the patient becomes convalescent, sitting up will cause a rapid in-

crease in the rate of the heart beat at first. This has to be regulated by continued rest. But the pulse is apt to be more or less accelerated on very slight exertion for several months or a year.

The eruption of typhoid may be absent. As a rule, however, there are a few reddish spots on the abdomen. They may be so few as to require careful searching to discover them, or they may be profuse. When the spots are numerous they spread over the trunk and extremities. Very rarely they may appear on the face.

The spots are usually very small—about the size of an ordinary pin head. They appear in crops beginning toward the end of the first week, and each crop lasting two or three days. When present the eruption is quite an important diagnostic sign.

It is a good plan, if the spots are few, to outline them with an indelible pencil each day. In this way new ones will be recognized, and the length of stay of old ones can be noted.

The digestive disturbances in typhoid are usually marked. Loss of appetite is the rule during the active stage of the disease. After convalescence sets in the patient is often ravenously hungry. Nausea and vomiting are rare. When they do occur they are of serious import. Constipation is the rule in the beginning. At the end of the first week diarrhea is the rule. This latter may become a very troublesome symptom. The characteristic stool is of the consistence of "pea soup." The stools may be involuntary. Rarely constipation persists throughout the course of the disease. Some tympanites may appear. All through the disease the tongue is apt to be coated. In severe cases the coating becomes dark. If extreme care is not exercised sordes will appear on the teeth.

After the fever has reached its height there may be hemorrhages of the bowel, due to rupture of a blood vessel in one of the ulcerated Peyer's patches. If the hemorrhage is slight the only evidence may be blood in the stools. If severe, the patient will become suddenly exhausted and go into a state of collapse. Even in such cases it may be several hours before blood is found in the stools. A sudden drop in temperature is always very

significant. If due to severe hemorrhage the pulse becomes very weak and rapid, the patient becomes colorless, and gasps for breath. The extremities become cold. The temperature drops suddenly.

If perforation of the intestinal wall occurs the patient may also become collapsed. But the tendency is for the temperature to shoot upward. Tympanites appears suddenly, and if the patient survives a few hours peritonitis develops. The abdomen becomes distended and painful.

The spleen is enlarged in typhoid as in numerous other conditions. Although taken by itself this is not pathognomonic, yet it is a valuable contributing sign.

Nose bleed is very common during the onset of typhoid.

Bronchitis is usual, so much so that it is rather a symptom than a complication. The associated cough may become very annoying.

The nervous system is usually more or less affected. There is almost always headache in the beginning. This may be comparatively slight and disappear after a few days, or it may last throughout the disease. Sometimes it is severe and persistent. I have seen one case where the headache was almost unbearable and lasted for weeks.

A mild delirium is very common. In some cases the delirium is violent. As a rule it is mild and the patient at first is talkative. Then, as the patient grows weaker, he becomes apathetic, and in severe cases goes into a stupor for several days or a week or two, from which he can be aroused only with difficulty. The sphincters become relaxed and the patient passes urine and feces involuntarily.

The blood in the beginning contains the bacillus typhosus. At first the bacilli are quite numerous; but they grow gradually less and less until at the end of the second or the beginning of the third week they have almost or entirely disappeared.

In children at a very early age, and even in the fetus, the typhoid germ has been found. Peyer's patches appear very early—Rotch has seen them in children, dead at the age of three days—so we may have not only the constitutional symptoms, fever, diarrhea, and so forth, but we may also have the typical ulceration of Peyer's patches.

The disease is rare in those over sixty years of age. It is also unusual for a patient to have a second attack—except as a relapse immediately following a first attack.

Relapse: After a patient has been through the complete cycle of symptoms and is apparently on the road to recovery, he may start in and go through the whole thing again. This is called a relapse. As a rule, a relapse is less severe than the original attack and runs a shorter course.

Relapse is probably due to a new autoinfection. There may be more than one relapse, although that is rare. The recrudescence of symptoms, when relapse occurs, usually begins about the end of the first week of normal temperature and is indicated by a new rise. It is quite important therefore that the temperature continues to be carefully recorded even after convalescence appears to have set in.

A few years ago Delafield reported a series of cases where there was a slight rise in temperature for a few days, but not of the duration or associated with the other symptoms of a true relapse. He considered it simply due to the readjustment of the system to a return to the ordinary diet and activities. It should cause careful watching, but is not of evil significance. Renewed rest is indicated.

Complications and Sequelæ: The most frequent complications have been mentioned under symptomatology; namely, bronchitis, which may be classed as a symptom, and hemorrhage or perforation of the bowel.

Typical lobar pneumonia sometimes develops. I have met with several cases. It usually supervenes when the fever is at its height, during the second or third week of the typhoid. In my cases so complicated the respirations were greatly increased —more than sixty per minute. Otherwise there was no notable change in the general condition, except that the patient was a little more sick.

Malaria is a rare complication. Osler says it occurred once in 685 cases at Johns Hopkins. I have seen one such case in my series, where the typhoid bacilli and the malaria plasmodium were both demonstrated.

Phlebitis is much more common. Thayer reports its occurrence

thirty-nine times in 1,463 cases. It occurred during the third week or later. Phlebitis is usually ushered in with fever, localized pain, and edema of the affected part.

Middle ear disease is sometimes a complication in typhoid as in the other acute infections. Its possibility must never be lost sight of.

Endocarditis only occasionally occurs. A true myocardial degeneration is much more frequent. This is indicated by feeble heart sounds, and by a pulse that is accelerated by very slight exertion.

Pregnancy usually terminates spontaneously during typhoid. I had a case in my service at the Flower Hospital in 1904, a woman five months pregnant, who had severe typhoid, complicated by lobar pneumonia. The fetus lived all through her illness and until she was far enough advanced in convalescence to go home. About two weeks after going home she was prematurely delivered.

The heart muscle is much weakened during typhoid and patients should make haste slowly in convalescing. I tell my typhoids they need not expect to feel quite at par for at least a year. Sometimes it takes longer to fully recover. The vast majority of cases gradually improve, and after some months are as good as ever. They must take life easy until the heart becomes steadied down as shown by a regular pulse. A few cases are left with permanent valvular heart lesions.

Neuritis is a very serious complication, because it takes months to recover from. Meningitis also may occur. It is serious.

Very rarely a patient is a complete nervous wreck after typhoid.

Lesions of the bones sometimes form troublesome sequelæ. They may take the form of periostitis, necrosis, or caries. The legs are the parts most often affected.

Boils followed in two of my cases. They have been reported by others.

Diagnosis: Frequently typhoid is difficult to diagnose during the first week or two. Clinically, it is well to remember that a continued fever without obvious cause is probably typhoid, malaria, tuberculosis or sepsis.

If typhoid is suspected the blood should be examined for the typhoid bacillus. If one is an expert bacteriologist he may find the typhoid bacillus during the first or second weeks. Beginning during the second week and continuing indefinitely the Widal reaction is found in typhoid fever.

At the bedside it is necessary to remember that the symptoms of the first week of typhoid are indefinite. There is malaise, rise in temperature, sometimes headache, loss of appetite, and digestive disturbance. Precisely the same symptoms may hold true of malarial infection. In typhoid the general symptoms will gradually become worse with unfolding of the typical typhoid fever curve. In malaria the malarial paroxysm of chill fever and sweat will develop. I had one such case in a household of four persons. The father had typhoid fever and recovered. The servant had typhoid fever and died. These cases occurred out of town. The first week the family was at home the son came down with the indefinite symptoms noted above, but instead of developing typhoid he developed a typical intermittent fever at the end of the week.

Miliary tuberculosis may be mistaken for typhoid, and vice versa. The two diseases may coexist. In tuberculosis the temperature curve is different, and there is absence of typhoid bacilli and of the Widal reaction.

Typhus fever may be differentiated from typhoid by the more sudden onset, the fall of temperature after a few days by crisis, the earlier eruption which does *not* disappear on pressure, and the absence of the Widal reaction.

In 1908 a method of serum diagnosis was presented to the profession for diagnosing any infectious disease, including typhoid and tuberculosis. It consists in adding two drops of the suspected serum to 1 c. c. of a preparation of 250,000 per c. c. suspended solution of the supposed bacilli, incubating for one half hour, add 1.5 c. c. of a hemolytic serum and incubate for two hours. Let stand twelve hours. In a positive reaction the blood is undissolved. In a negative reaction it forms a wine red transparent fluid.

Occasionally an acute pulmonary tuberculosis is mistaken for typhoid. I saw one such case in consultation in 1908. The

diagnosis was based on the general symptoms. Failure to examine the chest caused the error. The cough was assigned to the ordinary bronchitis so often found with typhoid.

When typhoid begins abruptly, with severe headache and other symptoms of the nervous system, it may lead to the mistaken diagnosis of cerebro-spinal meningitis. Lumbar puncture offers a means of differentiation.

Septic conditions may be mistaken for typhoid when the septic focus is not self evident; but the progress of the case will unfold the typical course of typhoid as against the more or less irregular course of sepsis. One case in my service at the Flower Hospital in 1913 ran a typical typhoid temperature and pulse rate. There was continued absence of the Widal reaction. Finally, after two weeks, a sub-diaphragmatic abscess appeared in the left side.

Prognosis: The prognosis is always in doubt until the patient is well on in convalescence. The average death rate is about ten per cent. In some epidemics it is nearly twice that. Profound disturbance of the nervous system is a bad sign. The various complications add to the gravity of the case, especially hemorrhage, perforation, or endocarditis.

Cases under homœopathic treatment seem to run a milder course as a rule and a larger percentage recover than under other methods. In an epidemic of typhoid fever in Stamford, Conn., during my residence there in 1895, there were 406 cases reported with 27 deaths, a mortality of 6.63 per cent. Of these cases 284 were treated by old school physicians with 22 deaths, a mortality of 7.74 per cent. The other 122 cases were treated by homœopathic physicians with 5 deaths, a mortality of 4.09 per cent. At New Haven, Conn., during 1891-1895 inclusive, old school physicians reported 458 cases with 119 deaths, a mortality of 25.98 per cent. Homœopathic physicians reported 60 cases with 12 deaths, a mortality of 20 per cent. The New Haven figures were reported to me by Dr. E. C. M. Hall. In the September, 1913, *Journal of the American Institute of Homœopathy*, Dr. George B. Peck reports statistics gathered by him from various Hospitals in the United States and foreign countries. In certain old school hospitals the average mortality from typhoid was

9.6 per cent., in numerous homœopathic hospitals it was 8.8 per cent.

Prophylaxis: Sporadic cases of typhoid cannot be guarded against, but after a case does develop secondary cases may be prevented by attention to detail in handling the discharges from the patient.

When an epidemic develops in a community it is best for the well to use boiled water. All milk should be sterilized until the source of the epidemic can be traced or until the epidemic ceases. Food should be protected from flies, for they are now known sometimes to be carriers of infection. In rural communities fresh uncooked vegetables may be contaminated and should be let alone.

Typhoid fever proved to be a veritable scourge during the Boer war and during the Spanish-American war in 1898. According to Surgeon J. R. Kean, U. S. A., the troops assembled at Jacksonville, Florida, in 1898, of 10,759 men, 1,729 positive and 2,693 probable cases of typhoid ocurred, with 248 deaths. At the army manoeuvres at San Antonio, Texas, in the summer of 1911, among 12,801 troops, there was one case of typhoid and no death. At San Antonio the troops were immunized with a bouillon culture of the typhoid bacillus. Each man was inoculated in the left arm with three doses at intervals of ten days. The first dose was 0.5 c. c., the second and third each 1. c. c. The point of inoculation was first washed, then cleansed with alcohol, then with iodine, and after the injection finally covered with collodion. No untoward results followed (*Journal of the American Medical Association,* August, 1911).

In a paper published in the *Chironian* of March, 1912, Capt. James M. Phalen, U. S. A. Medical Corps, reports on sixty thousand anti-typhoid inoculations in troops, covering a period of three years, following which but twelve cases of typhoid developed with no deaths.

The above figures would seem to establish typhoid immunization, first proposed by Sir A. E. Wright in 1895, on a sure foundation.

As a direct result of the army reports the authorities have required the nurses and physicians at Bellevue Hospital to be immunized against typhoid fever.

Notwithstanding the good results reported, some opposition has arisen against indiscriminate typhoid immunization. It is claimed that in the susceptible typhoid immunization seems to light up a latent tuberculosis. Moreover, the immunization holds good for only about two years.

There is often some slight reaction following immunization, symptoms of malaise, headache, rise in temperature, slight swelling about the site of injection, and so on, lasting for a day or so.

Treatment: A typhoid patient should be put to bed at once. The room should be a sunny one if possible, devoid of unnecessary furniture.

The mattress should be covered with a rubber sheet. Bed pan and urinal are imperative as the typhoid patient should under no circumstances be allowed to get up.

A temperature chart should be provided and the temperature, pulse and respiration recorded on it every four hours.

Disinfection: The feces, urine, sputa if any, in fact, all of the discharges and secretions of the body, may contain the typhoid bacillus. All excreta should, therefore, be received in an antiseptic solution and allowed to stand for a time in order that they may be made thoroughly innocuous before being disposed of. Vomited matter should be treated in the same way.

The solutions recommended by the American Public Health Association are as generally useful as any. Chloride of lime, four ounces to the gallon of water. Each bowel or bladder evacuation should be thoroughly mixed with a quart of this solution and allowed to stand one hour before being thrown away. Whatever sediment may remain can then do no harm. At Johns Hopkins, according to Osler, each stool is mixed with an equal quantity of milk of lime. Each of these is inexpensive.

Corrosive sublimate and permanganate of potash, each two drams to the gallon, may be used. This mixture must be allowed to stand in contact with discharges for four hours. It has the advantage over the lime in having no odor, but it is hard on lead pipes, and must never be used in metal vessels. Moreover it is highly poisonous.

Carbolic acid, five per cent., may be used, but it requires pro-

longed exposure. At Johns Hopkins the urine is mixed with this preparation in equal parts and allowed to stand.

All bed pans, urinals, rectal tubes, etc., should be kept sterilized by some one of the above solutions. They should be thoroughly scalded each time after being emptied, before the clean solution is put in for the next discharge. All dishes used by the patient should be scalded with boiling water and thoroughly washed immediately after use.

Clinical thermometers should be cleaned and kept in a solution of bichloride, one to five hundred, or in electrozone, one to four, or in pure alcohol. They should be rinsed off in clean water before each using.

The person of the patient where soiled by any of the discharges should immediately be thoroughly cleansed. Any of the following may be used: Chlorinated soda, one to three; or the chloride of lime solution mentioned above, one to three; or carbolic, two per cent.; or electrozone, one to four; or pure alcohol.

The hands of the attendant or nurse should always be sterilized after handling the patient, or after handling any of the clothing, dishes or other things used by the patient. If the hands have not been soiled alcohol may be used. Electrozone, one to four, may also be used. Tincture of green soap should be used, when soap is needed, with a sterile scrubbing brush.

All bed linen and clothing should be put in a five per cent. solution of carbolic acid for two hours before going to the laundry. Or, still better, they should be put in water and boiled for at least half an hour.

The mouth of the patient must be kept sweet and clean. In patients with great prostration there is apt to be an accumulation of sordes on the teeth if the mouth is neglected. This should be prevented. The buccal cavity should be wiped out frequently with a soft cloth dipped in cool water, or in water and boracic acid. If the mouth becomes very dry it may be moistened with glycerine.

To prevent the possible formation of bed sores the heels and region over the sacrum should be bathed frequently with spirits of camphor, with brandy or with alcohol and water half and half.

The patient should be encouraged to change position frequently. In case a bed sore seems imminent an air cushion under the affected part may be useful. In extreme cases a water bed may be desirable.

If in spite of all precautions a bed sore develops, the wound must be treated surgically. Bed sores are very stubborn. They must be thoroughly cleansed, dried and dusted with iodoform, aristol or some antiseptic powder. They may be cleansed with peroxide of hydrogen and water, one to four, or with electrozone and water, one to four, or with carbolic acid, five per cent. A dressing of balsam of Peru may be applied to the parts. After cleansing, a solution of witch hazel, one to three, may be used as a lotion in place of the powder. Oakum makes the best dressing after the cleansing is finished, ordinary oakum, such as is used in caulking ships. This makes a most sweet, clean and satisfactory covering.

Diet: Everyone is agreed that one of the most important factors in the treatment of typhoid is diet. But the opinion as to the proper diet is anything but unanimous. Some believe in no food at all during the fever stage, nothing but water *ad libitum*. Others believe milk to be the most satisfactory diet. Still others believe in gruels and broths but absolutely taboo milk in this disease. Within the last few years still another group of clinicians has advocated a very liberal diet. Some of the Russian physicians were, I believe, the first to recommend a liberal diet, and they have been followed to a certain extent in this country by Shattuck, of Boston, and Einhorn, of New York, among others. After watching the results of the various methods as carried out by my colleagues at the Metropolitan and at the Flower Hospitals, and after some experimenting with cases of my own, I must place myself with the last group, those who favor a liberal diet. I am opposed to the starvation theory.

In a disease as severe and lasting as long as typhoid the patient needs a liberal diet to make up for the great waste. I am sure that patients who are given a fair amount of suitable food have an easier time than those who are not. The fever is not so high, the toxemia is not so profound. They approach convalescene in better condition, and they recuperate more quickly.

The first requisite is plenty of pure, cool water for the patient to drink. Acidulated water or lemonade may be given if the patient prefers it, but pure water is the best. It should be offered at frequent and regular intervals even if not asked for by the patient. A few drops of dilute muriatic acid to the glass of water makes a useful and palatable acidulated water.

Milk is the most useful single food and should form the basis of the diet, although I never use it exclusively. Plain milk or peptonized milk may be used. To make it more palatable it may be flavored with coffee or diluted with water or lime water. It may be used as ice cream. Soft boiled eggs are palatable and useful. Broths or soups may be used with or without an added egg. Strumpell recommends in cases of extreme weakness shavings of raw beef. Mutton broth or beef tea may be useful, or beef in the form of somatose. Orange juice is also permissible. A roll or zwieback, thoroughly softened by soaking in milk or water, may be given. Another very valuable food in typhoid which I have used many times with great satisfaction both to the patient and to myself is the banana. It should be thoroughly ripe. It is very nutritious, is almost entirely digested, and absolutely harmless. It is keenly relished by patients who like bananas. I have had many patients who regularly ate one banana a day throughout the entire course of the disease.

I have had many cases go through a typhoid in comparative comfort and with little loss of weight, who had a cup of coffee about half milk for breakfast every day, a soft boiled egg at another feeding, a ripe banana at a third, filling out with milk toast, milk, broth of some kind, or soup, changing the various things from day to day to avoid monotony.

In a case treated in association with Dr. St. Clair Smith all food was withheld for a week after the patient went to bed. Then he began with milk that had been thoroughly peptonized for three hours; two ounces every four hours at first; after a couple of days the amount was gradually increased. At the end of another week broths were added.

Baths: The so-called Brand method of treatment of typhoid by means of cold baths is still favored by some. It is more easily

carried out in hospital practice than in private practice. When used the patient is lifted from his bed to the tub in a sheet. At first the water should be at 80°-85° F., being used cooler each day until 70° F. is reached. The bath is used whenever the temperature of the patient makes 103° F. or more. The bath should not last longer than fifteen minutes, less if the rectal temperature shows a drop of two degrees before the fifteen minutes are up. If the patient appears to be very cold afterwards, give him a dram of brandy in water. When the patient is taken out of the tub he should be wrapped in a dry sheet and a blanket and put to bed. Hemorrhage or perforation contra-indicates baths of this kind. When used the baths are said to clear up delirium, and to tone up a weakened heart. If the patient is not better afterwards, or seems to be in distress, do not repeat.

Personally, I prefer the sponge bath of water or of alcohol and water half and half. The sponge bath should be given carefully, one part of the body being bathed at a time. I use baths for cleanliness and comfort, not primarily to reduce the temperature. I doubt if knocking the temperature down, *per se,* is of any practical benefit. As the various symptoms improve the temperature will go down of itself.

Medical Treatment: The majority of cases of typhoid will do very well with good nursing and careful attention to the details of hygiene and diet. The old school use little in the way of medication for such cases. A careful reading of cases treated by anti-typhoid serum fails to convince me that it is as useful as the indicated homœopathic remedy. If complications occur then active interference may be called for.

Intestinal antiseptics need be mentioned only to be condemned. It is physically and chemically impossible to make the entire intestinal tract sterile with any preparation taken into the mouth. Anything powerful enough to do that would kill the patient. Moreover, typhoid is a systemic disease, and even if the intestinal tract could be made aseptic, it would be impossible to kill the typhoid bacillus in the blood and tissues with antiseptics.

Alcohol should be used only in the most extreme cases if at all. In profound prostration it may be of service. If used at all it is

best given in the form of whiskey diluted with an equal quantity of water, half an ounce at a time, and not oftener than every two hours. The majority of cases, however, will be much better off without it, even with a mild delirium.

If the heart needs stimulating strychnine, 1-60 of a grain, may be used every four hours. This drug must also be used only in extreme cases. I have seen it produce paralysis of the bowel when used indiscriminately, and this is a particularly dangerous complication in typhoid.

In case of hemorrhage of the bowel, opium given internally, and ice applied to the abdomen externally, are the most generally recommended measures. Personally, I prefer the indicated remedy, which may be *ipecac, hamamelis* or some other.

There is one exceedingly dangerous complication that should be specially mentioned under treatment. Namely, perforation of the bowel. This requires surgical treatment. J. A. Scott, of Philadelphia, in the *New York Medical Journal* of February 9, 1907, has an exhaustive study of this subject. He believes about one-third of the deaths in typhoid to be due to perforation. He states that this accident occurs most often between the fourteenth and twenty-first days of the disease. The time to operate is immediately a diagnosis of perforation is made. No case is too desperate for operation.

Typhoid fever is one of the acute infectious diseases, and runs a typical, self-limited course. In other words, sufferers from typhoid die or get well after a certain definite time no matter what is done for them. An occassional case will break off short, will be aborted. There is no way to do this with any certainty, but it does happen. By far the most important part of treatment is the general care and the nursing.

Typhoid may be made a comparatively mild disease by adding to good care the indicated homœopathic remedy and of all the drugs in the materia medica.

Bryonia is the most generally useful. Unless some other drug is unquestionably called for I always use *bryonia*. I prefer the third to sixth dilution, ten drops in four ounces of water, two teaspoonfuls every two hours. Begin its use as soon as the diag-

nosis is made, and continue it until the case is ended. It is indicated by the headache, by the intestinal symptoms, by the general condition of the patient.

Rhus toxicodendron may be indicated if the patient becomes restless, and has the characteristic red tipped tongue.

Baptisia is called for when the patient is in an apathetic condition, with profound toxemia and foul discharge. This remedy should be used low.

In 1913 Dr. Ralph R. Mellon, of the University of Michigan, reported in the *Medical Century* experiments made with *baptisia* that showed that low potencies, tincture and IX, produced in the human subject antibodies to the bacillus typhosus.

Arsenicum album is indicated in extreme prostration.

Muriatic acid is useful in low typhoid conditions with dry tongue, and sordes on the teeth.

Phosphoric acid is indicated when the mental symptoms precede severe physical symptoms.

Carbo vegetabilis and *nux vomica* in combination will be serviceable in case the patient is troubled much with eructations.

Terebinth is the best remedy for tympanitis. If very marked turpentine stupes may also be used.

These few remedies will be sufficient for the vast majority of patients. It must not be forgotten that any homœopathic remedy may be indicated, and when the symptoms are typical of any particular remedy it should be used. Again, the complications that may occur are liable to change the drug picture and the physician must act accordingly.

TYPHUS FEVER.

(Greek, τυφος, stupor.) So named because of the mental condition of the patient.

Synonyms: Spotted Fever. Hospital Fever. Jail Fever. The Exanthematous Typhus of the Germans. Petechial Fever.

Definition: An infectious disease, characterized by sudden onset, a high temperature lasting about two weeks and ending by crisis, and a petechial eruption which appears about the fifth day. Typhus is an epidemic disease.

Historical Note: Typhus is an old disease. Some of the great plagues mentioned by early writers were probably epidemics of typhus. The first accurate description of this disease was written by Fracastorius in 1546. The term "typhus" was first used by Sauvages in 1760 to describe certain low forms of fever. It was not until 1829-30 that typhus and typhoid were finally differentiated from each other.

In 1910 Dr. Nathan E. Brill, of New York, reported 255 cases of a type of fever somewhat like typhoid that he had met with during a number of years. A study of his report and observation of further cases identified it with typhus fever. This work was done by Anderson and Goldberger, of the Public Health Service. It is now supposed that many cases of this disease, a mild form of typhus, exist in our large cities.

Etiology: Typhus fever occurs almost exclusively in crowded and filthy places. There is no disease, perhaps, that is so surely a "filth disease." Want and poverty also play a part in predisposing to typhus by getting the victims into bad general condition. In 1912 it was announced that the body louse, pediculosis corporis, was responsible for the transmission of the disease. It is very contagious, many physicians and nurses caring for cases becoming victims of it themselves. This is easy to understand in view of the present theory of its transmission.

Children are as susceptible as adults.

Bacteriology: No specific germ is as yet discovered.

Morbid Anatomy and Pathology: There are no specific gross lesions. The blood is usually darker in color than normal, and very fluid.

Symptoms: The period of incubation varies from a few hours to two weeks. Ten to twelve days seems to be the average. During this period the patient may suffer from malaise and not feel well.

More often the disease begins somewhat abruptly with a chill or chills. The patient suffers from a severe headache. There is a sudden rise of temperature to 103° or 105° F. Stupor develops promptly. The patient has a dusky, almost livid, appearance. The surface of the body becomes congested, the conjunctiva injected.

The temperature reaches its height in a few hours and remains there from ten to fourteen days, when it drops by crisis.

The toxemia has a profound effect on the heart and blood. The heart becomes weakened. Minute extravasations of blood appear in the skin, forming the so-called "mulberry rash," which does not disappear on pressure.

The nervous symptoms are severe. The patient rapidly passes into a stupor. This may become a coma. Convulsions are rare but fatal.

In the mild form described by Brill the symptoms are like those outlined above, but usually mild.

I saw one case that I diagnosed as Brill's disease, mild typhus, at the Flower Hospital in 1913.

The patient, a Russian Hebrew aged 39 years, was admitted September 13. He was apathetic. The eyes were congested, the face flushed. Over the chest, body and arms was a profuse eruption of discrete reddish spots. The throat was also red. The temperature 103° F. He had been ill five days when he entered the hospital. I was in doubt about whether I had scarlet fever to deal with at first. The temperature remained up, but the eruption promptly began to fade. The general symptoms resembled typhoid—scarlet fever was eliminated on a second examination. The Widal reaction was negative. September 16 the temperature started down, and in forty-eight hours was normal. The patient was dismissed cured a few days later.

Complications and Sequelæ: Bronchitis and pneumonia may sometimes complicate typhus.

Diagnosis: Typhus and typhoid must be differentiated. The mental condition of typhus and typhoid resemble each other—hence their names. The two diseases are dissimilar, however, in their mode of onset. Typhus appears abruptly, typhoid slowly. The typhus temperature curve rises abruptly, remains high for ten days to two weeks and drops by crisis; the typhoid temperature curve rises gradually, remains high for two weeks, and drops gradually. The eruption of typhus appears earlier, is general and does not disappear on pressure; the typhoid eruption appears later, is usually limited to the trunk and does disappear on pressure. Typhoid gives the Widal reaction, typhus does not.

Cerebro-spinal meningitis has more pain in the back of the neck than typhus. Opisthotonos is not uncommon. Withdrawal of cerebro-spinal fluid gives opportunity for its examination. Cerebro-spinal meningitis has a specific organism in this fluid, typhus has none.

Prognosis: The prognosis is doubtful. The mortality varies from 10 to 20 per cent. in different epidemics.

At the old Ward's Island Homœopathic Hospital eighteen cases were treated in 1892 without a death. There have been a few mild cases in New York since, but no epidemic.

Treatment: As the disease is now supposed to be carried by body lice, measures must be taken to dispose of that possibility. Dry heat or steam must be used to fumigate clothing. The burning of sulphur, two pounds to each 1000 cubic feet of air space, is the best method of fumigating a room. Formaldehyde will not kill vermin.

The patient should be isolated and kept clean.

An ample supply of fresh air is essential in the sick room. The eighteen cases mentioned above as having been treated at the Ward's Island Homœopathic Hospital were kept out of doors in tents. They were all gathered from one or two cheap lodging houses. There were no secondary cases.

Disinfection of all discharges is the next important step. This should be done in the way described under typhoid fever.

The diet during the height of the disease should be liquid.

The homœopathic remedies mentioned under typhoid may be indicated in typhus. Besides those there may be mentioned:

Arnica for the general mental condition, together with the ecchymotic eruption.

Lachesis for the profound blood changes.

RELAPSING FEVER.

(Latin; *re,* again, back, *labor,* fall or slip.) So named because the symptoms recur.

Synonyms: Relapsing Typhus. Febris Recurrens. Famine Fever.

Definition: A specific fever caused by the spirillum of Ober-

meier. It is characterized by sudden onset, high temperature for six or seven days, then a period of about the same length of time of normal temperature. This cycle of symptoms is repeated a second, sometimes a third and even a fourth time.

Historical Note: Relapsing fever was first accurately described by Rutty, in 1739. Since then there have been various epidemics in different parts of the world. In 1873 Obermeier described a spirillum found in the blood of patients during the febrile paroxysm.

Etiology: At one time lack of food was supposed to cause relapsing fever, hence the name "famine fever." The disease usually occurs in epidemics, in times of destitution. Physicians and nurses are often attacked, and in hospitals other patients in the wards are frequently taken down with it.

The spirochetæ of Obermeier is the specific cause of relapsing fever. It seems to be transmitted by fomites. According to Tictin and Karlinski the bed-bug acts as an intermediate host.

No age or sex is exempt. In an epidemic in Philadelphia in 1869-70 of 1,164 cases, 225 were under twenty years of age, and 13 were between seventy and ninety years.

Bacteriology: The spirochetæ or spirillum of relapsing fever was first described in 1873, by Obermeier. It is a fine twisted, thread-like microbe, and very motile. It is found in the blood, but only during the paroxysm of fever. Between times it is not found except in the spleen.

Morbid Anatomy and Pathology: Recently it has been found that the heart muscle is degenerated, soft and flabby. The spleen is always very much enlarged. Otherwise there are no typical and characteristic pathological changes.

Symptoms: Some cases develop suddenly, others have a varying period of malaise before becoming really sick. The period of incubation is from one to fourteen days, rarely longer. The disease is usually ushered in by chills, and a violent headache and backache. Delirium is rare. The tongue is coated, there is nausea and vomiting. The temperature rises to 104°-106° F., with slight remissions. Thirst is excessive. The pulse rises to 140 or more. The patient becomes very restless.

Examination shows the liver and spleen to be much enlarged, more so than in any other disease. Jaundice is common.

From the third to the fifteenth day there is a sudden remission in all the symptoms. The temperature drops to normal. The patient is weak, but rapidly recovers his strength. The spirillum, found in the blood during the fever, disappears. The patient feels well.

Suddenly, at the end of a week, all the symptoms reappear. The temperature goes up, the headache and backache reappear, the nausea and vomiting recur. This relapse lasts from one to five days when all the symptoms again disappear. There may be a second, or more relapses. As many as eight have been recorded.

During the paroxysms albumin is found in the urine, though a true nephritis does not often occur.

Complications and Sequelæ: Parotitis and middle ear disease have been reported as complications in some epidemics.

Cardiac thrombosis is said to be more common in relapsing fever than in any disease except diphtheria.

Diagnosis: The temperature curve is characteristic. No other disease can be mistaken for it. The spirillum, of course, is pathognomonic. Typhus is the disease that most closely resembles relapsing fever during the paroxysms.

Prognosis: The mortality in relapsing fever is about ten per cent. In young people the prognosis is good.

Treatment: Relapsing fever is very contagious. Sandwith reported that of seventy-four convicts who volunteered to care for cases, thirty-nine contracted the disease in from two to six weeks. Owing to its communicability isolation of the patient is the first requisite.

The diet should be liquid.

In the way of medicines the old school have nothing to offer. Morphine is recommended to relieve the pains. Rabaglisti says small doses of *ipecac* frequently repeated sometimes relieved the nausea and vomiting. This is good Homœopathy, though the recommendation is from old school sources.

Bryonia would seem to be useful for the headache and backache.

Arsenicum album later on when the pulse becomes rapid and weak, and the patient restless.

Other remedies must be used as indicated.

MALARIA.

(Italian, *mala,* bad; *aria,* air.)

Synonyms: Intermittent Fever—Tertian and Quartan. Estivo-autumnal Fever. Swamp Fever. Fever and Ague, etc.

Definition: Malaria is a specific infectious fever caused by the plasmodium malariæ. There are three general types, the *tertian,* the *quartan,* and the *estivo-autumnal.*

Historical Note: Malaria was known to the ancients. Our knowledge of its nature is comparatively recent. According to Thayer, in Albutt's System of Medicine, second edition, there are three epochs in the advance of our knowledge of malaria. First, the introduction of the use of cinchona in 1640, and the discovery of its specific action in a limited class of febrile diseases. Second, the discovery of the plasmodium as the specific cause of malaria by Laveran in 1880. Third, the demonstration of the agency of the anopheles mosquito as the agent for its transmission by Ross in 1897.

Etiology: The cause of malaria is the plasmodium first described by Laveran, a French army surgeon, working in Algiers, in 1880. Before then and since malaria was and is known to be a disease peculiar to swampy regions. In 1897 Ross discovered that a certain mosquito, the anopheles, acted as an intermediate host. That is, a person is inoculated with malaria by the bite of a mosquito which has previously bitten and become infected from some person ill with the disease. Experimentally malaria has been transmitted by direct inoculation of a healthy person with the blood from an infected person.

Neither sex and no age is exempt. In malarious countries the children are nearly all infected, Koch found. The negro race seems to be less susceptible than other races.

Malaria exists wherever the anopheles is found. This is principally along low lying swampy coasts and rivers. It is more

prevalent and more severe in warm climates. It is not found at high altitudes. Heat and moisture are necessary. The anopheles are night biters, so that the old idea of night air being dangerous is based on fact.

Persons who have once had an attack of malaria seem to be liable to subsequent attacks. Even in the most malarious places a few persons seem to be immune to the disease. Another curious fact is that persons who have lived for a long time in a malarious country will often precipitate an attack on going to another climate. I have seen such cases. Malaria played havoc with many of the soldiers who fought in the Spanish-American war, after they had returned to the United States. A woman who has ever had malaria, if she later gives birth to a child, may have a malarial paroxysm during the lying-in period.

Bacteriology: The organisms of Laveran, the plasmodium malariæ, are of different varieties. They develop within and destroy the red corpuscles. The earliest forms appear in the blood during or shortly after the latter part of the malarial paroxysm. They are small, colorless disc-shaped bodies which occupy but a small portion of the red corpuscle. They possess ameboid movements. They gradually increase in size, becoming pigmented and filling the corpuscle. The blood corpuscles in consequence become much swollen. After a time they begin to segment. The *tertian* form completes its cycle in forty-eight hours. The *quartan* form takes sixty-four to seventy-two hours. It does not quite fill the red corpuscle, and the latter is therefore not increased in size. The *estivo-autumnal* type is supposed to take twelve to twenty-four hours to complete its cycle. After five days to a week crescent shaped bodies appear outside the red corpuscles, which bodies are supposed to be further developments of the plasmodia.

Morbid Anatomy and Pathology: Only the severer forms of infection cause death. Thayer, who has made an exhaustive study of malaria, says that in pernicious forms of the disease the parasites will attack individual organs, and sometimes cause such change of function as to dominate the clinical picture. The plasmodia are almost always most numerous in the spleen and in

the bone marrow. Nearly all the organs are deeply pigmented, but otherwise are not changed. The brunt of the infection seems to be borne by the spleen. This is enlarged, soft, deeply pigmented—almost black. Small points of necrosis may be found. The liver is enlarged, of a dark brown or slaty color. Amyloid changes may occur in these and other organs after long continued and repeated infections.

Symptoms: The clinical picture of the malarial paroxysms in intermittent fever, whether *tertian* or *quartan,* is the same. Incubation lasts about fourteen days in the *tertian* type, about three weeks in the *quartan* type, and about ten or twelve days in the *estivo-autumnal* type. Occasionally the incubation period is longer than the above figures in each of the forms of malaria.

The onset is usually of several days' duration. The patient complains of headache, backache and general indisposition. He has an irregular fever. He may feel weak. There are irregular chilly sensations, the patient perspires easily. He may even be delirious. These symptoms may last for several days or a week before the onset of the typical paroxysms.

The day of the onset of the paroxysm the patient feels weak and has aching of the limbs, sometimes headache and backache. He does not wish to do anything, he feels too sick and tired. Sometimes there is nausea and vomiting. After a few hours of this the cold stage comes on. The patient is very chilly or has an actual chill. The face is drawn and pinched. The patient looks cold. He wants to be covered and even then cannot get warm. He dreads the air. The pulse is rapid, 100 or more, and weak. This stage lasts two hours or so. Then the patient begins to get warm.

The fever, which began during the chill, reaches its height during this stage. The temperature may go to 105° or 106° F., or higher. There is frequently severe headache, pain in the region of the spleen; and backache. The face is flushed, the patient may be delirious. More rarely he is apathetic. There is intense thirst. This stage lasts four or five hours.

Some patients do not have a marked chill. In that case the temperature may go up and down again undetected and the

diagnosis be in doubt. A boy in my service at the Flower Hospital had his highest fever at 10 a. m.—103° F. At 8 and at 12 it was only 100° F. The temperature had been taken regularly every four hours without discovering the highest point for several days.

After the fever stage the patient begins to perspire freely. With the beginning of this stage the patient begins to feel better. The aching pains disappear, the temperature drops rapidly to normal or subnormal, the pulse becomes slow. Frequently the patient falls asleep.

During the intermission between paroxysms the patient feels well, is up and about as usual. The temperature meanwhile is subnormal—95° or 96° F.—and the pulse slow.

In the *tertian* type, forty-eight hours after the first paroxysm a second one starts in and goes through the same process of chill, fever and sweat. These paroxysms are repeated indefinitely unless controlled by medicine. Rarely, they may end spontaneously.

Cases occur of a double tertian infection. That is, one infection will cause the paroxysms every other day; the second infection will cause similar paroxysms on the intervening days at forty-eight hour intervals. A daily paroxysm is called *quotidian* fever—the double tertian infection.

The *quartan* type of malaria has the same kind of paroxysm every seventy-two hours. In this type also there may be a double infection, or a triple infection causing daily paroxysms.

Examination of the blood will show the particular type of organism responsible.

In either of the intermittent fever types of malaria, there is a tendency for a paroxysm to recur at the end of a week after the last one. The patient should be kept under observation then for a week or ten days after manifestations of malaria have ceased to appear.

The *estivo-autumnal* type of malaria is more irregular in its clinical manifestations than either the tertian or the quartan types. If the temperature is taken every two hours and recorded it will be found to have an intermittent character. According to Craig the temperature goes up rapidly, as in the ordinary forms of intermittent fever. There is then a pseudo crisis, a drop of

one or more degrees, lasting for a short time, followed by a rise to the previous high point or higher. Finally comes the real crisis, the temperature sometimes becoming subnormal. This final drop is slower than in the tertian or quartan types of malaria. The paroxysms due to the tertian and quartan forms last six to seven hours, whereas the paroxysms of the estivo-autumnal form lasts twenty to twenty-four hours.

In all three forms the urine, in about half the cases, contains albumin. The sweating stage is frequently accompanied by polyuria.

There are various forms of malaria specially named because of certain clinical features presented. All are due to infection by one or more of the three types of malarial parasite, and the peculiar symptoms are supposed to be due to the intensity of infection, to the combination of different sets of parasites, to the migrating of the organisms to certain parts of the body, or to some special susceptibility on the part of the patient.

Pernicious malarial fever is a dangerous disease. In malarious countries it is not uncommon. It usually occurs in those who have had previous attacks of intermittent fever. There are several forms described:

(a) The *comatose* form. Here the patient is overcome by the poison of the disease, and may suddenly become comatose, or after a day or two become so. It occurs in persons who have had previous attacks of the ordinary forms of malaria. The temperature is 103° or 104° F., pulse rapid but weak, respirations increased. The sphincters are relaxed. The skin is cold and covered with perspiration. The patient is in a state of collapse.

(b) The *algid* form is collapse accompanied by vomiting and diarrhea the disease simulating cholera. This has the congestive chills found in the southern states.

(c) *Dysenteric* cases occur that may be mistaken for dysentery. Blood examination will reveal the plasmodia, and quinine will cure the case.

In other cases jaundice and vomiting of bile are symptoms.

The *remittent* and *continuous* forms are severe types due probably to infections with several groups of organisms. They

develop from ordinary cases of intermittent fever. The diagnosis is made by the blood examination.

Typho-malarial fever is a misnomer, most cases so-called probably being typhoid fever. The two diseases sometimes coexist, but not often. I have seen one case. Osler says there was one in 689 at Johns Hopkins.

Masked malaria is the name given to certain cases in malarious districts that present marked intermissions, but without the fever—they are mostly neuralgias and dysenteries.

Malarial cachexia is the name given in malarious districts to cases of general debility marked by jaundice, gastric disturbances, diarrhea, certain nervous symptoms, more or less continued fever, anemia, etc. The spleen and liver eventually hypertrophy in these cases.

The intermittent fevers occur, but they are not common, in New York City. Most of the cases that I have seen, both in private practice and in hospital practice, give a history of having been somewhere out of town a few days or weeks previously. The cases of the severe types have all been a longer or shorter time in some malarious district. I have had several such cases from South America.

Complications and Sequelæ: The lighter forms of malaria may at any time take on a pernicious character if improperly treated, or they may develop the malarial cachexia. This is more particularly true where malaria is common.

Albuminuria is present in about half the cases. Nephritis occurs in about 3 per cent. (Craig.)

Typhoid fever occurs occasionally. Craig saw it eight times in 5,000 cases. Osler saw it once in 689 cases. I have met with it once in my service at the Metropolitan Hospital.

Various nervous and mental conditions occur with or follow malaria.

Pneumonia is sometimes a complication.

Craig states that sixty-five per cent. of the patients with malarial parasites in the blood observed at the Army General Hospital, Presidio of San Francisco, suffered at some time from dysentery.

Anemia is a serious sequel of the estivo-autumnal type.

Diagnosis: The most frequent mistake, in my experience, is

in mistaking malaria for typhoid fever, or vice versa. The prodromal stage of malaria, lasting several days, is very like the first week of typhoid. Only subsequent developments will make the clinical picture clear in many cases.

Another mistake that I have met with a number of times is this: A lying-in woman who at some time or other has had malaria—it may have been years before—will suddenly develop a temperature of 104° to 106° F., or even higher. The natural inference is that the patient has puerperal fever. As a matter of fact it may be a recurrence of malaria. By all means the physician should be on his guard against puerperal infection, but he should find out if his patient has ever had malaria. If she has, the probabilities are that quinine will reduce the temperature. I have had this happen in my private practice. I have seen it happen in hospital practice.

Tuberculosis is often mistaken for malaria. I have known of two instances where the patients were treated for malaria for months without result; then a deferred examination of the chest showed advanced pulmonary tuberculosis, and the patients shortly died.

In doubtful cases quinine may be administered. This will show its effects on malaria, it will have little or no influence over other diseases.

Finally, examination of the blood will make the diagnosis as regards malaria certain.

Prognosis: The prognosis of the milder types of malarial infection is usually good. In the pernicious forms it is always grave. Some of the patients may die, others again may become chronic invalids.

Treatment: Prophylaxis of malaria involves getting rid of the infecting mosquitoes. In large communities drainage of the land will do away with their breeding places. This costs money, but the reclamation of swampy lands will frequently make such drainage economically profitable.

Kerosene poured on the surface of ponds and stagnant pools —one ounce to fifteen square feet of surface—will kill the larvæ and also the older mosquitoes when they come to lay their eggs.

Small areas, cesspools, etc., should be screened.

Next to getting rid of the mosquitoes comes screening the

malarial patient so that mosquitoes cannot get to him. This not only protects other persons but also the patient from possible reinfection.

Craig recommends daily five grain doses of quinine as a personal prophylactic for those who live in malarial regions.

The malarial patient should be put to bed as long as he has fever. The diet should be light.

The principal drug to be considered is *quinine* in some form. This drug kills the malarial organism if given in sufficient dosage. Opinions differ as to the best way to administer quinine. Some give a large dose, ten grains, just before the expected paroxysm. I have tried various methods in the ordinary tertian type as found in this region, and believe the best results are gotten by giving one to three grains of quinine every two to four hours, until the desired result is accomplished. The following prescription has served me well:

℞. Sulph. quinia .....gr. lx.
Sulph. morph. .....gr. ⅛.
Mx. ft. pil. .....xx.
Sig. One pill every four hours.

Quinine has a decided and direct effect on the plasmodium malariæ when given in sufficient dosage. Moreover it is directly homeopathic to the symptoms in certain cases.

Hahnemann is said to have first had his attention called to the similarity between the symptoms of intermittent fever and the symptoms produced in a well person who had taken cinchona bark, when translating Cullen's Materia Medica from English into German. Cullen was the authority of his day on drug action. With this as a basis Hahnemann experimented for a number of years with cinchona and other drugs before he formulated his law of cure, *"similia similibus curentur."* If there is any one drug in the materia medica, therefore, that deserves the unqualified approval of the true Homeopath it is cinchona and its various derivatives. It should not be taboo as it is to so many. Like each of our other drugs it will do the work when indicated, and no other drug can take its place.

In the more severe forms of the estivo-autumnal type of malaria, if quinine is used, it may have to be used in massive doses. Ten, twenty, even forty grains at a dose may be necessary.

Arsenic in some form is the most useful drug in severe and irregular types of malaria. It is highly recommended by Trousseau. I have seen cases cleared up by it.

Many cases of malaria will present distinct indications for drugs other than quinine and arsenic.

Natrum muriaticum is a valuable remedy in intermittent fever with the paroxysms beginning regularly every other day at 9 or 10 a. m. and lasting till 4 p. m. I have cured such cases with it.

Bryonia is indicated with a chill every other day at 1 p. m. The headache and backache are almost unbearable. The urine dark, almost mahogany colored. Sometimes there is nausea, vomiting and great thirst. I have had a number of cases of this kind.

Gelsemium is another drug that I have verified in malaria when the patient complained of chilliness rather than a distinct chill; the face flushed, considerable headache, and the patient inclined to be apathetic.

Nux vomica is indicated in intermittent fever when the paroxysms, coming every other day, begin an hour or two earlier each time. These cases are not common, but when they do occur *nux vomica* will stop them. It may be necessary to use the solid extract in half grain or one grain doses.

One fact that must be borne in mind in all cases of malaria is this: The paroxysms have a tendency to return seven or fourteen days after they have apparently ceased. The patient should therefore be kept under observation for at least two weeks after he is supposed to be cured.

MALTA FEVER.

(So named because it is endemic in the Island of Malta.)

Synonyms: Mediterranean Fever. Rock Fever. Neapolitan Fever, etc.

Definition: A disease of long duration, characterized clinically, by continued fever, profuse perspiration, constipation, frequent relapses, rheumatic or neuralgic pains, swelling of joints, or orchitis; bacteriologically, by the presence in the blood of the

micrococcus melitensis; and anatomically, by congestion of the spleen and other organs. (Bruce, in Osler's Modern Medicine.)

Historical Note: Malta Fever is not confined to Malta, but is also found at Gibraltar and other places on the Mediterranean, and at certain tropical and sub-tropical places in both the Eastern and Western Hemispheres. In 1911 it was announced that many cases of a hitherto unclassified disease found in Mexico and in Texas along the Mexican border were really cases of Malta Fever. We are largely indebted to the English army surgeons for our knowledge of the disease. Col. Bruce isolated the specific germ in 1887. The amount of illness from this disease among British soldiers, on the Mediterranean, is very great.

Etiology: Malta Fever is caused by the micrococcus melitensis. This germ has been found in a large proportion of the cases examined in the urine. Experiments on animals and accidental laboratory infection have shown that the disease may be transmitted by contaminated food or drink, that it may be gotten by breathing the germs, that it may originate from accidental inoculation through the skin or mucous membranes. In Malta goat's milk is in general use. In 1905 it was discovered that many of the goats, though not sick, had the micrococcus melitensis in the blood and in their milk. Bruce thinks that probably this is the most frequent means of communication of the disease. The cases found in Mexico and Texas occurred where goat raising is an industry and where goat's milk is used.

No age nor either sex is exempt.

Bacteriology: The micrococcus melitensis is about 0.33 μ in diameter. It may grow singly, in pairs, or in short chains.

It grows on potato and in broth. It is non-motile. It stains and is decolorized by Gram's method. It agglutinates with specific animal serum; and renders milk alkaline without coagulation.

Morbid Anatomy and Pathology: The spleen is enlarged and contains the specific micrococcus in great numbers. There may be congestion of the large intestine and of the kidneys. The base of the lungs is always congested.

Symptoms: The incubation period averages about two weeks, it may be one to three weeks.

Invasion may be abrupt or gradual. Usually there is a feeling of malaise. There is headache, loss of appetite, sometimes nausea and even vomiting. Constipation is one of the most constant symptoms.

The lungs usually become congested. Rheumatic pains are very common late in the disease. The ankles, according to Notter, are most often affected; the sacro-iliac articulation and the ribs and sternum frequently so.

Palpitation is common. Purpura and hemorrhages are frequently encountered.

The temperature curve is very irregular, but shows a continued fever lasting usually for three to four months. Rarely one month is the limit.

The patient is despondent. The face presents a sallow and tired look. The memory is often impaired. Neuritis frequently occurs.

Orchitis occurs in about fifteen per cent. of the cases.

Complications and Sequelæ: Most of the complications have been mentioned in the foregoing section. Pneumonia and pleurisy sometimes occur. Neuritis, which eventually disappears, is not an infrequent sequel.

Pulmonary tuberculosis may sometimes supervene.

Diagnosis: The disease most often mistaken for Malta fever is typhoid fever. The Malta fever is marked by constipation, neuralgic and rheumatic pains, long irregular fever, and absence of rose spots. Typhoid more often has diarrhea, lacks the peculiar pains of Malta fever, has a definite fever curve, and presents the rose spots.

The finding of the micrococcus melitensis in the blood is pathognomonic. This cannot be done in all cases. The agglutination test may be made in a 1 to 20 dilution.

Prognosis: The prognosis is usually good. The mortality is about two per cent. Most of the other cases entirely recover.

Treatment: The Malta fever patient should be isolated. Care must be exercised by the attendants in handling the discharges, particularly the urine. The food and drink should be beyond suspicion.

Light diet is indicated. Not until the fever is gone for two

weeks should the ordinary diet be resumed. Notter says convalescence is rarely completed without change of climate. The patient should not be moved, however, during early stages of the disease.

Drug treatment is said to be useless. Quinine has no effect on the fever, the salicylates have no effect on the pains. Alcohol is of no value.

Hydrotherapy in the shape of baths and packs gives relief.

Various homœopathic drugs appear to be indicated from the symptomatology of the disease. It would be interesting to try what symptomatic prescribing could do.

From the symptoms it would seem as though *bryonia* would be a very valuable remedy. It has the fever and the rheumatic pains, and the constipation.

Other remedies that suggest themselves are *aconite, arsenic, cimicifuga, ipecac, rhus toxicodendron.*

DENGUE.

(*Dengue* is derived from a Spanish word meaning dandy, so named because patients affected with dengue have such a peculiar walk.)

Synonyms: Dandy Fever. Breakbone Fever.

Definition: An acute infectious disease met with only in tropical or subtropical climates, characterized by sudden onset, an initial rash, a period of remission, and a terminal rash, with pain in the tendons and muscles.

Historical Note: The first accurate description of this disease was made about 1765. The first American description was by Rush in 1780. A number of epidemics have occurred since in our Southern States as well as throughout the West Indies, Southern Europe, Africa and Asia.

Etiology: The disease exists only in tropical or subtropical climates and in very hot weather. It seems to travel by trade routes. It is no respecter of persons, all ages and both sexes being susceptible.

The theory has been advanced that the disease is transmitted by mosquitoes.

Bacteriology: No specific organism has been generally accepted, although several have been described.

Morbid Anatomy and Pathology: These are not known.

Symptoms: The incubation period is from a few hours to three days, rarely longer.

The patient usually is suddenly attacked. Manson says the victim often gets up in the morning feeling all right, but before he is able to dress is seized with pains in various parts of the body, develops a high fever, and is obliged to crawl into bed again.

The pains seem to be in the tendons and deep muscles about the joints, rather than in the joints themselves. They are particularly severe about the loins and in the head. These pains have given the name "breakbone fever" to the disease.

The fever ranges from 103° to 105° F., sometimes higher.

The face assumes a dusky hue, especially about the eyes and nose. This lasts two or three days. All the symptoms disappear by crisis at the end of that time and the patient feels good.

After a varying length of time, it may be as long as a week, a second rise of temperature occurs, not quite so high as the first.

With this rise of temperature the pains reappear. There is also a second eruption. This is erythematous in character usually in minute areas. Less often it is a general diffuse redness. It appears first in the palms of the hands and about the wrists. Thence it travels up the arms, over the body, and down the legs. It lasts two or three days and then disappears in the same order in which it appeared. It may be very irritating during the first twenty-four hours. It is followed by a fine desquamation.

The tongue is coated during the height of the symptoms each time, and occasionally there is nausea and vomiting.

In a few cases epistaxis occurs, when this happens the relief to the headache is immediate. Convalescence is somewhat slow.

Complications and Sequelæ: There are none of common occurrence although Manson believes the disease often leaves the patients more or less debilitated so that they are more susceptible to other infections.

Diagnosis: Sporadic cases are hard to differentiate from rheumatic fever at times. The characteristics of dengue are the severe symptoms—the remission—and the severe symptoms again. Rheumatism has no such remission. Moreover dengue has its characteristic eruption with each increase in symptoms, whereas rheumatism has no such eruption.

Yellow fever occurs in the same localities as dengue and sometimes both diseases are epidemic at the same time. Yellow fever is not of such sudden onset. The fever shows no such remission. The yellow fever patient becomes jaundiced instead of red.

Influenza exhibits some of the pains of dengue, but it does not show the eruption, or the remission. Epidemically, dengue and influenza are much alike in the rapidity with which they spread, and the great numbers of the population which they attack.

Prognosis: The prognosis is good. Almost no one ever dies of dengue. There is always an increase in the general death rate from other infections at the time of a dengue epidemic. As noted above, Manson believes this to be due to the fact that recoverers from dengue are more susceptible to other diseases.

Treatment: Isolation would be ideal if possible, but the disease travels so fast that this cannot be accomplished.

The dengue patient should go to bed and stay there till the end of the second exacerbation of symptoms. Otherwise the case is apt to be prolonged.

The diet should be liquid.

The old school recommends morphine for the pains. Manson says *aconite* may do good in the beginning, and that *belladonna* is good for the pains.

The homœopath, of course, will use the indicated remedy.

Aconite would seem to be indicated in the beginning.

Belladonna is indicated in cases of high fever and with the characteristic signs of congestion.

Rhus toxicodendron seems to fit the pains in the tendons closer than any other remedy.

Bryonia and *cimicifuga* should also be remembered.

YELLOW FEVER.

(So-called because of the color it makes the patient.)

Synonyms: Yellow Jack. Bilious Remittent Fever. Black Vomit. Gibraltar Fever.

Definition: Yellow fever is an acute infectious disease characterized by fever accompanied by jaundice, hemorrhages from the mucous membranes, and sometimes by black vomit.

Historical Note: Yellow fever is endemic in Central America, the West Indies, and along part of the coast of South America. From these centers it has at times been carried to other tropical and subtropical countries, usually entering the new place at the sea ports. Various theories were put forward as to the cause and method of transmission of yellow fever. In 1881 Dr. Charles Finlay, of Havana, declared the mosquito to be responsible for its spread. His ideas did not receive general recognition, however, until the work of the American Army Commission, appointed by Surgeon-General Sternberg, in 1900, and consisting of Drs. Reed, Carroll, Agramonte and Lazear, had carried out a long series of carefully conducted experiments, which showed that the mosquito was the intermediate host. Their work has been confirmed by a French Commission working in Brazil, and by numerous other observers.

Etiology: The specific germ of yellow fever has not been found. It has been proven that the disease is not transmitted by fomites or by direct contact. It is transmitted by infected mosquitoes—the stegomyia fasciata—mosquitoes who have first bitten a yellow fever patient. The mosquito must bite the fever patient during the first three days of the disease. The insect does not become dangerous to susceptible persons until twelve days later, and remains dangerous as long as it lives after that.

Negroes are less susceptible than white persons. One attack usually, not always, confers immunity for life. Young adults are most frequently attacked. Debilitated patients are most apt to die. Newcomers to a place are very susceptible.

Bacteriology: Many germs have been thought to cause yellow fever, but as yet none has proven to be the specific germ.

Morbid Anatomy and Pathology: Rigor mortis sets in early

and is marked. The skin and eyeballs are usually yellow. The liver is yellowish in color, hard and firm in consistency. The cells undergo fatty degeneration. The stomach and most of the small intestine present submucous hemorrhages and are frequently filled with black fluid blood. The kidneys show parenchymatous changes.

Symptoms: The incubation period is one to five days. It was formerly put down as two to three weeks, but this would include the time necessary for the mosquito to do its part.

There may be slight prodromal symptoms, but usually the onset of the disease is somewhat sudden. The disease usually sets in with a chill. There is headache and general aching all over. The face is flushed and eyes injected. An early sign is jaundice about the eyes. The temperature may be anywhere from 100° to 106° F. The temperature keeps up a little while—two or three days; then there is a remission. This may be the beginning of convalescence.

In the majority of cases there is a second increase in temperature. The disease may end by lysis after two or three days more. Or the temperature may go on increasing until death. As the second attack of fever comes on, the pulse rate slows. This is called Faget's sign. At this time also the jaundice becomes marked. Vomiting usually sets in—it may be almost black from admixture with blood. There is sensitiveness over the epigastrium. Frequently there is a blotchy looking eruption. Sometimes there are hemorrhages from the gums.

A quite frequent symptom is albuminuria. This occurs early, the second day, and persists throughout the disease. In severe cases there may be great diminution and even complete suppression of urine.

Occasionally there may be delirium, though usually the patient is conscious throughout the disease.

In all epidemics there is a vast difference in the severity of the cases. Some may be so mild as to escape recognition, others may run a fatal course in two or three days.

Complications and Sequelæ: The yellow fever patient that recovers, recovers promptly, and completely. There are no sequelæ to mention—except possibly boils.

Diagnosis: Dengue is not always easy to differentiate from yellow fever. Hemorrhages and jaundice occasionally appear in dengue. As a rule, facial appearance, slowing pulse with rising temperature, and albuminuria is sufficient to diagnose yellow fever.

Malaria has an enlarged spleen—yellow fever has not. Malaria has the characteristic organisms in the blood.

Prognosis: The mortality of yellow fever varies. It is greatest in poorly nourished subjects. In private practice the mortality may be as low as ten per cent. In some epidemics it has exceeded fifty per cent. Severe symptoms, headache, high temperature, and excessive vomiting, are danger signs.

Treatment: The yellow fever patient should be isolated. He should be protected from mosquitoes. If mosquitoes can be eliminated other cases will not follow. According to Carroll, in Osler's Modern Medicine, the stegomyia fasciata rests in the middle of the day, and non-immune persons can go about infected localities between 9 a. m. and 3 p. m. with impunity.

The yellow fever patient should be put to bed, the room darkened, but kept well ventilated. Food should be withheld for three or four days. Carbonated vichy should be given freely, always ice cold.

Cold applications will relieve the headache.

The discharges must be sterilized and disposed of.

Aconite is useful sometimes in the very beginning.

Belladonna may be used in the early stages with high temperature, full pulse, and congested face.

Arsenic is indicated in the vomiting—nothing will stay in the stomach—and the exhaustion.

Bryonia is indicated in cases with much headache or joint pains.

Carbo vegetabilis is called for in cases that go into collapse, the head is hot.

Veratrum album is indicated in collapse with vomiting and purging and cold sweat on the forehead.

Crotalus is called for in hemorrhagic cases.

Lachesis is useful in hemorrhagic cases plus despondency.

PLAGUE.

(Greek πλῆγη, a stroke.) So-called because of its suddenness of attack.

Synonyms: Oriental Plague. Black Death. Bubonic Plague—because of the buboes.

Definition: An acute infectious disease characterized by sudden onset, extreme weakness, high fever, and accompanied by swelling of the inguinal, femoral, axial, cervical, and mesenteric glands, by buboes, and sometimes by hemorrhages.

Historical Note: According to Calvert the affliction of the Philistines mentioned in the Bible in I. Samuel, chapters v and vi was the bubonic plague. According to Osler the first authentic account of the disease dates from the second century. The plague has passed over Europe in several great epidemics during the past few hundred years. In the fourteenth century, within five years, one-quarter of the population of Europe, 25,000,000 of people, died of it. In 1665 some seventy thousand persons died of the plague in London. That epidemic has been graphically described by De Foe, the author of Robinson Crusoe. It is a gruesome story.

Since 1894 plague has been gradually spreading throughout the world. Beginning at Hong Kong in that year, it reached India in 1896; Japan, the Philippines, Australia and Egypt in 1899; the United States, at San Francisco, and South America in 1900; Chili and Peru in 1903, Ecuador and South Africa in 1908.

For several years quite a number of cases appeared in San Francisco. Modern methods of sanitation have held it in check, although occasional cases have been reported from other parts of California.

Etiology: Plague is caused by the bacillus pestis, discovered in 1894, at Hong Kong, by Kitasato and Yersin working independently. It attacks rats and other animals, notably, ground squirrels. It is gradually coming to be believed that the bacillus is transmitted to man by certain fleas that normally inhabit rats. All epidemics of plague in man are preceded and accompanied by plague in rats. Infection usually enters the system by way

of the skin. Flies and bedbugs, as well as fleas, carry the plague germ, and human beings may be infected by the bite of either. All the discharges of the plague patient contain the germs which live a long time when exposed; therefore clothing or utensils that become soiled are possible agents for their distribution.

Plague reaches new countries by way of the sea port towns first, probably in the rats that all ships carry. Thence it spreads most rapidly in unsanitary surroundings and among the poorer classes. Young and old are equally susceptible, though strangers do not seem so susceptible as natives.

Bacteriology: The bacillus pestis is an encapsulated non-motile, short non-spore forming aerobic organism, with rounded ends, 1 to 1.8 by 0.4 to 0.7 μ in size. It stains with the usual stains, and is decolorized by Gram's method. It is found in the buboes, pustules and pulmonary lesions, in the blood and in the discharges.

Morbid Anatomy and Pathology: The lymphatic glands show the greatest changes. There is edema of the gland and surrounding tissue. On section a thin straw-colored fluid runs out, containing pus cells, red blood corpuscles, and plague bacilli. The edema of the subcutaneous tissue may be very extensive. The buboes themselves are hard. The internal lymphatics are always swollen whether the external ones are or not. The glands break down causing necrosis. Secondary glands are swollen. The lymphatics connecting them are distended. Femoral buboes are most frequent, next inguinal, then axillary, then cervical and iliac. The respiratory tract is congested. Lobular pneumonia is common. Petechial eruptions occur in the skin. The spleen is much enlarged, the liver slightly so, the stomach and intestines may be congested.

Symptoms: Mild bubonic plague, pestis minor, is characterized by slight glandular swellings without fever. Numerous cases of this kind frequently precede by weeks or months, epidemics of the true or severe bubonic plague.

Incubation lasts from three to ten days. If prodromal symptoms exist the patient appears somewhat dazed. There are pains in the limbs, muscular weakness, and malaise. The tongue, at first coated, later becomes extremely dry and mahogany colored. Vomiting may occur.

In other cases the onset is abrupt. The patient has a chill, and the temperature rises to 102° to 107° F. or higher. The pulse is rapid, 100 to 130, usually very weak. The skin is dry, the patient thirsty. Constipation is the rule. Nausea and vomiting may occur. The patient is dazed, there may be apathy, less often delirium. There is loss of the power to articulate. This stage lasts two to five days; occasionally longer. In favorable cases the fall of temperature is gradual. Other cases have a return of the high temperature after two or three days.

Buboes, inflammation of the lymph glands, is the characteristic feature of bubonic plague. They are usually small and hard. One is often more swollen than the others. Surrounding the buboe is often a subcutaneous edema, which may extend for several inches in every direction. In fatal cases the glands remain small and hard. If the buboe suppurates it is considered a favorable sign. If it suddenly disappears it usually means death. Sometimes the buboes are tender, at other times not. The buboes vary from the size of an almond to the size of an egg. About 70 per cent. of the cases present the buboes in the groin, 20 per cent. in the axilla, 10 per cent. in the cervical region.

Purpuric spots sometimes appear in the skin, the "tokens" of the seventeenth century.

Hemorrhage is not uncommon. Hemoptysis is the most serious form. There may be hematemesis with black vomit. Hemorrhages may occur from the kidneys and from the uterus. Any hemorrhage marks the case as severe.

Broncho-pneumonia is a common symptom.

Pneumonic plague as a special form of the disease was described by Dr. L. F. Childe in 1897. It exhibits no primary buboe. It is ushered in by a chill. The dazed or apathetic expression is the same as in the ordinary form. The lungs give the physical signs of lobular pneumonia. Hemorrhage from the lungs frequently occurs. The sputa is filled with the plague bacilli. Death occurs in practially all cases.

The *septicemic* form lacks the initial buboe, but the patient succumbs in a state of collapse in two or three days.

Complications and Sequelæ: Complications and sequelæ have been covered in describing the symptomatology. Pneumonia

and hemorrhages from various parts are the principle complications.

The mortality of plague is so high, from 70 to 80 per cent., that sequelæ have no chance. Paralysis may rarely result.

Diagnosis: Typical cases of plague during epidemics are easy to diagnose. But the mild cases before, or the first true cases may be mistaken for something else. Various infectious diseases may have an adenitis associated with them, but the clinical histories will be different. Whenever possible a bacteriological examination will clear up the diagnosis.

Prognosis: In epidemics of plague the mortality may range from forty to ninety-five per cent. Many of the lighter cases recover. The pneumonic and septicemic forms are nearly always fatal.

Treatment: One of the most important prophylactic measures is to annihilate all the rats. Epidemics of plague in human beings are preceded by its appearance in rats and rats are supposed to be its carriers from place to place. In California it has also been found in the ground squirrel. One of the established rules where plague has appeared of late years, in Japan, in San Francisco, and in other places, is to give a bounty for dead rats and to have each carcass examined bacteriologically. Another point is to rat proof not only buildings, but communities. This is now being done in Tokio and in San Francisco.

Haffkine's immunizing serum is said to protect those inoculated for six months.

Individuals affected must be isolated, and houses and places quarantined.

All of the discharges must be sterilized and destroyed. Clothing and utensils must be thoroughly sterilized. Rooms and houses where plague occurs must be thoroughly fumigated. As the plague germs are carried by fleas and other insects, burning sulphur, two pounds to the thousand cubic feet of air space, is the only safe method of fumigation. Formaldehyde gas will not kill vermin.

Hygienically the most important factor is an abundance of fresh air. The patient must be put to bed and under no circumstances allowed to get up until entirely well. Sudden death

frequently occurs in convalescents by neglect of this procedure.

The only therapeutic treatment offered by the old school is by means of serums. Yersin and Haffkine have each prepared a serum which has been used, but the results are still uncertain.

Alcoholic stimulation is advised for the collapse.

Surgical treatment of the buboes is contraindicated.

Arsenicum album heads the list of homœopathic remedies in its similarity to plague. It closely fits the average case.

Apis is useful in cases with edema.

Belladonna is indicated when the buboes are very sensitive.

Lachesis is indicated in prostration with purpura.

Crotalus is indicated, and has been used successfully in hemorrhagic cases.

Cobra (Naja), 1 to 1,000, in glycerine—the 3x dilution—was used successfully by Major H. E. Deane, of the English army in India.

CHOLERA.

(Greek χυλερω, an eave-trough. The dejections stream from the intestines like rain from an eave-trough. Dunbar.)

Synonym: Asiatic Cholera.

Definition: Cholera is an acute infectious disease, characterized by violent vomiting and purging, cramps, collapse, and suppression of urine.

Historical Note: Cholera is endemic in India and has existed there for ages. It was first described in the sixteenth century. Accurate knowledge of the disease, according to Macleod, dates from 1817.

From India cholera has spread to other parts of the world by way of trade routes, causing epidemics at irregular intervals. The first European epidemic began in 1826. From there it reached America in 1832. Since then there have been a number of outbreaks in Europe, fewer in the United States. For the past few years cholera has been raging in India, and during 1909 and 1910 reached epidemic proportions in Russia.

Etiology: Infection in cholera takes place by way of the digestive tract and most frequently is due to contaminated water. It may also be transmitted by means of flies through the food, or

by lack of cleanliness in handling discharges from patients sick with the disease. Persons with digestive disturbances are more susceptible than those in perfect health. Old persons are more susceptible than young.

Bacteriology: In 1883 Koch isolated the comma bacillus from the feces of cholera patients. This has been accepted as the specific cause of the disease. It is a slightly curved rod, shorter than the tubercle bacillus, but thicker. Some are more curved than others. It is found only in the discharges or intestinal contents. It grows on gelatine, potato, agar or in bouillon.

Guinea pigs may be inoculated. The serum of immunized animals causes the comma bacillus to aggregate in clumps—the serum reaction. Injected into the peritoneal cavity of immunized guinea pigs the cholera bacillus dies—Pfeiffer's phenomenon.

Morbid Anatomy and Pathology: After death the body of the cholera patient appears shriveled and livid. Rigor mortis appears early and is long continued. Contraction sometimes takes place after death causing movements of the body. Hyperemia of the digestive tract is found. Sometimes there are ecchymoses. There may be a catarrhal condition of the intestines. Sometimes a diphtheria-like membrane, more rarely necrosis, is found. The small intestine shows more changes than the large. The bowel contents are like the stools before death. They contain detritus, epithelial cells, red and white blood cells and various organisms.

The liver is congested. The spleen is not enlarged. The kidneys show acute parenchymatous changes. The right side of the heart and large veins are filled with blood. The blood itself is of high specific gravity—both red and white cells are increased in quantity. The hemoglobin is increased.

The lungs are dry and shrunken. The muscles are dry and sometimes ruptured.

Symptoms: Incubation lasts usually from one to three days. Rarely it may last several days longer. More often it is but twelve to twenty-four hours.

During an epidemic of cholera there is a great difference in the severity of different cases.

The *typical case,* the *algid type,* may be preceded for a day or two by diarrhea, or it may set in abruptly. The patient suddenly becomes very weak. Violent diarrhea sets in of large, frequent, watery stools. At first these have a fecal character, but soon they change to the characteristic "rice-water" movements, almost colorless and odorless, and containing whitish particles. The microscope shows epithelia, triple phosphates, and numerous bacteria of putrefaction, besides the specific comma bacillus. There is no tenesmus. In extreme cases the stools may contain blood.

The *fascies cholerica* develops very rapidly at this time. The nose becomes pinched, the skin becomes dry and loses its elasticity. The color becomes grayish and livid, the lips blue. The surface temperature becomes subnormal, whereas by rectum it may be 102° to 104° F. or higher.

The heart at first may be tumultuous, but in a very short time it becomes very weak, the sounds almost inaudible, and the pulse may disappear altogether.

The voice becomes weak and may be lost. Respiration is shallow and difficult. The abdomen usually remains soft, occasionally it becomes hard.

Cramps in the various muscles become severe. These are usually worst in the calves, but also occur in the muscles of the thighs, abdomen and arms.

The urine becomes scant and may cease altogether for several days. What urine there is usually contains albumin and casts.

The mind frequently remains clear till the end, although the patient may become apathetic.

The majority of cases of the type just described die. In the others a period of reaction sets in when the symptoms rapidly disappear, and the patients recover in a few days.

Varieties: The *mild type.* Some patients, during an epidemic, have practically no symptoms. They may have at most a slight diarrhea which passes off in a few days, or after a few days may develop a typical case.

Cholerine: Other patients will develop an acute gastro-enteritis. There will be vomiting and diarrhea. The patient is more or less

prostrated. Mild cramps occur in the calves of the legs. The urine becomes diminished and may contain albumin. The voice is weak, the extremities cool, the pulse small and fast. This so-called "cholerine" type usually lasts a week or ten days. Some days the patient is better, some days worse.

Complications and Sequelæ: Some cases of cholera run into a typhoid condition, with fever, full pulse and various skin lesions.

Other cases develop local inflammations, like pneumonia, erysipelas or diphtheritic membranes of the pharynx, larynx, intestinal tract or genitals. Sometimes pyemia develops.

Other cases again develop a true uremic condition due to nephritis.

Diagnosis: The diagnosis can be certainly made only by bacteriological examination and the finding of the comma bacillus.

Prognosis: The prognosis of cholera is always grave as the mildest cases may suddenly develop alarming symptoms and rapidly prove fatal. In epidemics the mortality ranges from fifty to sixty per cent.

Treatment: Prophylactic. To prevent the spread of cholera all discharges must be promptly disinfected and destroyed, all bed clothing, wearing apparel and eating utensils must be sterilized. The patient must be isolated.

During the past few years vaccination with variously prepared antitoxins has been tried as a prophylactic. Haffkine's preparation has so far seemed to give the best results. In 1909 out of a population of 1,078 persons 122 developed cholera. Of the 122 only one had received immunization, the other 121 had not.

Any slight digestive disturbance must be cared for, as persons whose digestion is upset are much more susceptible than others.

The diet during the disease and for some time after must be light and nutritious.

Morphine is extolled by some authorities and condemned by others. It relieves the cramps. Koch gave opium to his experimental animals to lessen peristalsis and thus allow the bacilli to multiply.

Water by hypodermaclysis and intravenously and rectally is a help.

Hahnemann recommended as a prophylaxis *cuprum* 200, one dose a week, and *veratrum album* 200, one dose the next, in alternation throughout an epidemic.

Homœopathy received its original impetus as a generally accepted method of treatment because of its remarkable success in the cholera epidemic in Europe about 1830. The comparative mortalities were 40 to 50 per cent. old school, 5 to 10 per cent. under homœopathic treatment.

The best homœopathic remedies are:

Veratrum album for the acute vomiting and purging with collapse.

Cuprum for cases where the cramps are excessive.

Camphor is an excellent remedy in the beginning. It must be frequently repeated. It may also be used as a prophylactic.

In a letter dated April 21, 1832, Hahnemann said that whoever took care of the cholera patient should take a few drops of spirits of camphor on sugar beforehand for protection. He also said that camphor fumes would destroy cholera contagion, and recommended that spirits of camphor should be distributed free during an epidemic. These statements occurred in a letter written to a friend in Holland who asked for advice. This and several other letters first appeared in the *Leipziger Populaere Zeitschrift*, and were translated by me and printed in the *Chironian* for September, 1911.

Phosphorus, phosphoric acid, carbo vegetabilis and *arsenicum* are also mentioned by Joslin in his little book on cholera published in 1849 and based largely on personal experience.

DYSENTERY.

(Greek, δύς, difficult; ἔντερον, bowels.)

Synonyms: Tormina. Bloody Flux.

Definition: Davidson (Allbutt and Rolleston System of Medicine) says: "Dysentery is the clinical expression for a group of congestive or inflammatory diseases of the large intestine, often terminating in necrosis, ulceration or gangrene; characterized by frequent mucous or serous stools, mixed with blood, and generally accompanied by more or less tormina and tenesmus, with or without fever."

Historical Note: Dysentery has occurred sporadically in all countries from the beginning of history. According to Shiga it is mentioned in the Papyrus Ebers as occurring in Egypt as early as 1600 B. C. Dysentery has also appeared in epidemic form at various times and places.

Two forms of dysentery are now described: (1) *Bacillary* dysentery, and (2) *Amebic* dysentery.

(1) In 1889 Shiga isolated and described the bacillus dysenteriæ in Japan, and in 1900 Flexner isolated and described a similar bacillus in the Philippines. Since then much investigation has been done by many observers confirming their work.

(2) The ameba dysenteriæ found in the amebic form was first described by Laube in 1859, and in 1875 by Lösch in St. Petersburg. In 1890 Osler found a case in his wards at Johns Hopkins. Since then this form has been found to be most common in the United States.

Both the bacillary and the amebic forms are found almost everywhere. In Manila of 1,328 cases studied 712 were bacillary, 561 were amebic.

Etiology: No age, sex, or race is exempt. Epidemics are probably produced by infection from some preceding case. Ingestion of the specific bacillus has caused dysentery in experiments in animals, in experiments on condemned criminals, accidentally in laboratory workers.

The bacilli and the amebæ of the respective forms of the disease are found in the discharges. Carelessness in handling the discharges themselves or in handling clothing, bedding or other articles soiled by them may spread the disease. Flies and other insects may carry the disease direct to susceptible persons, or may contaminate food and so spread it. Numbers of epidemics have been traced to infected water.

In temperate climates dysentery prevails more in hot weather and early autumn. In tropical countries it occurs all the year round. In such places altitude seems to lessen its incidence, probably because the heat is less intense.

Bacteriology: (1) The bacillus dysenteriæ is a short, thick rod, somewhat thicker than the typhoid bacillus. Slightly motile in its

early life. It grows on all culture media and is decolorized by Gram's method. It exhibits agglutination reaction with the serum of dysenteric cases. It is found in the stools, the intestinal contents and in the ulcerations of the intestines.

(2) The ameba dysenteriæ is from 1 to 20 μ in diameter. Place a drop of discharge containing mucus or blood on a slide and cover with a cover-glass. If the slide is gently warmed the ameba will be seen to move.

Morbid Anatomy and Pathology: In the bacillary form the large intestine presents an inflammatory condition with thickened mucosa. There may be a superficial necrosis. Sometimes the ileum is involved.

In the amebic form there are large areas of ulceration of the large intestine. The liver shows parenchymatous changes, and there may be one or more abscesses.

There is also a diphtheritic form of dysentery with membranous sloughs.

Symptoms: The clinical symptoms of dysentery are more or less sudden onset of diarrhea, with some pain over the bowels. The stools at first contain mucus, but in twenty-four to forty-eight hours blood is mixed with them, or they may be almost pure blood. Tenesmus is marked. There is almost constant desire for stool, and the movements may occur every few minutes. There may be fever up to 102° to 103° F. The pulse becomes rapid and weak and the patient very much exhausted. The tongue at first coated, becomes red and glazed. In most cases the mind remains clear. In others a low delirium sets in.

If the case recovers the stools and tenesmus gradually subside, the movements again take on a fecal character, and the patient convalesces.

Sometimes the case takes a chronic form, the movements become less frequent—three or four a day—but continuing for months. Most of these patients become emaciated, although some may keep up a fair degree of nutrition.

The summer diarrheas of children are frequently due to the bacillus dysenteriæ.

Complications and Sequelæ: Abscess of the liver is the most

frequent complication in the amebic form of dysentery. It is rare in the bacillary form.

Peritonitis sometimes complicates dysentery.

Malaria has been noted as a complication, also neuritis. Osler says Bright's disease is sometimes a sequel.

Diagnosis: Dysentery is characterized by the frequent bloody stools and tenesmus. The bacillary and amebic forms must be differentiated by the microscope. The agglutination test can also be used for the bacillary form.

Syphilis and carcinoma of the rectum may cause bloody stools, but would hardly be confounded with dysentery.

Typhoid fever may for a time be mistaken for dysentery, or vice versa, but the clinical course is different.

Prognosis: In bacillary dysentery some epidemics have a mortality as high as 20 or 30 per cent. In other forms the mortality is not so high. Cases may become chronic with a finally fatal issue.

Treatment: The patient must be put to bed and kept as quiet as possible. All discharges must be promptly and thoroughly disinfected to prevent secondary cases. All soiled clothing, bedding and utensils must be thoroughly disinfected.

Warm applications to the abdomen may be of service.

Acidulated drinks may be given or carbonated waters. The diet must be of the simplest. In the beginning broths, gruels and white of egg, a little every two hours. In subacute or chronic cases milk plain, or milk and lime water, milk and Vichy, or milk peptonized can be used. Return to the usual diet must be gradual.

Colon irrigation may be used of nitrate of silver, 10 to 30 grains to the pint, using large injections. Osler says a cocaine suppository may be used just to allay the irritability of the bowel. In the amebic form warm injections of a solution of quinine, 1-5,000 to 1-1,000, have been used at Johns Hopkins. Dr. Winchell, of Brooklyn, has found this very satisfactory.

Morphine may have to be given for the pain and tenesmus.

In South America *ipecac* in large doses, 20 to 40 grains of the powder, is the principle medicine. Shiga says it is useless in Japan.

Ipecac may be of service in homœopathic doses when indicated.

Mercurius corrosivus is the main reliance of the homœopathist, as it covers most closely the tenesmus and the character of the stools.

Aloes has tenesmus, frequent stools with cutting pains in the abdomen, and much gas.

Arsenicum album is indicated where the stools are ill-smelling, sometimes bloody, the patient much exhausted, and restless from his exhaustion. I have used *arsenic* after *mercurius corrosivus* with success sometimes.

Colocynth may be useful where there is colicky pain relieved by bending double.

Nux vomica is indicated after the abuse of other remedies.

Other remedies that may be thought of are *belladonna, carbo vegetabilis, china, podophyllum.*

CEREBRO-SPINAL FEVER.

Synonyms: Epidemic Cerebro-spinal Meningitis. Petechial Fever. Spotted Fever.

Definition: Cerebro-spinal fever is an acute infectious disease characterized by an inflammation of the meninges of the brain and spinal cord caused by the diplococcus intracellularis. It is accompanied by irregular systemic disturbances. Many cases have a petechial eruption, hence the names "spotted fever" and "petechial fever."

Historical Note: Cerebro-spinal fever was first described in 1805. Since then it has appeared in epidemic form in various parts of Europe and of the United States. In 1887 Weichselbaum discovered the diplococcus intracellularis, the specific germ of cerebro-spinal fever. In 1903-5 the disease prevailed quite generally in this country. In New York City in 1904 there were 3,029 deaths from cerebro-spinal fever, in 1905 there were 1,404 deaths. In 1909 there were 485 deaths from it in the entire state of New York.

In 1907-1908 Simon Flexner published the results of the treatment of cases of cerebro-spinal fever by the serum prepared by him.

Etiology: Cerebro-spinal fever is due to the bacillus intracellularis first described by Weichselbaum in 1887. Its mode of entrance into the body is probably by way of the nasal mucous membrane.

Unfavorable hygiene and cold weather seem to be predisposing causes. It is found most often in the poor quarters of large communities.

The disease occurs mostly in children and young adults. It frequently affects recruits in an army.

Nurses and medical attendants on meningitis cases are sometimes attacked.

Bacteriology: The meningococcus, or the diplococcus intracellularis, was discovered by Weichselbaum in 1887. It is a diplococcus consisting of paired hemispheres. It is found in the cerebro-spinal fluid in cases of epidemic cerebro-spinal meningitis. It is found frequently in the leucocytes, sometimes in the nucleus as well. It is sometimes found in the exudate in complicating arthritis and pneumonia.

The meningococcus will grow best in blood serum or agar-agar. It will not survive long under cultivation.

Morbid Anatomy and Pathology: In cerebro-spinal fever there is an excess of cerebro-spinal fluid. Instead of the fluid being clear, as is normal, it is at first cloudy or turbid, and later becomes fibro-purulent.

On examination by the microscope the meningococcus will be found in the fluid.

The meninges show thickening and changes due to inflammation.

A leucocytosis is the rule.

Symptoms: The onset of cerebro-spinal fever is usually abrupt. There is headache, retraction of the head and back, vomiting, chilliness, sometimes chills, frequently delirium and convulsions, and sometimes coma. All of these symptoms may be so sudden and so severe that the patient, overwhelmed by the toxemia, succumbs in a very few hours.

Other cases are ushered in abruptly with a chill or chilliness. There are headache and pains in the neck, back and legs. The

head is drawn back and there may be even opisthotonos. Sometimes the bending of the body is sideways, pleuro-thotonos, instead of backwards.

Convulsions are frequent, coma less so. Delirium may be an early symptom, especially in children. Projectile vomiting takes place.

The temperature rises to 102°, sometimes to 106° F. or higher. Chapin reports a case where the temperature was 108.6° F. The pulse and respiration are increased but the increase is variable and irregular.

Herpes appears about the face, and a petechial eruption often appears on the body. This last may be slight, or it may be profuse. In malignant cases the eruption may be purpuric. Chapin reports that the skin eruption was rare in the epidemic of 1904.

The various reflexes—the patellar, plantar, epigastric, either become exaggerated or are lost. Kernig's sign is present. This is an inability to straighten the leg when the thigh is at right angles to the body.

Eye symptoms are marked. There may be photophobia, often strabismus. Frequently the sight is lost. The eyes may be open but the patient apparently sees nothing. There is anesthesia of the eyeballs. On account of this lack of sensibility conjunctivitis is liable to develop.

Deafness is also a common symptom.

The excess of fluid in the ventricles permits the eliciting of Macewan's sign. This is done by having the patient sit upright with the head to one side. Percussion over the parietal or lower part of the frontal region gives a tympanitic note.

Hyperesthesias and anesthesias may be found in various parts of the body. Maier found a true muscle soreness rather than a hyperesthesia.

Twitchings of muscles or groups of muscles occur during the disease. Later the body may be in a state of tonic spasm.

Paralysis of different nerves occurs.

Leucocytosis is the rule.

Complications and Sequelæ: A more or less frequent complication of cerebro-spinal fever is pneumonia.

Hydrocephalus sometimes is a complication.

Sequelæ are usual and very distressing. One or more of the special senses may be permanently destroyed, particularly sight and hearing.

The brain may be permanently injured and the patient become idiotic.

Joint contractures may result.

Emaciation is common.

Diagnosis: Clinically, epidemic cerebro-spinal meningitis may resemble typhus fever, a comparatively rare disease in the United States. The retraction of the head is not present in typhus, it is in cerebro-spinal fever.

Tubercular meningitis and cerebro-spinal fever may resemble each other closely. The first is of slower onset; moreover, it is apt to be part of a general tubercular infection, or to occur where there is a family history of tuberculosis.

Typhoid fever has a very different history from cerebro-spinal fever.

In all cases of suspected cerebro-spinal fever lumbar puncture should be made and some of the fluid drawn off. In this disease the fluid is turbid, not clear, and the microscope will show the meningococcus.

Lumbar puncture is made primarily for diagnosis. It has also been found that lumbar puncture, by relieving intra-dural pressure, is beneficial as a method of treatment.

Technique: The skin where the puncture is to be made should be sterilized. This point of election may be between the third and fourth, or the fourth and fifth lumbar vertebræ, or between the fifth lumbar and the first sacral. An aspirating needle, or a small trocar and canula, may be used. It should be 9 to 10 cm. long with a lumen of 1mm. The needle should be inserted one-half cm. below the spinous process of the vertebra it is decided to puncture under, and one cm. to the right or left. The needle is then pushed upward, inward and forward. The canal is reached at a distance of 2 to 7 cm., according to the size and age of the patient, whether child or adult. If the canal is reached the cerebro-spinal fluid will run out drop by drop, if not there will be nothing.

Sometimes when the fluid is gotten a little blood is mixed with it. The microscope will differentiate the blood elements from other substances. The normal fluid is clear, in cerebro-spinal fever it is turbid.

Prognosis: The prognosis of cerebro-spinal fever is bad. The mortality is high. Patients who do not die are nearly always left in such a pitiable condition, with loss of mind, or loss of sight or hearing, or with some deformity, that it would seem as though death were preferable.

Treatment: During the New York epidemic of cerebro-spinal meningitis of 1904-1905 a special commission was appointed by the city board of health to investigate the entire subject. They concluded that the disease was undoubtedly mildly infectious. They also found that the contagion was probably transmitted by the nasal discharges.

In the light of these findings the patient suffering from epidemic cerebro-spinal meningitis should be isolated. He should be placed under the most advantageous hygienic surroundings. All discharges—particularly from the nose and throat—should be immediately destroyed.

The diet must be liquid at first. It should be full and nourishing during convalescence. It may be necessary to feed with a stomach tube.

Morphine may be used to quiet the patient. Ice bags applied to the head and spine are valuable adjuncts.

In 1907 Simon Flexner made known his antimeningitic serum prepared from the horse. Since then it has been largely used and has apparently reduced the mortality from 75 to 80 per cent. to less than thirty.

In 1908 Flexner and Jobling presented a paper to the American Pediatric Society reporting on 393 cases treated with the serum. The mortality was 25 per cent.

The following is a summary of the cases treated:

	Cases.	*Recovered.*	*Died.*	*Mortality.*
Under one year.....	22	11	11	50 %.
Between one and two years.	19	11	8	42.1%.
Between two and five years..	68	52	16	23.5%.
Between five and ten years..	79	70	9	11.4%.

Between ten and twenty years 105	80	25	23.8%.
Over twenty years..... 87	64	23	26.4%.
Unknown years 13	7	6	46.1%.

As to the period of injection:

	Cases.	*Recovered.*	*Died.*	*Mortality.*
1-3 days	123	107	16	13.5%.
4-7 days	126	96	30	23.8%.
Later	112	73	39	35 %.

Terminated by lysis, 200; by crisis, 70.

Injections of the serum clear the turbidity of the spinal fluid, they likewise cause the meningococcus to disappear. The leucocyte count goes down. The mentality clears up. The retractions and Kernig's sign do not disappear quite so rapidly.

At the same meeting of the American Pediatric Society, Dunn, of Boston, reported a successful use of the serum. He injected a dose of 20 to 30 cc. directly into the spine. If the patient was much improved in twenty-four hours the dose was not repeated. If there was not marked improvement the dose was repeated every twenty-four hours till a change for the better was noted.

Lumbar puncture, which should always be made for diagnostic purposes, has also been found to be of value as a method of treatment. (See above for technique.) There is an excess of fluid in cerebro-spinal meningitis, and puncture will take away the excess, thus relieving the pressure symptoms. Lumbar puncture may be repeated as often as occasion demands. I have known cure to result after many punctures extending over several months.

There are many homœopathic remedies that may be indicated in this disease. Among them are *gelsemium* for the chilliness, fever and stupor.

Belladonna for the sthenic cases with delirium.

Apis for the relief of pressure symptoms due to excess of fluid.

Cicuta for the opisthotonos.

Hellebore for the excess of fluid, especially if hydrocephalus is present, and other nervous symptoms.

Cimicifuga for tonic and clonic convulsions.

Opium, in potency, for stupor and opisthotonos.
Other remedies are *cuprum aceticum* and *zinc cyanatus.*

ACUTE ANTERIOR POLIOMYELITIS.

Synonym: Infantile Spinal Paralysis. (So-called because it occurs most often in children.)

Definition: Acute anterior poliomyelitis is a systemic disease, probably infectious, characterized by acute onset, with fever, the rapid development of local paralyses with wasting of the affected groups of muscles. The paralyses frequently leave permanent deformities.

Historical Note: Acute anterior poliomyelitis is said to have been accurately described first by Jacob von Heine in 1840. On account of its peculiar clinical history it has long been supposed to be an infectious disease. In 1910 Lewis and Flexner announced that they had been able to transmit the disease experimentally to monkeys. The virus seems to be located in, or to gain entrance by, the naso-pharynx.

Etiology: The specific organism has not yet been found. The majority of cases occur during warm weather.

By far the great majority of cases occur in young children between the ages of one and four years, though some cases occur in adults. Both sexes are equally susceptible.

Many observers have noted its appearance following the other acute infections. Exposure to chill, or a sudden checking of perspiration, sometimes seem to be exciting causes.

In 1912 the stable fly was accused of conveying the disease from one person to another.

Morbid Anatomy and Pathology: The lesion is in the anterior gray matter of the spinal cord. The blood vessels are much congested and surrounded by an exudate of round cells in the lymphatics. Hemorrhages may be seen. The texture of the gray matter is looser than normal. As the disease progresses the ganglion cells degenerate. The gray horns may shrink or they may become sclerosed.

Symptoms: The disease frequently occurs very abruptly without premonitory signs. Such cases are supposed to be due to a hemorrhage or to a thrombosis into the anterior horns.

The majority of cases start in in this way:—there is a sudden rise of temperature, to 105° F. or more. There is headache, occasionally convulsions, and in the beginning, especially in epidemics, some cases will have a certain amount of retraction of the head. Sometimes there is vomiting, less often diarrhea. Usually this stage of the disease lasts not longer than a week.

In rapid cases within a day or two, in others at the end of a week or longer, it will be noticed that the patient is paralyzed in the legs or arms, sometimes only one, sometimes all four, more often the legs only. With this paralysis atrophy of the affected muscles sets in. Occasionally the muscles of the trunk or face are attacked. At first the paralysis is quite extensive. It develops rapidly in one to six weeks, and then begins to subside until finally one group of muscles is left permanently disabled. This is more often in one leg, but sometimes in an arm. A few cases entirely recover. Even in those permanently disabled there may be some improvement as time goes on, but complete restoration of function is rare. The stage of improvement goes on for six months to a year then ceases.

Other than the paralysis and atrophy and the resultant deformity the patient becomes entirely well.

At the height of the disease there is no sensory disturbance and no trophic lesions result.

In the affected part there is the reaction of degeneration to electricity. The tendon reflexes are lost. If the disease has appeared in early childhood there is a general lack of development of the affected part which adds to the deformity.

Complications and Sequelæ: Neuritis sometimes accompanies acute anterior poliomyelitis.

Occasionally musclar atrophy develops late in life in those who have had infantile paralysis earlier.

Deformities due to permanent paralysis are frequent.

Diagnosis: The diagnosis of acute anterior poliomyelitis is usually easy.

Multiple neuritis may sometimes be confounded with acute anterior poliomyelitis. The former is usually symmetrical and does not limit itself to a group of muscles; it has wrist drop and foot drop, and there are sensory disturbances.

Prognosis: In some epidemics the mortality is as high as ten per cent. These cases die within a few days. After the stage of improvement sets in the prognosis as regards life is good. The prognosis as regards deformity is not so good, as nearly all cases are permanently disabled in some way. Treatment must be persevered in for months and years.

Treatment: The most important factor in the treatment of anterior poliomyelitis at the beginning is absolute rest in bed, but the patient must not lie on the back all of the time. When paralysis sets in the paralyzed parts must be kept warm.

Infantile paralysis must be handled like any other infectious disease, that is, the patient must be isolated, and all bedding, clothing and utensils coming in contact with the patient must be sterilized. All the discharges from the body must be destroyed, particularly discharges from the nose and throat. Lewis and Flexner have found the discharges of the nose and throat to be infectious, although they have not isolated a specific germ. Peroxide of hydrogen, 1 per cent., kills the virus, according to Morse. This may be used as a nasal spray.

The patient must be fed up and kept well nourished.

Starr says warm baths are serviceable. Stumpell recommends ice caps at the very beginning.

During the acute stage hygienic treatment and the indicated homœopathic remedy are best.

After the acute symptoms have subsided, six weeks to two months after the onset, massage and electricity may be added to the treatment in an effort to restore the paralyzed parts. This part of the treatment must be continued for at least a year. Either the galvanic or the faradic current may be used.

Aconite is useful in the beginning. It may also be continued if neuritis is a marked symptom. It is good for the fever, for restlessness and for nerve pain.

Gelsemium is another remedy that may be indicated. The patient is dull, the fever quite high, paralysis is present.

Belladonna is indicated in high fever with hot dry skin and congestion of the face.

Calcarea carbonica may be indicated in fat, flabby children or in marasmic cases. Both conditions are forms of malnutrition.

Cowperthwaite recommends fluid extract of *ergot* in three to ten drop doses every three hours.

Other remedies may suggest themselves.

LEPROSY.

(Greek, λεπρα, leprosy.)

Synonyms: Lepra Arabum. Elephantiasis Græcorum.

Definition: Leprosy is a chronic systemic infectious disease characterized by macules, infiltrations or tubercular lesions of the skin and mucous membranes, accompanied by disturbances of sensibility of the parts affected. Sometimes the lesions go on to ulceration.

Historical Note: Leprosy is one of the oldest of known diseases. It is frequently mentioned in the Bible, although it is supposed that syphilis and leprosy were confounded. The lepra bacillus was discovered by Hansen in 1871.

Etiology: Leprosy is endemic in certain parts of all the continents and in many of the islands of the sea. Nearly always these places are on or near the sea coast. Jonathan Hutchinson, of England, believes, on this account, that it is due to eating decayed fish. After a tour of many leprous communities Hutchinson stated that leprosy was more prevalent among the Roman Catholics of these places on account of their eating fish on Fridays and fast days.

There is some doubt as to whether the disease is hereditary. Healthy children have been born of leprous parents and have remained healthy. On the other hand, cases are often found in blood relatives. Such cases have usually been subjected to the same influences as those who already had the disease, so it is impossible to say how much is due to heredity and how much is due to environment.

The bed bug has been accused of transmitting the disease. Something more than contact with the patient seems to be necessary, as in many instances the husband or the wife is diseased, while the other partner and the children remain healthy. In places where it is endemic the victims live in unsanitary surroundings.

Hansen made a special trip to this country to trace the one

hundred and sixty known Norwegian lepers who had settled in our Northwestern States. Not one of their descendants to the great-grandchildren had ever developed the disease.

Where leprosy is not endemic no physician or attendant in charge of cases has been known to contract the disease.

The virus probably enters through some abrasion of the skin or mucous membrane.

Bacteriology: The bacillus leprâ, discovered by Hansen, in 1871, closely resembles the tubercle bacillus in appearance and in its relation to stains. It is larger in size, and is difficult to cultivate.

Morbid Anatomy and Pathology: In well marked cases of leprosy there is a macular eruption which later becomes nodular in character. The skin becomes swollen and tumefied. Ulceration may develop, and bullæ may appear on the ends of the fingers and toes. The nose and larynx may become involved. Neuritis with resulting contractions may occur.

The bacilli are found in the affected areas.

Symptoms: In places where leprosy is not endemic, like New York City, cases appear, but they are either imported, or else are cases occurring in persons who have lived for a considerable time in a leprous community. One of my patients lived in a leprous country twenty years; four years after coming to New York leprosy appeared.

The period of incubation is long and uncertain. Cases have developed in persons after an absence of ten, twenty or even forty years from all known sources of infection.

There are ill defined prodromal symptoms that may last for months. One of my cases preceding the development of skin lesions, had lost some fifteen pounds in weight and was weak and drowsy most of the time. As Duhring remarks, in countries where leprosy is endemic these long continued prodromata would excite suspicion. In this country, where leprosy is not endemic, they would be put down to tuberculosis, or malaria, or some other cause.

There are two well marked types of leprosy, (1) the non-tubercular, macular or anesthetic, and (2) the tubercular or

nodular. Characteristic cases are seen of each type, and cases are seen presenting features of both types. It is said that all tubercular or nodular cases are macular at first; but all macular cases do not necessarily become tubercular.

(1) *Non-tubercular type:* In the beginning brownish or reddish discolorations of the skin appear. These spots are quite large and irregular in outline, and appear on all parts of the body—face, trunk, and extremities. They tend to fade from the center, new spots appearing to take their place. At first they are not raised, later they are. Usually there is disturbance of sensation over the affected areas, commonly anesthesia.

A neuritis may appear at this stage causing some deformity, particularly of the fingers. In one of my cases the little finger of each hand was claw-like.

(2) *Tubercular type:* As the disease progresses, tumefaction of the skin sets in and nodules appear. This process is especially liable to attack the face. When severe the ears are enlarged, the nose becomes big, the lips thickened, and the whole expression is changed,—the so-called "leonine face."

Late in the disease the ends of the fingers and toes become more or less anesthetic, bullæ appear on them, and sometimes ulceration takes place. In extreme cases the terminal phalanges may slough off. In one of my cases the handling of hot dishes raised blisters. Ulceration may occur at any nodular point on any part of the body. One of the Chinamen under my care at the Metropolitan Hospital was literally covered with ulcerations when he entered the hospital.

When the nose and larynx become involved they may be so swollen as to interfere with breathing. Sometimes the eyes become affected and the patient becomes blind. One of my patients, a Dane, was so afflicted.

After symptoms appear the course of the disease is slow, taking a number of years to reach its full development. It may be marked by periods of advance and quiescence.

Complications and Sequelæ: Invasion of the larynx, sepsis and gangrene are some of the end results of leprosy. Kidney lesions are said to develop in about thirty per cent. of the cases, tuberculosis in about twenty per cent.

Diagnosis: In its early stages leprosy may be mistaken for syphilis. The two diseases may co-exist. The unfolding of the symptoms of each is very different. The eruption of syphilis, early, is always in much smaller spots than that of leprosy. In syphilis bad enough to present ulceration the history should guide us. The microscope will serve to differentiate.

In lupus the nodules are small and more circumscribed, and do not produce the extensive thickening of the brows and ears that leprosy does. Lupus is usually limited to one or two places, whereas leprosy affects numerous areas on every part of the body from head to foot.

In leucoderma or vitiligo the change of color, loss of pigmentation, is the only evidence of disease. In leprosy the skin becomes red or brown, later thickened, and the maculæ are either anesthetic or, less often hyperesthetic.

Prognosis: The prognosis of leprosy is bad. The disease runs a course of eight to fifteen years. A few cases recover. A good many survive the active stage of the disease and live for years with more or less deformity. Abraham says about half the deaths are caused by invasion of the disease into the larynx and other organs and to pyemia and gangrene. About twenty per cent. are caused by tuberculosis, about thirty per cent. by kidney complications.

Treatment: As the exact mode of infection is unknown, the only method of prophylaxis is to avoid residence in a leprous country.

Patients should be placed under the best possible hygienic conditions. Removal to a benign climate is always of benefit. The diet should be generous and nutritious.

Ulcerations will have to be treated antiseptically and surgically.

Chaulmoogra oil, a fixed oil made from the gynocardia odorata, an East Indian tree, is the standard remedy of the old school. It is given in capsules of a few drops up to one hundred drops daily. A few cures have been reported.

The late Henry M. Dearborn reported one case cured with *hydrocotyle.*

Belladonna helped two of my cases during acute exacerbations with fever and reddened skin.

Arsenic was used in one case with ulcerations.

Sepia may be used for the brown discolorations of the skin.

Lachesis may be indicated in the severer forms with nose and throat involvement.

ACUTE MILIARY TUBERCULOSIS.

Definition: There is a general infection of many of the organs of the body in this form of tuberculosis. Buhl—according to Osler—declared "miliary tuberculosis to bear the same relation to the primary lesion as pyemia does to a focus of suppuration."

Historical Note: Buhl was the first to give a definite idea of miliary tuberculosis by stating that the infection was distributed by the blood from some primary focus. Weigert was the first to show that in these cases there was tuberculosis of the blood vessels, especially of the large venous trunks. Ponfick found in some cases tuberculosis of the thoracic duct. It is thus easy to see how the infection may be sent to the various organs.

Etiology: There must be a focus somewhere. It may be a tubercular gland, or sometimes a long standing tuberculosis of the bones or joints is the starting point.

Strümpell says it frequently follows pleuritic effusion—which was itself tubercular in origin.

Occasionally miliary tuberculosis follows some other of the acute infections, as typhoid fever or measles.

Bacteriology: The bacillus tuberculosis is found in the affected parts.

Morbid Anatomy and Pathology: The tubercles are generally distributed throughout the organs of the body in miliary tuberculosis. According to Strumpell the lungs, liver and spleen are always affected; next in frequency the kidneys, thyroid gland, marrow of the bones, heart, and choroid; finally the serous membranes and meninges.

In one of my cases, a girl, aged 17, tubercles were found everywhere at the autopsy; they were thick on the meninges of the brain, the cerebellum and in and on the heart; they were also in the lungs, spleen, kidneys, liver and bladder.

Symptoms: Miliary tuberculosis is frequently overlooked on

account of the irregularity of the symptoms. The diagnosis is often made only at the autopsy table. The general run of cases may be grouped in three divisions, according to the predominating symptoms, always remembering that cases may occur that will not exactly fit in any of them.

1. The *typhoid form.* In this form of acute miliary tuberculosis the patient quite suddenly becomes very sick. There is fever, rapid pulse, possibly some cough and increased frequency of respiration. The respirations are noticeably deep. As no special symptoms pointing to lesion of any particular organ appear, the patient may be thought to have typhoid fever. Clinically the case may have all the appearance of typhoid, even to a rash. As the disease progresses the patient becomes rapidly worse, the temperature rising and the pulse increasing in rapidity. After two or three weeks, as a rule, either lung or brain symptoms develop giving a clue to the true condition. Quite a characteristic symptom is an increasing cyanosis and dyspnea without very definite physical signs in the lungs. Or there may develop a rigidity of the muscles of the neck with unconsciousness or convulsions. Tubercles are said to appear in the choroid where an expert may detect them with the ophthalmoscope.

2. The *pulmonary form.* In this form the symptoms may point more especially to the lungs. The disease is apt to set in quite abruptly, the patient will cough and have pleuritic pains. But the disease progresses continuously to a fatal termination. The general symptoms, fever, increased pulse rate, cough and dyspnea are marked.

3. The *meningeal form,* or *tubercular meningitis.* In this form the disturbance of the nervous system is profound. There is headache, stupor, increasing to coma, possibly convulsions. There is rigidity of the neck muscles with a tendency to bend the head backward. The respirations are deep and rapid, and the cyanosis marked. Cough may or may not be present.

Complications and Sequelæ: Cases of miliary tuberculosis are practically always fatal and leave no chance for complications.

Diagnosis: A patient who suddenly becomes severely ill, with great prostration, progressive wasting and anemia, high and irregular fever, rapid pulse, deep and hurried respirations, more

or less cough and marked cyanosis, without definite symptoms pointing to some special lesion, should always suggest miliary tuberculosis. This is especially true if the patient is known to have had some comparatively insignificant or quiescent tubercular lesion for a considerable time.

The most frequent source of error is in mistaking miliary tuberculosis for typhoid. Clinically typhoid is more apt to run a regular course. The typhoid temperature curve is quite characteristic. The microscope may be necessary to make sure in doubtful cases. The demonstration of the typhoid bacillus or of the tubercle bacillus will make the diagnosis almost certain. Demonstration of the Widal reaction will show the case to be one of typhoid. The various tuberculin reactions will be of help in tuberculosis. Both diseases may occur in the same patient.

The meningeal form, tubercular meningitis, can be positively diagnosticated by lumbar puncture and examination of the spinal fluid under the microscope.

Prognosis: The prognosis is bad. Practically all the cases die. If a case of miliary tuberculosis recovers, it is probable that a mistake has been made in diagnosis.

The patient referred to above that came to autopsy, was a girl aged 17 years. When she entered the hospital she could walk. She grew rapidly worse and died in seven days. She was unconscious for two days before she died.

Treatment: Treatment can only be palliative. Sponge baths are of service in the fever. The diet should be light and nutritious. Somatose, a dram once or twice a day, dissolved in water or in milk may be added to the diet.

In the meningeal type—tubercular meningitis—inunctions of iodoform through the scalp are said to have been of service.

Lumbar puncture may be of temporary benefit in tubercular meningitis.

The use of *tuberculin* in potency may be tried.

Iodium may help some in cases with great emaciation.

Calcarea carbonica has given some help in very young children with symptoms of marasmus.

Inunctions of *iodoform* may also be of some benefit in very young children with symptoms of marasmus.

TUBERCULOSIS OF THE LUNGS.

(Greek, *φθωις*, to consume.)

(Latin, *tuberculum*, nodule.)

Synonyms: Pulmonary Tuberculosis. Phthisis. Consumption.

Definition: The name *tuberculosis* is given to this disease because after death caseous nodular masses, or tubercles, are found in the lungs.

It is called *phthisis* or *consumption* because tuberculosis of the lungs is always accompanied by wasting of the muscular tissue, and in far advanced cases there is much emaciation.

Tuberculosis of the lungs is a systemic disease accompanied by fever, wasting and with local destruction of lung tissue.

Tuberculosis may attack any organ of the body, but the lung cases far outnumber all other cases combined.

Historical Note: Tuberculosis is an ancient disease. Various theories have existed as to its cause, but it was not until 1882 that Robert Koch published his studies and the discovery of the tubercle bacillus. This is now accepted as the specific organism. The disease has, at various times, been considered infectious, and at other times as hereditary.

Etiology: Much has been written as to the etiology of tuberculosis and many ideas have at one time and another held the attention of the medical world. It is, perhaps, sufficient to note here that the specific cause is the tubercle bacillus. That experiment shows that whether this organism be ingested or whether it be breathed in, pulmonary tuberculosis may be the result. Robert Koch and his followers believe that inhalation is the most frequent mode of infection. Calmette, Landouzy, Ravenal and other competent observers believe that ingestion is the most frequent mode of infection.

But the introduction of the germ into the body is not in itself sufficient to produce tuberculosis. The tubercle bacillus is omnipresent; everyone must inhale and ingest millions of them, yet the majority of us escape infection. There must be some special susceptibility of the individual affected necessary to

admit of development of the disease. What that susceptibility is we do not know.

It used to be thought that tuberculosis was hereditary. Recently the idea has been advanced that the children of tuberculous parents are to a certain extent immune.

In 1903 Von Behring advanced the theory that infection took place by ingesting infected milk during infancy, that the disease remained latent for months or years as the case might be, and then manifested itself. Whether Von Behring's idea is correct or not, the belief is growing that infection does take place early in life in all cases, and that there is a long latent or incubation period. In 1910, Philip, of Edinburgh, reported on a group of cases that he had followed for many years, in all of which he had been able to detect enlarged glands during childhood. Whatever may be the method of infection I do not believe the average healthy adult is easily infected.

Cases of tuberculosis may present themselves for treatment at any age. Of 9,903 cases at the Tuberculosis Infirmary of the Metropolitan Hospital, classified by ages, there were 20 under ten years of age, 869 from eleven to twenty, 2,222 from twenty-one to thirty, 2,513 from thirty-one to forty, 2,027 from forty-one to fifty, 1,269 from fifty-one to sixty, 616 from sixty-one to seventy, 345 from seventy-one to eighty, 20 from eighty-one to ninety, 2 from ninety-one to one hundred. Of the 9,903 cases, 6,762, or 68.3 per cent. were between the ages of twenty-one and fifty. Of 600 women patients in my service 494, or 82 per cent. were between the ages of sixteen and forty-five, the child-bearing period. This does not mean that the tuberculosis began at these ages, it simply means that the patients broke down at these ages and could work no longer. They were cut off, the vast majority of them, at the time when mentally and physically they should have been at their best.

In hospital practice men patients predominate. In insane asylums more women die of tuberculosis. In the community at large it is probable that the sexes are affected in about equal numbers.

All races and all nationalities are affected. It used to be supposed that Hebrews were, to a certain extent, immune. This

can no longer be maintained. In Philadelphia, at the Phipps Institute, and in New York, at the Tuberculosis Infirmary, the percentage of Jewish patients is in excess of their percentage of the total population of the community.

Bad hygienic surroundings, over work, and dissipation seem to be predisposing factors. The disease appears to be more prevalent among persons who will not or can not take proper care of themselves.

In a series of studies made by Mr. Frederick L. Hoffman, statistician of the Prudential Life Insurance Company, he showed that the mortality from tuberculosis was highest in those working at trades where the workers were constantly subjected to the inhalation of metalic and mineral dust.

In 1901 Denison, of Colorado, put forth the theory that breathing devitalized air was an important predisposing factor. That improperly ventilated living and working rooms were responsible for the devitalized air. The vast majority of women patients at the Tuberculosis Infirmary have been domestics.

Certain diseases, as measles and grippe are recognized as predisposing to tuberculosis. Pneumonia is certainly sometimes a predisposing cause. Flint says not; that where tuberculosis seems to follow pneumonia tuberculosis probably preexisted. Kidd, in Allbutt's System of Medicine, says tuberculosis following pneumonia is "infinitely rare." I have found, at least, one per cent. of my hospital cases dating their illness from a pneumonia.

In women the development of the case frequently starts from pregnancy or child-birth.

Traumatism is said sometimes to be the beginning of a tuberculosis. I have met with two cases that seemed indisputable. A woman at the Tuberculosis Infirmary reported that her illness dated from a kick in the chest that was immediately followed by a pulmonary hemorrhage.

In October, 1913, I saw a young girl who had an active tuberculosis in the left lung. A few years before she had been badly scalded and the left side of the chest was one great scar.

Bacteriology: The tubercle bacillus is a short rod-like structure that appears in the discharges of a tuberculous subject. It has been found in the urine and feces, as well as in the sputum.

Boiling kills it. A heat of 140° F. will kill it after twenty minutes. Cold has no effect on it. Direct sunlight kills the bacillus in a few hours, diffused light usually in a few days; in one or two experiments it resisted atmospheric influences for several months. Its virulence lasts longer in a dark, moist atmosphere than elsewhere.

The material to be examined must be spread in a very thin layer on a clean cover glass. It should then be fixed by heating in the flame of a bunsen burner or alcohol lamp. Stain with carbol-fuchsin solution and steam. Wash in water. Decolorize with 30 per cent. solution of nitric acid in water. Wash in water. Wash in 95 per cent. alcohol. Wash in water and mount. The tubercle bacilli are stained red.

Smegma bacilli and leprosy bacilli look and act like tubercle bacilli. With this in mind the microscopic diagnosis should not be difficult. The smegma bacilli are not apt to get in the sputa, and leprosy is very rare in this country. If the urine is investigated bacteriologically then the smegma bacilli must be borne in mind. There are ways to distinguish the two, but the process is tedious. In doubtful cases, where it is very important to recognize tuberculosis, inoculation of guinea pigs will clear up the diagnosis.

Morbid Anatomy and Pathology: The pathological lesion of pulmonary tuberculosis begins as a catarrhal condition of the air cells usually in the apex of the lung. Soon the cells become filled with an exudate. This causes distension and cutting off of the blood supply. Nodules of varying size are formed in the lung by groups of these infiltrated cells. At first these nodules are hard, later they break down by infiltration necrosis, forming cavities.

The process may extend by continuity of tissue through the bronchioles, through the lymphatics or through the blood vessels. Usually more than one part of a lung is affected. One lung is always more diseased than the other, but it is very rare to find one alone diseased.

In some cases the tubercles become encapsulated by an overgrowth of connective tissue and then become calcified—healed.

In most cases where the disease is at all extensive there are

pleural adhesions, and sometimes it is impossible to remove the lung at the necropsy without tearing it. It is heavier than normal.

Besides the usual pleuritic adhesions I have found a true pneumonic condition in a number of cases, post-mortem. Emphysema and bronchitis of the non-tubercular part of the lung I have found to be common.

Pericarditis, with and without effusion, occurs. In quite a percentage of my autopsies I have found incompetent mitral valves, and in a lesser number incompetent aortic valves. I have never found the aortic valves alone diseased, the condition has always been associated with the mitral lesion. The heart is more often small and the muscles soft.

The large parenchymatous kidney occurs quite often, the small interstitial one less often. I have found cystic degeneration once.

Tubercular peritonitis is sometimes found.

Sometimes tubercular lesions are found in other organs.

I found a perforated ulcer of the stomach in one case, and extensive brain abscess in another.

Symptoms: Probably the first symptom of pulmonary tuberculosis is a constantly increased pulse rate. Then comes the slight rise of temperature every afternoon—99° F. or a trifle higher. In women the temperature curve will rise higher during the menstrual flow. There is frequently a slight hacking cough, or a more or less constant desire to clear the throat. There is slight dyspnea on exertion. The patient tires easily and does not feel quite up to the mark. A patient presenting any or all of the above symptoms should be stripped to the waist and given a careful physical examination. Many cases will begin, so far as the patient can tell, with a cold and cough, which hangs on until he becomes alarmed and seeks medical advice. Such cases require the most careful treatment, and even so may run on to final death.

Others, again, have a more or less severe hemorrhage or hemorrhages as their seeming starting point. Such cases are apparently the ones that are the most hopeful. Care and change of climate frequently cure them.

Cases of influenza, or whooping-cough, or pneumonia that seem to be unnecessarily protracted need to be constantly watched, for sometimes they run into tuberculosis.

After the development of symptoms, pulmonary tuberculosis in progressive cases, runs an average course of about two years or longer.

The *pulse.* As I have said, a slightly increased pulse rate is probably the earliest symptom of tuberculosis. In the beginning it may range from 85 to 100. Sometimes when in bed at rest it will become normal, but it is easily accelerated by moving about. I consider the pulse rate such an important symptom that I am never willing to make a diagnosis of tuberculosis when the pulse is normal. I do not believe we can have pulmonary tuberculosis with a slow pulse.

As the disease progresses the pulse rate increases. In advanced cases the pulse is frequently 120 or more, and late in the disease it may be 150 or 160. Towards the end I have seen it as high as 200.

The *temperature.* Next in importance comes the temperature. In the early cases this will be 99° F. or a little more each afternoon or evening. The patient may not feel any discomfort from it, and it may only be discovered by the use of the clinical thermometer. In suspected cases the temperature should be recorded every two to four hours for a week to make sure. In some patients the temperature may be subnormal in the morning. A daily variation of more than one degree is always suspicious. The temperature of the healthy individual varies about that much.

As the disease progresses the temperature will go up a little higher, perhaps to 100° or 101° F. In women, if a continuous record is kept it will be higher about the menstrual period than between times. In advanced cases menstruation ceases.

Late in the disease the temperature gets higher still with greater variations. It may be 103° or 104° F. at night, dropping to 100° or lower in the morning. We may have a true septic temperature, due to mixed infection, the addition of the streptococcus and the staphylococcus to the tubercle bacillus.

The *cough.* At first the cough may consist merely of a dry

hack. Or it may not be a cough so much as a clearing of the throat. Later on the cough becomes more troublesome. At first it is apt to be limited to the morning on first getting up. Then on going to bed as well. Finally more or less throughout the day and frequently at night.

The *expectoration.* The expectoration varies as much as the cough. It may be very scant, then more on first getting up, then very considerable every time the patient coughs. It finally becomes profuse and purulent.

Respiration. The respirations are somewhat increased early, especially on exertion. There may be slight dyspnea on going upstairs or on making any extra effort. The patient tires easily.

Hemorrhage. Hemorrhage from the lungs is quite common in tuberculosis. It had occurred in 44 per cent. of ten thousand cases tabulated at the Tuberculosis Infirmary.

Hemorrhages vary in frequency and quantity. There is the hemorrhage that appears early in the disease. It may be slight, merely discoloring the sputum, or it may be quite profuse. There may be one or two hemorrhages or there may be many. The blood is usually coughed up, and if there is much it is bright red and frothy.

I have seen a very few cases where the hemorrhage was more or less continuous for several weeks, finally killing the patient. In the great majority of cases, nearly all, in fact, hemorrhage very early in the disease is not of evil omen. Hemorrhages that first occur after the patient has been ill for a year or two usually mean that the disease is progressing.

There is another type of hemorrhage that is always serious. That is the hemorrhage that appears first very late in the disease. Not infrequently the bleeding is sudden, profuse, and fatal. I have seen a number of such cases. The patients were literally drowned in their own blood in a very few minutes.

Night Sweats: Many patients will state that they have had chills and fever for some time, still others will add night sweats. Night sweats were reported by 60 per cent. of ten thousand patients at the Tuberculosis Infirmary. Night sweats are not pathognomonic of tuberculosis, they sometimes occur in other conditions, notably in sepsis.

There is wasting late in the disease some patients becoming extremely emaciated. The skin becomes dry in advanced cases.

The mental condition varies. Tuberculosis has received such a tremendous amount of advertising of recent years that most patients are depressed when first told they have tuberculosis. Towards the end many are hopeful.

I conducted some psychological experiments in my wards a few years ago for the psychological department of Columbia University. I found no evidence of mental brilliancy due to the tubercle bacillus.

Physical Examination: When first examined the physical signs of tuberculosis may not be very marked, even in patients apparently quite sick. Other patients with very indefinite general symptoms may be found to have active lesions in the lungs. At any rate, the physician should not make the mistake of overlooking the true cause of the trouble simply by neglecting to make an examination of the chest.

For such an examination the patient must be stripped to the waist. The light in the room must be good.

Although pulmonary tuberculosis may begin in any part of the lung, in the vast majority of cases the initial lesion will appear in one or the other apex. I have seen but one case, postmortem, where the disease was confined to a lower lobe. As the case progresses the lower lobes frequently become involved.

Inspection will show the size and shape of the chest. In early cases there will be no emaciation. Later in the disease there will be. Frequently there will be a sinking in of the supraclavicular spaces, and in advanced cases the shoulder blades are prominent. The shape of the chest conveys no information. I have examined, on the average of two thousand patients a year, at the Tuberculosis Infirmary, for ten years, and there is no typical phthisical chest.

In tuberculosis there is always increased frequency of respiration—to a marked degree, forty or more, in advanced cases. Inspection will usually show diminished expansion. A normal chest can be expanded three to three and a half inches. Early in tuberculosis this will be lessened; later it will be much lessened. A normal chest expands equally on each side. In

early cases one side may expand and contract less than the other, or one side may lag behind the other. This is particularly true where, as so frequently happens, the pleura is involved. Later in the disease this asymetry is usually more marked.

Palpation will give the same information as regards frequency and regularity of respiration as does inspection. Sometimes a slight difference in the extent or regularity of expansion between the two sides can be felt when it cannot be seen.

Vocal fremitus is elicited by palpation. It is more marked over consolidated areas of diseased lung. Normally the right side gives a more marked fremitus than the left. If the two sides are equal it means that there is consolidation on the left side.

In tuberculosis the skin frequently has a different feel from the normal. It is drier. In advanced cases it may feel rough and harsh. It may or may not feel feverish.

Percussion. Percussion will enable the examiner to locate areas of consolidation. The percussion note is duller over such places than over normal lung tissue. The corresponding areas on the two sides should be compared, for it is often only by comparison that one can tell with certainty in doubtful cases what one hears. In advanced cases the margin of error is not so great.

One of the most valuable signs of early pulmonary tuberculosis elicited by percussion is the narrowing of the apex in the root of the neck,—Krönig's sign. The percussion must be performed lightly, above and parallel to the clavicle. Under normal circumstances the resonant area will be two to three inches or more wide, and the sides will be nearly equal. A difference between the two sides of half an inch or more usually means trouble on the narrower side. A resonant area of one and a half inches or less in width is indicative of disease. Of course, the actual width at which resonance may be found, in health, depends somewhat on the size of the individual. Deformity of either side makes this test useless.

Auscultation: Auscultation will reveal the presence or absence of rales. A rale is an adventitious sound and may be roughly classified as fine or coarse. More detailed subdivisions

are only confusing. In badly diseased lungs coarse rales may be heard over the affected areas. In early cases the rales are not so coarse or so easily heard, and sometimes can only be elicited by having the patient cough first as suggested by Garvin. At the beginning of the changes in the lung rales are most often heard just below the clavicle and near the sternum. The next most frequent place is above the scapula and along its spinal border. The third most frequent place is in the axillary region. Final judgment should not be pronounced until these three areas have been most carefully gone over.

Another important early sign, where there is no change in the lung except a slight catarrhal condition of the apex, is the transmission through the stethoscope of whispered sounds. This can be heard above the scapulæ at the back. Normally the breathing sounds on the right side are heard loudest. Whispering sounds are not plainly transmitted in normal lungs. In slight consolidation whispering sounds are plainly transmitted in the supra-scapular region.

In hemorrhagic cases there is often a very fine crackling sound over a limited area. I have frequently found this in the front of the chest below the clavicle and near the sternum. I have frequently diagnosed hemorrhage from this peculiar sound, when I had neglected to ask about hemorrhage first; and I have occasionally prognosticated hemorrhage from that sign, and proven correct, when hemorrhage had not previously occurred. It is of ill omen to me and I do not like to hear it. After a considerable hemorrhage fine bubling sounds can often be heard over the affected spot. Occasionally it is impossible to locate the site of the hemorrhage.

The *heart* must be ausculated. In tuberculosis there is an accentuated second sound over the pulmonary area. Frequently a blowing sound can be heard with the first sound of the heart at the same place. I am in doubt as to an explanation for this.

As the disease progresses all of the above physical signs become more marked and can be elicited over larger areas. If the disease becomes arrested or tends towards a cure the signs gradually disappear. Occasionally the signs of disease will disappear altogether. More often the rales will disappear, but it will be possible to map out small areas of consolidation.

Complications and Sequelæ: Any abnormal condition may be found in association with pulmonary tuberculosis.

Quite a number of cases are complicated by tuberculosis of the larynx. I always look on this as an extremely grave accident.

Pleurisy is always associated with advanced cases of pulmonary tuberculosis. Sometimes there is considerable effusion into the pleural cavity.

Bronchitis is frequent. Asthma and emphysema are sometimes found, contrary to the belief formerly held.

An intercurrent or a terminal pneumonia is not rare.

In advanced cases cavities are formed in the lungs by the breaking down of the tubercles. Unless the cavities are large however, I have always felt diffident about locating them. I have been too often disappointed in failing to confirm my diagnosis in the post-mortem room.

The heart, as a rule, is small. Exceptions occur. I have found mitral and aortic lesions in a considerable percentage of cases, and have confirmed the diagnosis by autopsy.

I have seen many skin lesions. I had one case of multiple fibroma, and one of vitiligo.

I have also met with cancer of various organs.

Chronic parenchymatous nephritis sometimes occurs, less often interstitial nephritis.

Multiple neuritis has occurred in from one to two per cent. of the cases among the women in my service. There must be some relation between that and tuberculosis, because I have seen more cases of neuritis in the tuberculosis wards than I used to see in the general medical wards at the Metropolitan.

Diagnosis: The diagnosis of a typical case of pulmonary tuberculosis is not difficult. Nevertheless, many mistakes are made, woeful mistakes, that permit of the patient becoming far advanced in the disease before it is discovered.

The indefinite early symptoms are perhaps most often mistaken for malaria. The physician should never be satisfied to treat a doubtful case without making a complete physical examination of his patient. Malaria and tuberculosis may coexist.

Strange as it may seem, typhoid fever and tuberculosis may be mistaken for each other. I saw such a case in consultation. The patient had been in bed and had been treated for typhoid fever about a month. Physical examination showed an acute pulmonary tuberculosis in an active stage.

Syphilis of the lungs, said by all authorities to be very rare, is clinically a counterpart of tuberculosis and is indistinguishable from it. Of course, the tubercle bacillus is not found in the sputa, but that is sometimes difficult to find even in true cases of tuberculosis. Syphilis may coexist with tuberculosis. I have found it in one per cent. of my hospital cases. The Wassermann test may be applied when in doubt.

In pneumonia of the upper lobes of the lungs the physical signs are the same as those of tuberculosis at certain stages of the disease, but the history of the case is different. The microscope will prove of assistance in differentiating between the pneumococcus and the tubercle bacillus.

Of recent years *tuberculin* has been used for diagnostic purposes in doubtful cases. Subcutaneous injection of *tuberculin* will produce a marked reaction in tuberculosis subjects. It is hardly a safe procedure, however, as it may light up a latent lesion. In doses of .015 c.c. it causes fever and local reaction about the seat of the disease.

Calmette introduced the ophthalmo-reaction. That consists in the instillation of a drop of a preparation of Koch's old *tuberculin* into the eye. If the patient has tuberculosis, the blood-vessels of the conjunctiva become injected, there is swelling of the membrane and increased lachrymation. In exceptional instances this has caused permanent damage to the eye.

The most satisfactory of the *tuberculin* tests is the so-called von Pirquet skin reaction. This consists in cleansing the skin at the site of the test, scarifying the spot and rubbing in a drop or two of *tuberculin* in the same way that one uses vaccine virus. If the reaction is positive there will be an inflamed area of skin about the site of the scarification in about twenty-four hours. This will last for a couple of days. If there is no tuberculosis there will be no reaction.

Neither the ophthalmo-reaction nor the skin reaction will lo-

cate the lesion, they simply tell that there is one, or there has been one. These reactions will be positive even in healed lesions. In the desperately ill in the last stages the reaction does not appear.

The x-ray picture can be used as an additional method of diagnosis. It will help to localize the lesion perhaps. But I believe it to be only a help. Careful physical examination will tell as much, sometimes more.

Prognosis: One-third of the bodies that come to autopsy from other causes show healed tubercular foci in the lungs. It has even been said that every one more than fifteen years of age will show such healed lesions. Therefore the prognosis in general is good. In the vast majority of such cases, however, tuberculosis has not been suspected during life.

In cases that are detected early, cases that are clearly tuberculosis, proper and long continued treatment will frequently produce a cure. The prognosis is therefore fairly good, although a few cases will not respond to the best of care.

Cases that get a good start are not so favorable. At the Tuberculosis Infirmary the mortality is thirty per cent. More than half of those that die die within thirty days after entering.

Death is most often due to a general infection, as instanced by the high temperature, rapid pulse, and so on. A few patients die of profuse hemorrhage, possibly the only one that has occurred.

Prophylaxis: If all the tubercle bacilli in the world could be obliterated tuberculosis would cease. But that is impossible. It is therefore necessary to be content with what can be done. The bacilli have been found in the sputa, in the urine, and in the feces. All of those discharges must be sterilized and destroyed in order to prevent as far as possible infection from any given case. In coughing a fine spray is given off which contains the bacilli. Patients should therefore be taught to hold something before the mouth when coughing to receive this spray. A piece of cheese cloth or paper napkin, something that can be destroyed, is the best thing to use. They can be put in paper bags temporarily. Very sick and weak patients can expectorate on these. Other patients can use a

sputum cup. There are many kinds on the market. Paper ones are the best, as they can be destroyed. Those of metal, glass or crockery must be thoroughly sterilized twice a day. They should contain a small quantity of carbolic acid solution, five per cent., or some other antiseptic.

The urine and feces must also be promptly disposed of. If the patient is in bed a bed pan containing an antiseptic solution must be used.

The bed clothing, dishes and eating utensils require thorough cleansing.

As a patient must be in a susceptible condition to develop tuberculosis it is necessary to keep the body of those exposed at the highest state of efficiency. That means every one, for everyone is, of necessity, exposed.

Bad living and working conditions must be avoided. Overwork, worry and dissipation must be avoided where possible.

The most important time to prevent the disease is during childhood, as it is coming to be the accepted idea that most patients receive the infection early in life. This requires proper hygienic care from the moment of birth. If the child is fed artificially, pure milk, as free from germ life as possible, must be used. If the milk is of doubtful origin it is best sterilized.

Later the nose and throat must be put in good condition if diseased in any way.

Treatment: The great tripod in the treatment of tuberculosis is rest, fresh air and sufficient food.

Rest is, perhaps, the most important single factor, rest physical and mental. A patient with a temperature of 100° F. or more requires rest in bed, rest where the air is pure and fresh. If the patient is where he can be out of doors, complete physical rest in a steamer chair may do. The point to be emphasized is that a temperature of 100° F. means absolute rest.

So far as possible the patient's mind must be at ease. He should have no cares or worries. Travel, for those able to do it, early in the disease, may be of great help. It keeps the patient interested.

Fresh air is the second desideratum. Fresh air does not necessarily mean cold air. The patient should spend as much

time as possible in the open. In sanatoria this is part of the treatment. A patient can do much for himself and still live at home. The windows can be kept open at night when the patient is asleep. In the day time he can use a porch, or, as some of my dispensary patients do, can spend his time in the parks or along the water front—if there happens to be one.

The patient must be warmly clad in cold weather, and the head must be protected at night if he sleep in a cold place. The clothing must only be warm enough for comfort, however, and not heavy enough to induce profuse perspiration.

Climate: In this connection I wish to say something about climate. Tuberculosis may appear in any climate, it may be cured in any climate. But certain places seem to be particularly beneficial to the consumptive. A clear dry climate with a maximum amount of sunshine is best; a certain amount of altitude may be advantageous. The ideal place in this country is through Western Texas, New Mexico and Arizona. This section is dry, comparatively, there is a maximum of sunshine and a minimum of rain, and the altitude ranges from three to seven thousand feet. Such a climate allows the patient to be out of doors most of the time. The air is aseptic and healing. The altitude is sufficient to cause deeper inhalations and consequent opening up of the diseased lung tissue.

Although climate is not a specific of itself, and all patients sent to certain regions will not get well, yet I believe a change of climate is a most important and valuable adjunct in the treatment of tuberculosis. Beside the change of climate, the patient is taken from the environment where his disease has developed. His whole routine of living is changed in a way that even with the best of intentions could not be done at home.

It has for years been recommended that patients going to a place with a considerable altitude should not make the change too abruptly. I wish to emphasize the fact that when a patient, after a sojourn of several months at an altitude, decides to go home, he should not make the change to the home climate too abruptly. A year's experience in the west in search of health for myself, a year in which I had the opportunity to study the effect of climate on many patients besides myself, led me to this belief.

Since 1897, whenever I have sent a patient away, if the patient could afford it, I have mapped out a more or less extended trip covering several months. Every patient who has followed that scheme has benefited by it. My favorite route is through Western Texas, New Mexico, and old Mexico, as far as the City of Mexico. Patients may go from New York to Galveston by steamer, or the trip may be made all by rail. An ocean voyage of itself is beneficial in early cases, for nowhere is the air so pure and free from dust. A trip of this sort, stopping a few days or weeks in different places, affords a variety and interest that could not be gotten in any other way. It prevents ennui, which, from personal experience, is bad for the lung invalid. It also prevents too constant association with other health seekers which should also be avoided.

Diet: The lung invalid needs a good, wholesome, nourishing diet, and plenty of it. Eggs and milk should form the basis of it. Good red meat and vegetables, at least once a day, for those with a good digestion, is also essential. Overfeeding or indigestible foods should be avoided. In many cases the digestion is impaired and improper feeding will aggravate that. The digestion must be conserved at all hazards, for when the digestion fails the case is hopeless.

Drugs: *Tuberculin.* Of recent years *tuberculin* in some form or other has come to be used as a remedy. Many different preparations are used, and each has its advocates. In the early days of the *tuberculin* treatment considerable doses were used. Now many authorities begin with very small doses, one-millionth to one-billionth of a milligram or less—the homœopathic 6x to the 9x potency. The dose is not repeated as long as any effect is noted, from once a week to once a month. This is the accepted old school method of to-day. Could anything approach more nearly the true homœopathic method? *Tuberculin* is given hypodermatically by the majority of physicians. It may be used by mouth.

If there is marked reaction, that is, increase in temperature, increase in malaise, loss of weight, appearance of hoarseness, or signs of blood, the dose must not be repeated until reaction ceases. The dose should never be increased so long as the last

one produces any reaction, it may be decreased with advantage. Reports show that some cases recover under this treatment. Other cases will improve for a time and then start on the downward path and go very rapidly. Far advanced cases do not stand the *tuberculin* treatment well, it seems to hasten the end.

Bacillinum: I have used this in the 30x and 200th potencies, a dose twice a month. In early cases it has been very satisfactory, in late cases it has not. *Bacillinum* must be given at intervals over a long period of time. It undoubtedly helps the cough and general condition.

Of the ordinary homœopathic drugs *bryonia* seems to be the most useful. It is of particular value when there are pleuritic pains. It is also good for the cough, worse on going into a warm room.

Hepar is useful in certain cases with a loose cough aggravated by cold, dry winds, the slightest draft annoys.

Ipecac is indicated in cases with nausea and vomiting. The rales heard are very fine.

Nux vomica is useful in thin, spare people. The patient is worse in the morning. He is very irritable.

Kali bichromicum is useful in an accompanying bronchitis with much cough and thick, tenacious expectoration.

Arsenicum iodide, Boericke says, most closely resembles tuberculosis. It is indicated by a profound prostration, rapid, irritable pulse, recurring fever, sweats, and emaciation.

Arsenicum album is also of value in cases with great prostration.

Iodium is indicated for cases with hoarseness and emaciation.

Phosphorus is indicated when aphonia takes place. Talking and laughing make the patient cough.

Pulsatilla is a most excellent remedy in early cases. It is indicated with much thick expectoration. It is especially valuable in women.

Calcarea carbonica may be used, especially in children who are marasmic and have head sweats.

Kali carbonicum has stitching pains in the chest. There is aggravation of the cough at 3 a. m.

Silica is useful in cases with profuse night sweats, and profuse pus-like expectoration.

Lycopodium is indicated in cases that seem to begin with a pneumonia. Expectoration is salty. There is hectic fever.

In case of hemorrhage, *aconite* is useful if the patient is frightened, fears death, and has a rapid pulse due to this fear.

Ferrum phosphoricum is useful in hemorrhage without the fear. There is considerable fever. It is the remedy I use most often.

Many other remedies may be indicated, but these few will cover the majority of cases.

GONORRHEA.

(Greek, γονη, semen; ρεω, to flow.)

Definition: An infectious disease characterized by a discharge from the urethra or vagina and caused by the gonococcus. The gonococcus may invade other parts of the body, notably the pelvic organs in women, and the endocardium in both sexes. It may attack the eye. It may affect the rectum, mouth or other mucous membrane.

Historical Note: Gonorrhea and syphilis were formerly considered to be different manifestations of one disease. It is only since about 1850 that the two have been differentiated. In 1879 Neisser found the gonococcus. Since then gonorrhea has been recognized as a distinct disease entity. Furthermore, investigation has revealed the fact that the gonococcus may travel to various parts of the body causing serious lesions, even death. It therefore becomes essential to consider gonorrhea as a constitutional possibility, rather than as a purely local condition.

Etiology: The essential cause of gonorrhea is the gonococcus. By far the most frequent source of contamination is sexual intercourse. The disease may be transmitted by means of the vaginal syringe or the use of soiled towels, etc. The bladder, prostate, kidneys, uterus or ovaries may become infected by extension from the genital tract. The endocardium and joints may become infected, probably through the blood current. The eyes of the new born may be infected in passing through the

genital tract at birth. Or the eye of the adult may be infected by carelessness. This is serious in either case. Any age and both sexes are susceptible. In the nature of things young adults are most often affected.

During the past few years many cases of gonorrheal infection have been found in female infants and little girls, especially in institutions. The source of contagion is not always clear, but is probably through infected toilets or unclean towels or clothing. The cases are usually mild. Some five or six cases were discovered in the children's ward at the Flower Hospital in August, 1914, during my service.

Bacteriology: The gonococcus consists of two hemispheres with a line between. It is decolorized by Gram's method. Cultures must be made with some albuminous fluid; hydrocele fluid or pleuritic fluid may be used.

Morbid Anatomy and Pathology: Inflammation of the parts affected have no special pathological feature other than the presence of the gonococcus.

Symptoms: Gonorrheal urethritis usually appears in three to ten days after exposure.

In men a urethral discharge appears which at the beginning is colorless, but in a few hours becomes thick and creamy in consistency, and may be white or yellowish or slightly greenish in color. The meatus is inflamed, in fact, the whole glans may be red and swollen. The books distinguish between an anterior and a posterior urethritis. In posterior urethritis the prostate is usually swollen and inflamed.

Frequent painful urination is one of the symptoms of gonorrhea. Painful erections and chordee also occur.

After a week or ten days the acute symptoms subside and the disease becomes subacute. This lasts one to two months and may run into gleet—chronic gonorrhea.

In women the urethra is not always the seat of the disease. The vagina, sometimes the cervix, is the part affected. The glands of Bartholin are nearly always involved. The discharge is apt to cause more or less irritation, redness and swelling of the labia.

Complications and Sequelæ: The complications are of most con-

cern to the general practitioner. *First,* there are the complications arising from extension of the disease to other parts.

Stricture of the urethra is common in men.

In men also the various genito-urinary glands may become involved by continuity. Or the disease may cause an epididymitis and orchitis. All of these conditions are accompanied with heat, swelling, tenderness, and pain.

Occasionally cystitis occurs, more rarely the ureters and kidneys become involved. I have known this last accident to cause death in a few weeks.

In women the inflammation may extend to the uterus and Fallopian tubes. Even to the ovaries and peritoneum. Gonorrhea then becomes a distinctly surgical disease.

Second, complications by infection.

Gonorrhea may affect the eye. In the new born, infected during birth, gonorrheal ophthalmia may cause blindness.

If the eye of the adult becomes infected, only the most prompt and vigorous treatment will save the eye from destruction.

The gonococcus may be transferred from one part to another by accident.

Third, systemic infection.

One of the most frequent complications of gonorrhea is gonorrheal rheumatism. This may affect one or several joints. It usually comes on during or just after an acute attack of gonorrhea and is extremely persistent and hard to control. It is most often monarticular, more frequently, perhaps, a knee or an ankle. There is swelling and effusion in the joint. It is painful and tender.

Spondylitis deformans may also result from gonorrhea. This is a serious condition.

Tender heels is another gonorrheal complication or sequel.

A general septic condition may occur with an infective endocarditis. This is a very grave condition, as it almost invariably ends in death. The endocarditis may come on early, Osler reports a death within two weeks after the urethral discharge appeared. More often it comes on considerably later. I saw one case in my wards at the Metropolitan Hospital where gonococci were demonstrated in the endocardium at the autopsy.

Diagnosis: In men a urethral discharge is usually due to the gonococcus. At any rate the microscope will clear up the diagnosis.

In women a vaginal discharge may result from various causes, so that the use of the microscope is imperative to make the diagnosis certain.

A suspicious ophthalmia also requires the utmost care in diagnosis, as the gonococcus is particularly malignant in the eye.

Prognosis: A simple gonorrhea may be cured, although at best a cure is very uncertain. Many patients have recurrent attacks which are probably only recrudescences of the original trouble.

Stricture, with resulting damming back of the urine, and finally cystitis may occur.

Direct extension to the bladder and kidneys, or to the pelvic organs in women, is always serious and may prove fatal.

Gonorrheal rheumatism is a tedious condition.

Infective endocarditis from the gonococcus is fatal.

Gonorrheal ophthalmia may result in loss of the eye.

The prognosis of gonorrhea is therefore always doubtful, and when certain of its complications ensue is very grave.

Treatment: The best preventive is avoidance of illicit intercourse. A patient with gonorrhea should avoid all sexual excitement or intercourse so long as a trace of discharge remains.

Recently, 1912, Dr. W. J. Robinson, of New York, has opposed this idea. He thinks the ideal way to empty the seminal vesicles is by intercourse and believes that to be beneficial after the acute stage has subsided.

The utmost care should be used to prevent infection of the eyes.

Absolute cleanliness is the first essential. The penis should be soaked in hot water that has been boiled, several times a day. Women should take sitz baths. During the acute stage injections in either sex are not advisable.

After the acute symptoms have subsided injections of 1 per cent. protargol, or boric acid saturated solution may be used in the male urethra. Or argyrol, 10 per cent., every 2 to 3 hours

for ten days or two weeks may be used. In women douches of the same may be used—but the uterus must not be invaded. I have known douches to carry the infection into the uterus, whence the infection spread to the tubes and septic peritonitis was the result. A radical operation, removing uterus, tubes and ovaries, was required to save the patient's life.

Eye cases require skilled treatment by skilled oculists. Boracic acid, protargol or argyrol instillations must be used as required. Ice cold cloths must be applied every two or three minutes, night and day, until the inflammation subsides. The cloths must be destroyed and not used a second time.

One case developed gonorrheal ophthalmia at age of three days. It was cured in five days with argyrol, 10 to 25 per cent. in water every 3 to 4 hours.

Gleet—chronic gonorrhea—may require deep injections. Argyrol 10 to 20 per cent. in water daily may be used.

Patients with acute gonorrhea must avoid all sexual excitement. They must not be too active—rest in bed is of great benefit during the acute stage. Men should wear suspensory bandages when up and about.

If orchitis occurs the patient will of necessity be in bed, as this is excessively painful and moving about is impossible. The testicles must be supported even in bed, and the weight of the bed clothing must be kept off them.

Gonorrheal cystitis requires irrigation of the bladder. A gonorrheal kidney may need surgical treatment. Surgical interference is also needed in gonorrheal pelvic inflammation in women.

Gonorrheal rheumatism requires rest in bed. Osler reports good results from the hot air treatment. The use of the Esmarch bandage sometimes gives relief. In synovitis removal of the fluid by puncture may be necessary. Fuller believes seminal vesiculotomy is a sure cure. (*Medical Record*, June 22, 1912.)

In general sepsis with infective endocarditis the case must be treated symptomatically.

Aconite is indicated in the beginning of a gonorrhea.

Gelsemium is also useful in the acute stage.

Pulsatilla is indicated when the discharge is thick and creamy. Later on *pulsatilla* may cause an old gleet to light up and start anew a thick discharge. It will cure if continued. *Pulsatilla* may also be of service in orchitis.

Pulsatilla cured the following in a woman. Pudenda swollen —pain and intense itching—thick excoriating white, creamy discharge—ulceration—cured in a month.

Carbo vegetabilis cured gonorrhea in a woman:—Pain in right ovary, abdomen swollen and bloated, pudenda swollen—thick, excoriating, white, creamy discharge, prompt relief—less pain and swelling.

Phytolacca is a splendid remedy for orchitis.

Bryonia—case of gonorrheal rheumatism in left knee—slightest motion painful, swollen and red, cannot bear bed clothes. Cured.

Many other remedies may be indicated.

SYPHILIS.

Synonyms: Lues. Pox.

Definition: Syphilis is a contagious disease that is transmitted by contact or by inheritance. It is a constitutional disease, with well marked local manifestations that appear at different stages of the disease. It runs a chronic course, divided into three stages—primary, secondary, and tertiary.

Historical Note: Syphilis is probably one of the most ancient of diseases. Its origin is lost in antiquity. During the middle ages, following the Crusades, it had an enormous impetus throughout Europe. At that time it was customary to give it the name of some country other than the one the victim lived in, as the Spanish disease, the Naples disease, etc.

Syphilis, as a distinct disease entity, separated from other venereal diseases, has been so recognized only since about 1850.

Etiology: Syphilis is transmitted by contact of an abraded surface with the discharges from syphilitic lesions. No age and neither sex is exempt. The majority of cases are transmitted by sexual congress, and the primary sore appears about the

genitals. But extra-genital infection is not uncommon. The mucous patches that appear in the mouth are infectious, and the disease may be transmitted by kissing, by using eating or drinking utensils that have been used by a syphilitic, or by contact with anything that has been contaminated by a syphilitic. Sometimes physicians become inoculated through the fingers in examining syphilitic patients.

Syphilis may also be inherited from either parent, occasionally it may pass to a grandchild.

Bacteriology: For more than two centuries and a half various organisms have been described by various writers as being the cause of syphilis. It was not until 1905, however, that an organism was discovered that has come to be generally accepted as the true specific cause. In that year Schaudinn and Hoffman, of Berlin, gave a demonstration of the *spirochete pallida.*

This organism is a long, delicate spiral, varying in length from 4 to 14 μ pointed at both ends. The spirals are tighter at the center than at the ends, and the number of twists varies from two or three to twenty or more. Very delicate flagellæ are sometimes found at the ends. It is motile on its own axis and lengthwise. The organisms may be found separately or in groups.

The spirochete pallida is found in the primary sore, in the secondary lesions, and in the blood and tissues. It may be seen in a hanging drop of fluid from the part examined. If a stained specimen is desired, Osler and Churchman (Osler, Modern Medicine) state that most stains have no effect on it and none stain very deeply. They recommend, (1) fixation, (2) place in azar-eosin for twenty-four hours, (3) then wash with water and examine.

It must be differentiated from the spirochete refringens by its greater delicacy, its very slight refractibility, its great resistance to stains, and by its tight cork-screw like spirals. The spirochete refringens is the opposite of all these, though found most frequently in the same places.

Although to a certain extent still sub judice it would seem as though the fact that the spirochete pallida is found in syphilis at all stages, and never elsewhere, that its place was established.

Morbid Anatomy and Pathology: *Acquired Syphilis.* I. *First Stage.* The primary sore, the Hunterian chancre, is a small sharply defined ulcer with indurated edges. The opening has a punched out appearance. The discharge is slight and non-purulent. After a few days the surrounding lymphatics may become swollen.

II. *Second Stage.* Most of the pathological changes in this stage occur in the skin and mucous membranes. The eruption at this stage may be a simple roseola, or it may be papular, scaly, or vesicular in character. They tend to recover.

III. *Third Stage.* Here the pathological changes take place in the deeper structures, as well as in the skin. The tertiary skin lesions are apt to be pustular, tend to ulcerate, and are serpiginous.

In the respiratory and digestive tracts syphilis may cause ulceration.

In the internal organs gummata are common, also amyloid degeneration.

Arterio sclerosis is a frequent tertiary manifestation. Associated with this, or as a result, are the changes that take place in the kidneys—chronic interstitial nephritis—and in the central nervous system—cerebral softening and hemorrhage.

Aneurysm, tabes, and general paralysis are end results.

In *congenital* syphilis the liver is enlarged. Various ulcerations may take place.

Symptoms: *First or Primary Stage.* After exposure the incubation stage is ten to fifty days or more. At the end of that time there develops an itching and an irritation at the infected point, with a slightly raised papule or pimple. It may be quite small or it may be a half inch or more in diameter. It sometimes gives no discomfort. Occasionally it passes away, but usually it breaks down in the center after a few days, and gives off a slight watery discharge. The sides become hard (Hunterian chancre) and indurated. The opening is clean cut.

Usually, a few days after the appearance of the primary sore, the lymphatic glands adjacent become swollen, forming lumps that can be distinctly felt. They never resume their original size.

The initial sore most often appears on a mucous membrane, usually the genitals, for obvious reasons. It may appear on the lips. Or it may appear on the fingers or other skin surface through an abrasion. Statistics show that 90 per cent. of chancres are genital, 10 per cent. extragenital.

This stage is very contagious.

Secondary Stage. Also known as the constitutional exanthemic or the condylomatous stage.

About two months after the appearance of the primary sore, the secondary symptoms begin to appear. They indicate the involvement of the general system. The disease has ceased to be purely local and has become constitutional.

There is frequently malaise, slight rise of temperature, sore throat, and more or less of an eruption. The hair may fall out. There is apt to be headache, sometimes mental depression. Anemia is common. Albuminuria exists and sometimes acute nephritis.

The most constant symptom of this stage is the eruption. This is most often roseola like; it may be papular, scaly, vesicular, or bullous. It is symmetrical and tends to appear on the fronts of the extremities rather than on the backs. It may be slight in character and extent, appearing only on the sides of the body. It may be very profuse. The characteristic color seen most often is a raw ham color.

The eruption may appear on the mucous membranes. When this occurs the spots may end in small clean cut ulcers. The so-called "mucous patches" are reddish, slightly raised, frequently eroded spots, found most often on the tonsils and soft palate. Close scrutiny shows them to be exuding a thin watery fluid. They may be anywhere in the buccal cavity. The sore throat of this stage is a sharply defined erythema.

If mucous patches occur about the genitals and anus there is usually an hyperplasia of tissue forming what are known as condylomata. The discharge from mucous patches is very contagious.

There is a general swelling of the lymphatics throughout the body in this stage. The glands are not much swollen and are painless. They do not tend to ulcerate. The epitrochlear and

mastoid glands, swollen in syphilis, are more or less diagnostic as they are rarely swollen in other conditions.

Anemia in this stage may be marked. The red cells may drop to 2,500,000 or even less.

Acute arteritis may occur with subsequent rupture or thrombosis.

The fever may simulate that of other diseases. It has been mistaken for rheumatic fever when there were severe joint pains, for malaria, for typhoid and for tuberculosis. In doubtful cases nodes should be looked for.

Iritis is another frequent symptom that may appear during this stage.

Periostitis of the long bones, with or without joint pains occurs. Sometimes periostitis affects the bones of the head causing persistent headaches.

Tertiary Stage: The stage of gumma. After eighteen months to two years, in the majority of cases, the secondary symptoms subside and the case appears to be quiescent. Then, after another interval of two or three, sometimes fifteen or twenty years, occasionally after thirty or more years, tertiary symptoms develop. The changes that take place now are characterized by asymmetrical distribution, and by a tendency to get worse. If improved by treatment, they tend to relapse when treatment is stopped.

Syphilitic lesions of the skin and mucous membranes in the tertiary stage usually proceed to ulceration. The lesions take on a crescentic shape, they are serpiginous and unilateral.

Rapid ulceration of the pharynx and uvula sometimes occurs. Ulceration may likewise occur at any point throughout the digestive tract. Hemorrhage may result from the ulceration.

Gummata may appear in the submucous and subcutaneous tissues and in the internal organs, or nodes may appear. These show a tendency to break down. They are found in all the organs and tissues of the body. The symptoms vary, of course, with the part affected.

Amyloid changes may take place in the spleen, liver and kidneys. Acute nephritis may occur.

The most important changes in this stage of syphilis take place in the blood vessels. All the coats of the arteries are subject to inflammation. This frequently leads to rupture causing hemorrhage. Cerebral syphilis may be caused by gumma in the brain tissue itself. More frequently diseased cerebral arteries break down and death is not uncommon from apoplexy. I have seen death from this cause fifteen months after the initial sore. I have seen cerebral hemorrhage due to syphilis twenty years after the initial sore.

Syphilitic inflammation of the aorta is the most frequent cause of aortic aneurysm.

It will be seen from what has already been said regarding the multiform lesions in tertiary syphilis that the symptoms of the disease are legion. It would, perhaps, be better to say that given a sick patient, syphilis should always be considered as a possibility if the case is at all obscure no matter what symptoms the patient may present.

Fourth Stage. Some writers add, as a fourth stage, certain diseases that come on later in life and that nearly always occur in patients who have previously had syphilis. Fournier calls them parasyphilitic diseases. Locomotor ataxia, or tabes dorsalis, is one of these. A very large percentage of these cases are preceded by syphilis—some few authorities claim 100 per cent. In 80 or 90 per cent. syphilitic history can be obtained. A few cases in very young subjects occur as the result of hereditary syphilis.

General paresis is another disease that in the vast majority of instances can be traced to a previous syphilis.

There is a form of neurasthenia and of epilepsy also mentioned by writers.

Also a buccal leukoplasia leading to cancer.

Since the discovery of the spirochete pallida, and since the development of the Wassermann reaction, it has been found that many of these cases of the so-called fourth stage or parasyphilis are really still syphilis. In some the spirochete are found, in the majority there is reaction to the Wassermann test.

Congenital Syphilis: Syphilis is one of the few diseases actually inherited. A child may be born with it. If the child inherits it from the father alone, it may present all the evidence of syphilis, and yet the mother have no signs of it even if she nurses the child. This is known as Colles law. Of late it has been found that such mothers react to the Wassermann test.

If a woman has syphilis she is very apt to miscarry. If she gives birth to a viable child, the infant may, at first, appear to be healthy. If the child has congenital syphilis, in a few days or weeks it may develop snuffles. Then it gradually wastes away. The face assumes a wrinkled, old man appearance. It has a peculiar disagreeable odor. Moist eruptions sometimes appear. Condylomata are common. Later periostitis and ulceration occur. I saw an infant in the clinic at Bellevue Hospital have some of the phalanges of the fingers slough off from congenital syphilis.

If the child survives it is weak, and is very susceptible to other infectious diseases.

In other cases the child does not show evidence of syphilis until later, the eighth or tenth year perhaps. Cases of hereditary syphilis may crop out in the second generation.

Deformities are common. One of the important signs of hereditary syphilis is Hutchinson's teeth. This consists of a deformity of the upper central incisors of the second dentition. They are peg shaped with a curved notch on the cutting edge.

Enlarged liver and spleen are quite characteristic. There is frequently retarded growth—mental and physical.

Interstitial keratitis is common. Deafness is common.

Complications and Sequelæ: Some of the complications and sequelæ of syphilis have already been mentioned. It has been said, "Once a syphilitic, always a syphilitic." That is a good axiom to bear in mind. It may help to straighten out obscure cases.

In the secondary stage the most dangerous complication is the endarteritis, which may end in cerebral hemorrhage.

In the tertiary stage we still have a possible endarteritis to deal with, with a possible cerebral hemorrhage, or we may have brain symptoms from gumma.

Gumma may appear in the other organs of the body.

Years after the original sore, locomotor ataxia or general paresis may develop.

Diagnosis: In a typical case the diagnosis is easy. There is the history of exposure, the typical sore appearing three to eight weeks later, and the constitutional symptoms, sore throat, secondary eruption, etc., six to eight weeks after the initial sore. Other cases are hard to diagnose. When the initial sore is extragenital it is frequently impossible to say whether it is syphilitic or not. Very often its syphilitic character is not suspected. The secondary symptoms will sometimes be the first clue to the nature of the disease.

The secondary stage, sore throat, eruption, loss of hair, etc., is usually definite enough. It should be remembered that the skin eruption of this stage is symmetrical. Induration is a characteristic feature. On the limbs it covers the flexor surfaces.

The tertiary stage attacks the deeper structures. The history of the case will usually furnish the clue to the cause.

We may employ Justus' test. This is an estimation of the hemoglobin before and after mercury inunction. If there is a 5 per cent. drop it means syphilis.

The blood and lesions may be examined microscopically for the spirochete pallida.

The Wassermann reaction is of great value if done by an expert.

Noguchi has devised a skin reaction. This is inoculation with a preparation called luetin. A red papule surrounded by a zone of redness appears. Osler says this reaction is more constant in the tertiary stage.

Prognosis: The prognosis of syphilis is uncertain. The expression has been used, "Once a syphilitic, always a syphilitic." It is true, the majority of patients apparently get well after two or three years, and nothing more is seen of syphilitic lesions. On the other hand, tertiary symptoms may develop ten, twenty, thirty, even fifty, years after the initial lesion. Also locomotor ataxia or general paresis may develop years after the disease has apparently disappeared. In other cases the disease may run a malignant course and kill the patient in a few months.

Treatment: Prophylaxis. The army and navy have adopted a method of personal prophylaxis for the men. It consists of enough mercury ointment for inunction of the parts immediately after a suspicious exposure. As long as primary or secondary symptoms are present the syphilitic is a possible source of contagion. He should be told and taught how to prevent contamination of others. Sex relations must be interdicted. He must not kiss anyone. His eating and drinking utensils must be thoroughly sterilized. Care must be exercised not to soil any object with the discharges from the lesions wherever situated, the primary sore or the mucous patches. Any object that is soiled inadvertently must be sterilized or destroyed.

The primary sore requires no treatment locally unless it breaks down, then dressing it with a 1-2,000 bichloride solution, or dusting it with some powder containing mercury, is the best procedure. Complete excision has been tried but it is useless. That excision should be useless is easy to understand when it is remembered that the sore does not appear until three to six weeks after exposure, and that during that time incubation has been taking place, and the system has become generally involved.

Cauterization has been recommended and advised against. My experience is against it. The most rapid case I ever saw—death in fifteen months—was a case in which I cauterized the primary sore.

When the diagnosis of syphilis is positive *mercury* in some form is the accepted method of treatment. This should begin immediately without waiting for secondary symptoms. If there is doubt as to the chancre being syphilitic wait for secondary symptoms so as to be sure. Or use some of the laboratory tests.

Fournier's method of treatment is probably the most reliable. Give protoiodide of mercury by mouth, ¼ grain every day for two months. Then stop a month or six weeks. Then another course of six weeks. And so on, using the mercury in this way for two or three years.

For the tertiary stage he uses *iodide of potassium*. He recommends thirty to forty-five grains a day in solution to be given a month or two, then a rest, then repeat, and so on for two or three years or more.

Inunctions of mercury may be given if the symptoms are very severe. Or injections may be used in emergency. The iodide of potassium may also be injected in very large doses if an immediate and powerful effect is desired.

Ehrlich's *salvarsan* and *neo salvarsan* are used very extensively now. They need to be given intravenously. At first one dose was said to cure. Now more are given. Most syphilographers follow the salvarsan with the usual mercury treatment.

Personally, I have treated some cases of syphilis very satisfactorily with the indicated homœopathic remedy.

Mercurius solubilis I have used in potency over a long period for an ordinary case with the initial sore and a mild secondary sore throat and eruption.

Mercurius protoiodide 2x has served in other cases.

Kali hydroiodicum, iodide of potassium, I have used in potency.

Phytolacca has cleared up syphilitic glandular enlargements.

Graphites has cured syphilitic pustular eruptions for me.

The main point in treatment is to continue it for at least two years, sometimes much longer, or until the Wassermann test becomes negative.

SECTION II.

Diseases Common to Man and Animals.

ACTINOMYCOSIS.

(Greek, ἀκτίς, a ray; μύκης, a fungus.)

Synonyms: Lumpy Jaw, or Big Jaw, in cattle.

Definition: An infectious disease caused by the ray fungus or *streptothrix actinomyces.*

Historical Note: This disease was formerly described under various names as occurring in cattle. In 1876 Bollinger gave an accurate description of the micro-organism as it appears in cattle. In 1877 Israel described a similar organism taken from the human subject. In 1879 Ponfick showed the identity of the two. Since then numerous cases have been described throughout various parts of Europe and the United States.

Etiology: The disease is more prevalent in animals than in the human subject. The germ is supposed to enter the body by way of an abraded mucous membrane in the mouth, or through carious teeth. It is thought to come from grain. Men are more often attacked than women.

Bacteriology: The specific organism is found in the diseased tissues in the form of clusters having a radiate structure. Stengel says there are two forms of the organism—the pathogenic being anaërobic. It may be cultivated on agar, or in raw or boiled hen's eggs. The organism can best be gotten from minute granules which are found in the pus from the affected tissues. These granules are yellowish in color and are approximately one millimeter, more or less, in diameter.

Morbid Anatomy and Pathology: In actinomycosis the tissues affected become swollen. There is infiltration and proliferation of the connective tissue. Later this may break down with formation of pus and ulceration. Sinuses form in the diseased parts. The lesions occur mostly in the mouth and about the

bones of the jaw. They may occur in the lungs, intestines, and other parts of the body. By metastasis localized lesions may occur anywhere. The disease simulates pyemia and can only be differentiated from that by the finding of the actinomyces in the pus.

Symptoms: The most common form of actinomycosis is that which attacks the jaw. The tissues become infiltrated and swollen. The swelling includes the bone and the tissues about the neck. Later suppuration takes place and sinuses develop.

The disease may extend down the intestinal tract. It may be primary in any of the internal organs. It may present itself principally as a disease of the lungs. When this latter happens the physical signs are those of chronic bronchitis or of pulmonary tuberculosis, but the lesions are usually unilateral.

There is usually an irregular septic fever. In the pulmonary form there is cough and expectoration. Metastasis occurs. There may be necrosis of the bones.

Complications and Sequelæ: The complications and sequelæ are merely the extensions of the symptoms to suppuration of the soft tissues, or to necrosis of the bones.

Diagnosis: Actinomycosis presents many of the symptoms of pyemia; for example, the septic fever, metastatic abscesses, and so on. The finding of the specific organism will differentiate it from other pus conditions.

On account of the gross appearance of the more common form, that found in the bones of the jaw, the disease was often mistaken for osteo-sarcoma, according to Osler. The microscope will assure the diagnosis.

In the pulmonary form the demonstration of the actinomyces in the expectoration will show the nature of the infection.

Prognosis: The disease runs a more or less chronic course of from eight to twelve months. If a vital part is not attacked there may be recovery. In the pulmonary form the result is usually death.

Treatment: The treatment is surgical in the majority of cases. Only one internal remedy is mentioned by authorities, and that is *iodide of potassium* in large quantities—forty to sixty grains a day.

Injections of *tuberculin* cured one case for Billroth.

Homœopathic literature is silent as to the treatment of this disease, so far as I know. There is no reason to suppose, however, that such remedies as *arsenicum, silicea, hepar* and other drugs useful for pus formation and necrosis should not be of service.

ANTHRAX.

(Greek, ἄνθραξ, a coal.)

Synonyms: Malignant Pustule. Wool Sorters' Disease. Rag Pickers' Disease. Splenic Fever.

Definition: A highly contagious disease due to the *bacillus anthracis*. It is common in sheep and cattle. In man it is due to accidental contamination from animals affected with it.

Historical Note: Anthrax is a very ancient disease. It is supposed that the plague of murrain and boils visited upon the Egyptians and their cattle in the time of Moses was anthrax (see Exodus, ix). Extensive epidemics of anthrax have occurred throughout Europe at various times. The bacillus anthracis was first described in 1850 by Rayer and Davaine. In 1876 Koch worked out its life history. He cultivated the bacillus outside the body, and produced the disease experimentally by inoculation. (Bell and Legge in Allbutt's System of Medicine, second edition, 1906.)

Etiology: This is the most widespread disease known excepting only tuberculosis. The specific cause is the bacillus anthracis. Osler says the disease is much more widespread in Asia and Europe than in this country. In man the disease is gotten by accidental inoculation, usually in handling hides or hair from diseased animals. Hence the names "Wool sorters' disease," "Rag pickers' disease," and similar titles.

In cattle the bacillus seems to be gotten in grazing rather than in direct contact. Pasteur supposed the bacillus to be in the soil and to be brought to the surface by earth worms. It has been found that once a field is infected it is almost impossible to eradicate it. After a time animals belonging to an infected area do not seem to be as susceptible as ones brought from another place. The disease may also be transmitted from animal to

animal by the bite of insects. An abraded surface is necessary for its entrance in man or animal.

Bacteriology: The anthrax bacillus grows in long segmented threads. The segments vary in length but are usually two or three times as long as broad with square or rounded ends. The bacillus is among the largest in size. The threads are closely interwoven and can be grown in various culture media. They are found in the pustules.

Morbid Anatomy and Pathology: The tissues near the seat of the lesion are much infiltrated and swollen. The point of entrance itself first shows as a pimple, then in thirty-six hours or so a dark eschar develops in the center with a few small vesicles about it. Around these again develops the much swollen and inflamed tissue. The lymphatics radiating from this area become red and swollen. The spleen becomes tremenduously enlarged.

After death the blood is found to be dark. Hemorrhagic extravasations are found about the pustule. Rigor mortis is marked.

Symptoms: The incubation period of anthrax is from a few hours to three days. There are two forms of the disease (a) *external* and (b) *internal.*

(a) *External anthrax* appears on some part of the body that is usually exposed. Within a few hours after inoculation a reddish papule appears accompanied by itching nad burning; later this becomes vesicular. After twenty-four to thirty-six hours a blackish eschar forms, which, if the case is favorable, is thrown off leaving a cavity. Surrounding the initial papule, in all cases, is a row of smaller vesicles. There is marked infiltration of the surrounding tissues with swelling and edema. The radiating lymphatics are red, swollen and tender. The case may end in recovery, or general sepsis and death may occur. The temperature is at first high and accompanied with chills and prostration. Later the temperature may become subnormal.

(b) *Internal anthrax* is due to the ingestion or inhalation of the specific bacillus. When the initial lesion occurs in the intestine we have symptoms of acute poisoning, vomiting, diarrhea, sudden rise of temperature and profound prostration.

If the initial lesion is in the lungs there is rise of temperature, chilliness, prostration, aching all over, cough, and all the signs of a septic pneumonia.

Internal anthrax is practically always fatal.

Diagnosis: The rapidity of the development of the pustule, the eschar, the vesicles surrounding it, and the great edema make diagnosis of the external form certain. In the internal form the occupation of the patient should cause suspicion in a patient suddenly overcome with the intestinal or pulmonary symptoms noted above. Although comparatively rare in the United States, cases have occurred, especially in tanneries.

Prognosis: Anthrax is always a serious disease. In the external form the mortality has run as high as twenty-six per cent. The internal form is nearly always fatal in a few days.

Treatment: Infected animals should be cremated. In places where hides, wool or hair are handled precautions should be taken to prevent anthrax. In England, according to Bell and Legge (Allbutt's System of Medicine, Vol. II.), the factory laws require the following:—Sorting of wools on perforated tables with exhaust ventilating system underneath. Burning of dust so collected. Washing of clothes and eating utensils. Exclusion of persons with uncovered cuts or sores. Requisites for treating wounds. Absolute cleanliness of workrooms and destruction of clippings. Notices posted telling what to do in case of suspected infection.

In the external form of anthrax complete extirpation by the knife or cautery as early as possible is the best treatment. In the internal form the only remedy mentioned by the text-books is *ipecacuanha* powder in ten grain doses. Muskett believes it a specific.

Bell and Legge also recommend the use of anthrax serum. They quote various writers who have used it with success. They inject 30 or 40 c.c. in 10 c.c. doses in different parts of the abdominal wall at the same time. A rise in temperature is a favorable indication. If the symptoms are not better in twenty-four hours the injections are repeated. In fifty-six cases where this treatment alone was used the duration of the illness was eight to fourteen days.

Anthracinum is the nosode for anthrax. It covers the symptoms of pustule, surrounding edema, and so on.

Lachesis is indicated in the profound septic condition, especially if the case becomes purpuric showing disintegration of the blood.

Secale is indicated in cases better from cold, worse from warmth. The part is gangrenous.

Arsenicum album is indicated if the patient gets into a typhoid condition, with great restlessness from exhaustion. Worse at night.

FOOT AND MOUTH DISEASE.

Synonyms: Aphthous Fever. Epidemic Stomatitis.

Definition: An acute infectious disease met with mostly in cattle, pigs and sheep, more rarely in man.

Historical Note: Probably foot and mouth disease has existed for a long time in Eastern Europe. It was described by Italian writers in the sixteenth century. Its first description in Great Britain appeared in the Veterinarian in 1839 (M'Fadyean in Allbutt's System of Medicine). It is not common in the United States, though there have been outbreaks of it.

Etiology: It occurs as an epidemic and spreads with great rapidity when it gets started. No specific germ has been isolated. Every case though is supposed to originate from some preceding one.

In man its occurrence is accidental. It may come from using milk, butter, or cheese from infected cattle. It may also occur by direct infection through a wound or abraded skin or mucous membrane.

Bacteriology: The fluid from the foot and mouth disease vesicle is virulent, even when passed through a porcelain filter. No germ has been discovered.

Morbid Anatomy and Pathology: The characteristic lesion is a series of vesicles which appear on the gums and mucous membranes of the mouth, and on the feet. In cows these vesicles often appear on the udder as well. The vesicles are small and grayish. The fluid first is clear, then turbid. Movements of the jaw frequently break them, leaving a raw surface. There is also salivation.

Symptoms: The period of incubation seems to be short, two or three days or less. In man the symptoms frequently appear within twenty-four hours after infection.

There is first fever, sometimes slight chilliness and diarrhea. After twenty-four to forty-eitght hours the affected parts—usually the lips, gums, and buccal mucous membrane, become swollen and covered with vesicles. In cattle the feet are affected, and in cows the udders also. In men the fingers and hands are often attacked, and in women the breasts are usually affected.

The symptoms last eight to ten days and then disappear, as a rule. Very young children may die from the disease.

Complications and Sequelæ: In foot and mouth disease other pathogenic organisms sometimes enter through the raw surfaces aggravating the symptoms.

Diagnosis: The diagnosis of foot and mouth disease should be easy as the appearance is quite characteristic. As a rule, the disease appears as an epidemic among animals and, at the same time, cases occur in man.

Prognosis: In man the prognosis is good except in very young children.

Treatment: When foot and mouth disease prevails all milk used should be boiled.

Antiseptic precautions should be used by those caring for animals.

A serum has been prepared by Loeffler, which appears to be useful in animals.

Apis is indicated by the vesicular nature of the disease. The patient is apathetic. Worse from warmth.

Rhus toxicodendron has a vesicular eruption. The patient is restless. Worse from cold.

Croton tiglium has a vesicular eruption that tends to pustulate. There is intense itching.

Mercurius is said by Hughes to be almost specific for the stomatitis.

GLANDERS.

(Latin, *glans,* an acorn.)

Synonyms: Farcy. (Latin, *farcio,* to stuff.) Equinia. (Latin, *equus,* a horse.)

Definition: An infectious disease of the horse and ass characterized by purulent discharges from the nose and development of nodules in the nares and under the skin. It is sometimes communicated to man.

Historical Note: Aristotle in 335 B. C. described a disease of the ass which was probably glanders. Vegetius in the fifth century described farcy. Many other writers since have written on the subject. In 1783 its communicability to man was recognized. Löffler and Schütz isolated and described the bacillus in 1882.

Etiology: The disease is caused by the bacillus mallei. In man the disease occurs usually in those who have to do with horses. The infection takes place through an abraded surface of the skin or mucous membrane. It may be transmissible from man to man.

Bacteriology: The bacillus mallei is somewhat similar in appearance to the tubercle bacillus. It is of varying length, with rounded ends, non-motile, and capable of cultivation on various media.

Morbid Anatomy and Pathology: Small granulomatous tumors develop in the nasal passages (glanders) or under the skin (farcy). These tumors are composed of lymphoid and epithelioid cells, and are found filled with bacilli. The tumors tend to suppuration forming ulcers in mucous membranes and abscesses in the skin.

Symptoms: There is an acute and there is a chronic form of glanders. *Acute.* The period of incubation is most often only a few days, rarely two or three weeks. The onset of the *acute* form is ushered in by chills, fever and aching pains in various parts of the body. At the point of inoculation there are redness, swelling and tenderness. In mucous membranes the inflammation extends along the lymphatics. Under the skin nodules (farcy buds) develop. An ichorous discharge which soon changes to blood and pus comes from the nose. There is a development of papules which change to pustules about the face and sometimes over the joints. This has been mistaken for small-pox.

The lesions of the mucous membrane suppurate, and ulceration, sometimes necrosis, takes place. In the so-called farcy buds through the skin suppuration may also take place forming abscesses. The patient succumbs in a week or ten days to a general septic condition.

The *chronic* form is of slower onset. The lesions are scanty but persistent. The symptoms may last for months when the patient will eventually recover, or more rarely there will be an abrupt onset of the acute form and death in a few days.

Complications and Sequelæ: Osler says subacute pneumonia is common in the acute form.

Diagnosis: An acute and apparently virulent type of coryza occurring in any one who is constantly with horses should arouse suspicion. If the coryza is accompanied by the pustular eruption and farcy buds the diagnosis is certain. If in doubt the microscope or the inoculation of guinea pigs will settle the question. More recently *mallein*—a toxin of the bacillus mallei—has been used for diagnostic purposes. Injected in an animal suffering from glanders it will cause a febrile reaction; in healthy animals none.

The acute form in the beginning has sometimes been mistaken for typhoid fever. In the chronic form the ulcers may be mistaken for syphilis or tuberculosis.

Prognosis: The acute form is nearly always fatal in a few days —eight to ten. The chronic form may run for months and the patient recover. More rarely the patient finally dies.

Treatment: Prompt isolation of animals affected with glanders is of the first importance as a prophylactic measure. Individuals handling horses should use care not to become infected through an abraded surface.

A few favorable reports have been published on the use of *mallein* injections.

Hughes suggests *kali bichromicum* as being "exquisitely homœopathic" to the nasal and skin symptoms of glanders.

Mercurius if there is a tendency to pus formation, and to lymphatic involvement.

Crotalus or *lachesis* in case of profound sepsis.

HYDROPHOBIA.

(Greek, ὕδωρ, water; φοβος, fear.)

Synonym: Rabies.

Definition: A disease of animals, more particularly of dogs, due to a specific virus and communicable to man. It is characterized by spasm of the muscles of deglutition. The name, hydrophobia, signifies a fear of water, one of the prominent symptoms in man. At the end there is a general paralysis.

Historical Note: This disease is mentioned by many of the Greek and Roman classical writers, who described its unpleasant symptoms very accurately. Extensive observations have been carried on by many observers for the last two centuries. In 1813 Magendie and Bouchet produced rabies by inoculation. Pasteur, during the last quarter of the nineteenth century, finally developed a protective serum which seems to serve as a preventive—not as a cure.

Etiology: The cause of hydrophobia is contained in the saliva of the rabid animal. It has never been isolated in its purity. It is transmitted by the bite of the affected animal, most often the dog, less often the cat, sometimes by the wolf or skunk. Noguchi, of New York, announced that he had finally isolated the specific virus of rabies in 1913.

Morbid Anatomy and Pathology: The changes in this disease are in the central nervous system and are microscopic. The virus exists only in the medulla, spinal cord and peripheral nerves. There is an accumulation of round cells about the nerve cells. There is degeneration of nerve cells and peripheral ganglia.

Symptoms: The period of incubation is six weeks to two years.

In the first stage there is a feeling of anorexia, general discomfort and depression. There is pain about the seat of the wound and the lymphatics may be swollen and painful. There may be a slight rise in temperature. There is dread of liquids and, perhaps, some pain on swallowing of them. This stage lasts a day or two.

In the second stage, or the stage of excitement, the slightest

thing sends the patient off into violent spasms, particularly of the pharynx and larynx. There is a sense of oppression and fear, and dyspnea is marked. Sometimes these toxic convulsions extend to the trunk and limbs. The temperature may go to 103° F., or higher. The patient is unable to swallow, and the mere sight of water frequently causes the convulsions (hence the name hydrophobia). During the convulsions the patient is delirious and often maniacal. They last from a few minutes to half an hour. In the intervals the mind is clear. There is great thirst, and usually salivation. This stage lasts a day and a half to three days.

The third stage lasts six to eighteen hours. There may be a few violent convulsions, but, as a rule, the patient becomes quiet, unconsciousness develops, the heart gradually becomes weaker and weaker, and death closes the scene.

Diagnosis: The diagnosis is easy, especially if we know the person has been bitten. There is a pseudo-hydrophobia which is an hysterical manifestation occurring in nervous people sometimes. But the main symptom is the fear of water. The patient does not die but recovers from the hysterical form.

Prognosis: True hydrophobia is always fatal.

Treatment: Treatment is purely prophylactic. When once developed hydrophobia is not curable.

The best prophylaxis is to have all dogs muzzled. This has practically eradicated the disease in some parts of Germany.

When bitten, the victim should immediately have the wound cauterized with caustic potash or pure carbolic acid. Chloroform may be administered to allay the spasms. Injections of morphine may be used for the same purpose.

Pasteur developed a prophylactic serum. He carried the virus (part of a spinal cord) through a series of rabbits by inoculating them successively, the virus from each being more powerful than that of the one preceding, until the period of incubation was only seven days. He used the cords of these last to inoculate human beings who have had suspicious bites. The results have been uniformly successful in the many places where it has been tried. That is, the mortality is reduced to one per

cent. or less. Pasteur's method serves only to prevent, not to cure.

Hughes speaks of *belladonna* and *stramonium* as being homœopathic to hydrophobia. The pathogenesis of *stramonium* says the sight of water or of glittering objects will throw the patient into spasms.

Strychnine in potency might be tried for the spasms.

MILK SICKNESS.

Synonym: The Trembles.

Definition: An infectious disease of man and animals, formerly very prevalent in the United States, west of the Alleghenies. In animals it is known as the "trembles."

Etiology: It seems to be transmitted to man by using the flesh or milk of affected cows, or by using butter or cheese made from such milk. Cattle seem to be infected in some way from the soil.

Morbid Anatomy and Pathology: No special lesions are found.

Symptoms: Incubation is short—two or three days. There is a feeling of malaise. The breath is fetid, constipation marked. The tongue becomes swollen and tremulous. There are nausea and vomiting. Fever is slight. Restlessness, delirium and coma may intervene, and death close the scene in a few days.

Diagnosis: The diagnosis may be difficult unless it is known that animals are affected.

Prognosis: The prognosis is favorable, as a rule, though it sometimes proves fatal.

Treatment: Prophylaxis consists in not eating the products of infected cattle.

Nux vomica is indicated for the nausea and vomiting and the constipation.

Arsenicum album is indicated in the nausea, vomiting and restlessness with or without delirium.

Ipecac is indicated in nausea and vomiting.

PSITTACOSIS.

(Greek, ψιττακός, a parrot.)

Definition: An infectious disease of birds sometimes communicated to man. The respiratory symptoms are the most marked.

Historical Note: According to J. M. French it was introduced into Paris in 1892 by a cargo of parrots and parrakeets. I am indebted to that author for all the information here written.

Etiology: Psittacosis originates in contact with birds or articles contaminated with their dejections.

Bacteriology: The bacillus of Nocard is supposed to be the specific cause. It is a motile rod with cilia.

Morbid Anatomy and Pathology: The lesions are usually those of bronchial catarrh. Occasionally a diphtheritic membrane is developed.

Symptoms: Incubation lasts one to two weeks. Malaise with nausea and vomiting develops. The onset may be sudden. The temperature rises to 103° or 104° F., lasts four or five days, subsides for a few days, then returns a few days. This may go on two to three weeks. The physical signs are those of broncho-pneumonia.

Diagnosis: The diagnosis is based on whether or not the patient has been in contact with parrots, and by the finding of the bacillus.

Prognosis: Reported cases have a mortality of twenty to forty per cent.

Treatment: The patient should be isolated. Cold sponges are useful. Liquid diet should be given.

Ipecac is indicated more closely than any other remedy. It has nausea and vomiting, and it has the respiratory symptoms.

Antimonium tartaricum is somewhat similar, except that the breathing sounds would be coarser.

Kali bichromicum would be indicated if the characteristic stringy mucus appeared.

develop early as a rule are more dangerous than those that come later. My case developed one week after the original wound was made by stepping on a nail, and the patient died in twenty-four hours.

Treatment: Prophylaxis is the first essential. Tetanus is much less frequent now than it used to be because of the greater care exercised in cleansing and keeping clean wounds of any kind.

An antitoxin has been prepared for the treatment of tetanus, but its curative properties are not yet well established. It was used in my case without success. As a prophylactic it has been used extensively for a few years in certain cities after fourth of July celebrations. It seems to have been of some value.

The spasms may be controlled with chloroform inhalations, with morphine injections, or with doses of bromide of potassium.

Passiflora is used in the West Indies. The dose is half a dram to a dram of the tincture.

Homœopathy has accomplished cures in tetanus. *Strychnine* is the drug most often indicated. It should be given in potency from the third to the thirtieth.

Nux vomica might cover the case. The spasmodic symptoms are less marked than in *strychnine.*

Hydrocyanic acid is mentioned by Hughes. It also has convulsions with trismus. The third to the thirtieth potency is best.

Goodno mentions *physostigma;* its keynote is tetanus, rigidity of muscles.

VERRUCA.

Synonyms: Carrion's Disease (named after Daniel Carrion). Oroya Fever.

Definition: An infectious disease characterized by irregular fever, anemia, rheumatic pains, and an eruption of bleeding tumors.

Historical Note: According to Giltner the disease appears to be limited to certain parts of Peru. It is supposed to have carried off many of the ancient Peruvians, and many of the

followers of Pizarro during his conquest of Peru. It has been described since 1730 by Peruvian writers. The disease was brought into prominence in 1870 during the construction of the Oroya railway over the Andes.

A Commission sent out from the Harvard Medical School reported in 1913 that verruca and Oroya fever differed in some respects.

Etiology: At present the disease is found only in certain narrow valleys on the western slopes of the Andes in Peru at an altitude of 2,000 to 6,000 feet. Apparently it can be contracted only by going to the place where it is endemic. Simply passing through the country is sufficient. Natives are immune.

Both sexes and all ages are susceptible.

Animals are as liable to infection as are human beings.

It is probably transmitted by some insect. Townsend says a biting gnat acts as an intermediate host.

Bacteriology: A bacillus has been described by Barton as being the specific organism of verruca. It somewhat resembles the tubercle bacillus but is thicker. It grows on bouillon. It is found in the red blood cells.

Morbid Anatomy and Pathology: Giltner says the organs are pale and bloodless. The liver and spleen and lymphatic glands are enlarged. There is hyperplasia of Peyer's patches and congestion of the intestinal mucous membrane. The skin sometimes presents a petechial, later a warty eruption that may ulcerate.

Symptoms: The period of incubation is fifteen to forty days. The onset is like that of other acute infections. Malaise, headache, chilliness, fever, pains in the legs, thirst, and insomnia. The glands are enlarged.

The fever is intermitting or remitting, going to 104° F. or higher at night. This lasts about four weeks.

The most prominent symptom is the progressive anemia. The red cells drop to 1,000,000 or less. There is a marked leucocytosis and increase of polynuclears. As a result the skin and mucous membranes are waxy looking.

In mild cases the patients may be up and about. In severe

cases the patients sometimes have hemorrhage from the nose and bowels. They suffer from vertigo and syncope.

The respirations are hurried. There is marked dyspnea. The pulse is rapid, up to 140. There is delirium. Edema of the feet and legs sometimes comes on.

Another distinctive feature is the eruption. This comes on after the fever goes down, usually from one to five months later. The general symptoms become severe again at this time. The eruption appears on the skin and on the mucous membranes. It is also found on the peritoneum, all the organs, the bones and the brain. It may be miliary or globular, or anything in between. The spots vary in size from a pin head to a pea. They may be localized or general, discrete or confluent. According to Giltner exfoliation and bleeding occur and the large spots ulcerate.

The globular form develops into tender tumors that may attain the size of an orange. These break down and ulcerate also.

The eruptive stage lasts from several months to several years.

Complications and Sequelæ: Tuberculosis, pneumonia and typhoid are frequent complications. Septicemia is often an end result.

Diagnosis: The disease was formerly considered a form of malaria. Quinine affects malaria, but not verruca. The plasmodia are found in malaria. There is no lymphatic enlargement in malaria.

Yaws is somewhat similar in appearance, but the general health is not involved.

Prognosis: The prognosis is always grave, the mortality in severe cases being as high as ninety per cent.

Treatment: Prophylaxis. Avoid the locality where it exists is the only sure preventive.

Symptomatic treatment would call for remedies like

Phytolacca for enlarged glands.

Thuja for warty growths.

Anthracin for ulcerating surfaces.

Arsenicum album for a general septic condition.

SECTION III.

Worms and Intestinal Parasites.

PIN WORMS.

Synonym: Oxyuris Vermicularis.

Definition: A small round worm. The female is about twelve millimeters long, the male three or four.

Description: The eggs get into the human stomach where they are hatched. Thence they proceed to the lower part of the small intestine and cecum. When mature they travel down the intestine to the rectum. The female goes outside the anus to lay her eggs.

Etiology: The ova are probably first ingested in water or green vegetables like lettuce that have been washed in water. Autoreinfection is possible.

Symptoms: The principal symptom is the itching at night when the worms crawl through the anus. This causes restlessness and irritation, and occasionally anemia. Occasionally they are the cause of sexual irritation as well.

Pin worms are much more common in children, though they may be present in adult life.

Complications and Sequelæ: In rare cases peri-rectal abscesses occur containing the worms.

Diagnosis: The diagnosis is made by finding the worms or eggs in the stools.

Prognosis: The prognosis is good. In rare cases only are the worms hard to get rid of.

Treatment: Absolute cleanliness is the first requisite in prophylaxis. *Santonin* is the usually recommended remedy. Enemas of salt and water should be used with the medicine. Enemas need to be given daily for ten days or two weeks. I am in the habit of giving Rubinat water, half a glass to a glassful first, then the enemas.

With these I use *aconite* if the patient is restless and feverish.
Cina if no other remedy is specially indicated.
Symptomatic prescriptions are used in all cases.

ROUND WORMS.

Synonym: Ascaris Lumbricoides.

Definition: Ascarides are round worms of a pale-reddish or yellowish-brown color. The male is four to eight inches long, the female seven to twelve inches. This is said to be the most common human parasite.

Description: This worm is cylindrical in shape, and tapered at the ends. Toward the tail the body is somewhat flattened. It has a striated appearance. The ova are small and are produced in great quantities. Their usual habitat is the small intestine. One to ten is the usual number found. Rarely they are very numerous.

Etiology: Ingestion of ova is the mode of infection. They may reproduce themselves in the patient. No age is exempt, though they are found most often in children, probably because they are more careless in their habits.

Symptoms: Lumbricoides may exist without producing any symptoms. In other cases there may be nausea and vomiting, colicky pains in the abdomen, sometimes diarrhea. In nervous children there may be restlessness, grinding of the teeth, irritability of temper.

Sometimes the worms migrate. They may get into the stomach and be vomited. They may get into the nose or Eustachian tube. Sometimes they travel into the gall ducts. Osler says death has occured from asphyxiation by migration into the larynx.

Complications and Sequelæ: Serious symptoms may result from migration to other parts.

Diagnosis: The diagnosis is made certain only by finding the worms.

Prognosis: The prognosis is good except in the rare cases of migration to vulnerable parts.

Treatment: *Santonin* is the best remedy. It may be given in doses from one-quarter to two grains, according to the age. It should be given in the form of a troche on an empty stomach, and followed by a purge. Rubinat water is my favorite. It may be given one-half to one glassful. Enemas may also be used. The *santonin* after the first day may be given each morning before breakfast for two or three days.

For the general irritation sometimes caused homœopathic remedies may be necessary, but they will be of no particular help until the worms are gotten rid of.

TAPE WORMS.

Synonyms: Cestodes. Teniæ.

Definition: Tape worms are long flat worms consisting of a head and numerous segments which, in the adult stage, inhabit the intestines of man and some animals. In the larval stage they exist in the muscular tissue of animals eaten by man.

Description: Of the various kinds of tape worms only three are found in man with sufficient frequency to require special notice: (1) *Tenia saginata* or *mediocanellata,* (2) *tenia solium,* and (3) *bothriocephalus latus.*

A tape worm consists of a head or scolex approximating in size the head of a pin, and numerous flat segments or proglottides joined together in a long band behind the head. Each segment consists of a body longer than it is wide and containing both male and female sexual organs. The uterus is filled with eggs. The head or scolex has suckers and usually claw-like hooks with all of which it attaches itself to the intestine of its host.

The teniæ may exist in man in the fully developed tape worm state in the intestine, and occasionally in the larval state in the muscles and parenchymatous organs. The bothriocephalus exists in man only as the developed tape worm. The larvæ in muscular tissue are also known as "measles."

(1) The *tenia saginata* or *mediocanellata,* the unarmed or beef tape worm, exists in the larval state mostly in cattle-beef. It is found in the muscular tissue and in the heart. It attains a

length of twelve to twenty feet or more. The head is larger than that of the *solium*. It has no hooks. The uterus is filled with eggs. These eggs when discharged do not develop in the original host, but if they get into the stomach of the cow the outside shell is dissolved and the embryo finds its way into the muscular tissue where it becomes encysted and develops into a scolex. When such muscular tissue is eaten raw or not well cooked, the cysts are destroyed, and the scolex attaches itself to the intestine of its new host, there to grow its segments and become a full fledged tape worm. This is the most common form in the United States.

(2) The *tenia solium*, the armed or pork tape worm, goes through a similar process with the hog as the intermediate host. It is not so large as the *saginata*, averaging but six to nine feet in length. All its parts are smaller than the beef worm. The head, besides having suckers, also has hooklets. There is next the head a narrow neck nearly an inch long. This form is not so common in the United States.

(3) The *bothriocephalus latus*, or fish tape worm, is found in certain parts of Europe and Japan. Osler says it is found in the United States only in imported cases. It has no hooklets. It attains a length of twenty to thirty feet.

Etiology: The patient becomes infected from raw or underdone meat or fish.

Symptoms: Tape worms may exist without producing any symptoms. At other times there may be anorexia, nausea, vomiting, sometimes abdominal pains. In nervous persons the suspicion of tape worm may cause anxiety, depression and even hypochondriasis. Some patients may have large appetites and yet not be well nourished. Occasionally the worm will produce anemia.

Diagnosis: The diagnosis is made sure by the finding of segments or ova in the stools. If tape worm is suspected a cathartic should be given to remove some of the segments.

Prognosis: A tape worm rarely does any permanent damage.

Treatment: The best prophylactic is never to eat any but thoroughly cooked flesh food. If segments of tape worm are discovered in the stools they should be destroyed to prevent infecting others or to prevent auto-infection.

When tape worm is known to exist, and it is decided to get rid of it, the patient must be put on a very light diet for two or three days. An occasional mild cathartic should be given together with saline enemas.

Remedies should be given on an empty stomach. The ethereal extract of male fern in two dram doses followed in a couple of hours by a purgative is often efficient. A fresh preparation is desirable.

Three ounces of pumpkin seed, thoroughly bruised and macerated, may be used.

A boy of 16 in the Flower Hospital for "stomach trouble" passed six feet of tape-worm, which made the diagnosis for us. He was kept without food for twenty-four hours. Then he was given one dram of male fern in black coffee, followed in an hour and a half by one ounce of compound powder of jalap. About three hours later he had nausea and vomiting and passed eighteen feet of worm with the head.

I have been able to get rid of tape-worm piecemeal by the following method, extending over a week. Withhold food for twenty-four hours, then in the morning a full tumbler of Rubinat water. After a day or two repeat. After another day or two repeat a third time if necessary.

I usually give *nux vomica* tincture throughout as a remedy.

An absolute cure is not assured unless the head comes away. As this is small it may be overlooked. The dejecta should be received in a vessel containing warm water so that the worm will not be broken and can be examined. After removal of a large part of the worm, even if the head does not come away, there may be no further symptoms.

PSOROSPERMOSIS.

(Greek, ψώρα, the itch; σπέρμα, seed.)

Synonym: Psorospermiasis.

Definition: Psorospermosis is a disease caused by psorosperms which cause local disturbance in the organs affected. Psorosperms are sometimes known as sporozoa, or gregarinidæ. As

they attack the cells they are also called cytozoa. They are a low form of protozoa. The most frequent forms attacking man are the coccidia, rarely the sarcosporidia.

Historical Note: The name psorosperm, according to Bulloch, was first used by Müller in 1841 to designate certain organisms found in diseases of fish. Later the term was used to describe organisms found at times in man. More recently many conditions formerly supposed to be due to psorosperms have been found to be due to other causes. This is especially true of skin lesions.

Etiology: The coccidium oviforme found in rabbits has caused more cases in the human subject than any other form. The coccidium bigemineus found in cats and dogs has been found in man. Likewise a coccidium found in pigs and known as "Rainey's tubes" has affected a few subjects.

The coccidia cause disease of the internal organs of animals harboring them and are thrown off in the stools. In this way fresh vegetables, like lettuce, may become soiled and the organisms carried thence into the human stomach.

Bacteriology; The coccidiæ are oval in shape, about twice as long as wide, and give off spores.

Morbid Anatomy and Pathology: The coccidium oviforme causes nodules in the liver which resemble tubercles. Sometimes cysts are found. The other forms are found in the epithelia of the intestines, sometimes in the kidneys.

Symptoms: There is malaise, chilliness and slight rise in temperature indicating systemic infection. If the liver is the seat of the trouble it becomes tender and swollen, and cysts or nodules may become palpable. If the digestive tract is the seat of trouble, there are nausea, vomiting and diarrhea. There may be general tenderness over the abdomen and even peritonitis. If the organisms reach the kidneys there will be hematuria.

Diagnosis: The diagnosis will of necessity be based on finding the psorosperms with the microscope. The symptoms are not idiopathic and may be the same as those of mild poisoning.

Prognosis: Death may occur in ten days to two weeks. The majority of cases probably recover.

Treatment: Prophylaxis may be carried out by having all green vegetables thoroughly cleansed before use.

Various authorities recommend enemas of 1-5000 to 1-1000 of quinine in the intestinal form of the disease.

HYDATID DISEASE.

(Greek, ὑδατις, a vesicle.)

Synonym: Ecchinococus Disease.

Definition: Hydatid disease is disease caused by the cysts of the *tenia ecchinococcus.* These may occur in various parts of the human body.

Historical Note: Clinically, hydatid disease was known as far back as the time of Hippocrates. According to Stirling and Verco (Allbutt's System of Medicine), Redi, in 1684, first suggested that it probably had its origin in animal parasites. During the past century more definite knowledge has been gotten as to the life history of the ecchinococci and the way in which they enter the human subject and cause trouble.

Description: The *tenia ecchinococcus* has its normal habitat in the small intestine of the dog. The worm is about 5 millimeters in length. Its head is provided with hooklets and suckers. The remainder of the worm consists of four or five segments. The adult proglottides, filled with ova, are given off in the feces. Thence they reach the intestinal tract of herbivorous animals, or of man, where the external membrane is dissolved and the embryo set free.

From the digestive tract it finds its way, probably through the lymphatics, into some viscus where it remains and develops. This, the larval or bladder stage, consists of a membrane enclosing a granular mass. This granular mass becomes liquified and the whole mass enlarged. Being a foreign body within the structures of the host, it excites the growth of connective tissue outside of itself which growth tends to enclose it.

New heads, or scolices, form within the bladder or cysts. In some cases daughter bladders are thrown out thus forming many cysts. If these cysts are discharged and the scolices reach the intestine of the dog, each scolex develops into a full grown worm.

Etiology: Man acts only as an intermediate host, and infection is by accidental ingestion. The disease is rare in the United States. It is common in Iceland and Australia, where dogs are very numerous.

Neither sex is exempt. According to Stirling and Verco no age is exempt, although the incidence of the disease increases to the close of the fifth decade.

Morbid Anatomy and Pathology: In by far the largest number of cases of hydatid disease the liver is affected—60 to 70 per cent.; next in order of frequency the lungs—10 to 12 per cent.; then the other abdominal organs; and lastly the nervous system.

The formation of hydatids in the tissues form enlargements of the particular part affected. The microscope will show the hooklets of the scolices in the fluid of the cysts.

Symptoms: The ecchinococcus cysts are of slow growth. Not until they are of considerable size do they cause any particular symptoms other than those of tumor. Rarely they cause pain by pressure on a nerve. Occasionally, by pressure on a vein, they cause an impediment in the return flow of blood, and consequent edema.

The hydatid tumor is a benign tumor and may cause no systemic disturbance whatsoever. Quite large growths have been found at autopsy, which were not suspected during life. If for any reason the tumor ruptures, however, suppuration is apt to take place and all the classical symptoms of pus infection appear:—namely, chills, fluctuations of temperature, increased heart action, emaciation and so on.

The liver is the viscus most often affected. In the majority of cases there is gradual enlargement which may become enormous, extending up under the ribs and down to the pelvis. Usually the border of the liver is smooth. In the so-called multilocular form it may be nodular. In a few cases fluctuation may be elicited. Ordinarily the only symptoms are from weight and pressure.

The tumor may rupture, causing septic symptoms. Or, it may rupture into the hepatic veins, then into the pulmonary vessels, causing death. It may rupture into the chest, or into the abdominal cavity.

Hydatid tumor of the lungs will give the symptoms of pleuritic effusion:—that is, restricted motion, loss of fremitus, dullness on percussion, and weakened respiratory sound on auscultation.

Hydatid tumor of the other abdominal viscera or of the nervous system causes symptoms of enlargement and of pressure only; there are no characteristic symptoms.

Rupture of hydatid tumor in any part of the body is frequently followed by urticaria.

Complications and Sequelæ: A rupture of a hydatid tumor into the chest or abdomen usually causes acute inflammatory symptoms that tend to run into a chronic septic poisoning. Possibility of sudden death has been mentioned.

Diagnosis: Discovery of the hooklets or scolices by microscopic examination of the tumor fluid is pathognomonic of hydatids. Rarely the fluid is sterile, then the diagnosis is in doubt.

Hydatid tumor may appear in the kidney and on rupture cause renal colic. The microscope must be called into use here as elsewhere.

Prognosis: Some cases of hydatid disease go on to spontaneous cure. Most cases gradually get worse unless relieved by surgical measures. Tumors that rupture may be suddenly fatal, or more often suppurate and kill by sepsis.

Treatment: Prophylactic treatment is the best. Persons living where hydatid disease is prevalent should use the utmost care in handling dogs. Fresh vegetables or water used should be beyond suspicion of contamination by excreta.

In case the disease exists medicines are said to have no effect. Complete surgical removal is the only cure.

TRICHINIASIS.

Synonyms: Trichinosis. Trichinelliasis.

Definition: Trichiniasis is a disease caused by the *trichina spiralis*, a small worm whose supposed normal habitat is the rat. Hogs are accidentally infected by the rats. The embryos are encysted in the muscular tissue. It is invariably introduced into man by the eating of raw or insufficiently cooked

pork. The embryos are set free in the intestines. In a few days the worms mature and produce numerous embryos which migrate to the muscular tissues of the new host where they coil themselves up and become encysted—muscle trichinæ.

Historical Note: Trichinosis was first recognized as an important disease in 1860, when Zenken, of Dresden, discovered the trichinæ in the body of a girl who was thought to have died of typhoid. Since then many epidemics have been reported. The disease is now known to be widespread.

Etiology: As noted above, trichiniasis is caused by eating infected pork. Meat infected with trichinæ is said to be "measly."

Morbid Anatomy and Pathology: The worm can be found in the infected tissues with a low power lens. The worms are coiled up in a lemon shaped cyst containing an albuminous fluid.

Symptoms: The first effect of ingestion of trichinæ is to cause acute gastro-enteric disturbance—nausea, vomiting, pain, diarrhea, and so on. During the first week or ten days the worms are maturing and throwing off embryos. The embryos migrate, finding a resting place in the muscular tissues.

At the end of ten days pains develop in the muscles. The muscles become swollen and tender to the touch. The patient has a temperature of 103° F., or higher—even 106°.

A remarkable feature of the disease at this time is the appearance of edema of the face which subsides in a few days. During the fourth to fifth week a general edema takes place involving the entire body except the genitals. A miliary rash is quite constant. Eosinophilia is marked.

At the end of five or six weeks, in favorable cases, the symptoms gradually subside and the patient eventually recovers.

Complications and Sequelæ: The complications of trichinosis are really due to the part affected. If the diaphragm or the intercostal muscles are affected we have embarrassment of respiration. If the laryngeal and pharyngeal muscles, speech and swallowing are interfered with.

In severe infection the patient may run into a typhoid condition and eventually die from exhaustion.

Diagnosis: Trichinosis usually occurs among groups of persons who have partaken of the same food at some function. It is more prevalent among Germans, who eat various foods prepared from uncooked pork. If trichinosis is suspected a portion of the food should be obtained, if possible, for microscopic examination.

The severity of the initial gastro-intestinal symptoms has caused it to be mistaken for cholera.

The long continued diarrhea, fever, and other systemic symptoms have caused it to be mistaken for typhoid.

The myositis simulates muscular rheumatism.

Prognosis: The mortality in different epidemics varies from one to thirty per cent. The disease runs a more favorable course in children than in adults.

Treatment: Trichinosis can be avoided by never eating of uncooked or underdone pork.

After the worms reach the muscles medicines are of no avail. If it is known that trichinæ have been ingested as many as possible should be gotten rid of while they occupy the intestinal tract by means of emetics and cathartics.

TREMATODOSIS.

(Greek, τρημα, a pore.)

Synonyms: Flukes. Distomatosis.

Definition: Disease caused by trematode or fluke infection is called trematodosis.

Historical Note: Most of the trematodes have been discovered and described since 1875, a few have been known for a century. They are more common in Africa, Asia and Europe than in the United States.

Description: A number of forms of trematodes affecting man have been described. They are flat, unsegmented, leaf shaped organisms. The life history is somewhat obscure, though they are supposed to pass through two intermediate hosts before reaching the intestines, bile ducts, or lungs of man, where their presence causes disease symptoms.

Etiology: Nearly all flukes are supposed to reach the human subject through infected water.

Morbid Anatomy and Pathology: The presence of the flukes sets up an inflammation. They may be found in sections of the tissue. In severe cases the eggs are found in the discharges.

Symptoms: The *lung* flukes produce cough with hemoptysis. The onset is usually slow. As the disease progresses the cough and hemoptysis become more frequent. The patient emaciates. Physical exertion or excesses aggravate the condition.

The *liver* flukes cause cirrhosis of that viscus. At first there is loss of appetite and diarrhea. The liver becomes swollen and tender. As the disease progresses the patient wastes away. Ascites is common.

Diagnosis: The diagnosis of lung flukes may be made by finding the ova in the sputa. The diagnosis of liver flukes by finding the ova in the feces.

Prognosis: The prognosis is doubtful.

Treatment: There is no specific treatment. Build up the patient. If he lives long enough the flukes may die in situ.

BILHARZIASIS.

Synonyms: Bilharziosis Egyptian Hematuria. Endemic Hematuria.

Definition: Bilharziosis is disease caused by the blood fluke —Bilharz.

Historical Note: The original blood fluke description was by Bilharz in 1852. During recent years various other forms have been described principally by Japanese investigators.

Description: The Bilharz fluke is about 2-8 mm. in length. One form inhabits the urogenital system mainly, the other the rectum.

Etiology: Until recently it was supposed that infection came through drinking water. Now some observers claim that infection may take place directly through the skin.

Morbid Anatomy and Pathology: The ova are the cause of the symptoms produced. They bury themselves in the mucosa of the parts affected producing chronic inflammation.

Symptoms: As a rule, the onset is gradual. The urinary form shows itself by bloody urine. The hemorrhages are not severe but more or less constant. There is pain in the region of the bladder as in cystitis. In some cases the vagina and vulva are attacked.

The intestinal form shows itself as a bloody diarrhea.

Complications and Sequelæ: Vesical calculi and urinary fistula may result.

Diagnosis: The diagnosis is made by finding the ova in the discharges.

Prognosis: The prognosis, as a rule, is favorable. The disease runs two or more years and gradually subsides.

Treatment is unsatisfactory. The only thing to do is to build up the patient.

Prophylaxis consists in avoiding infected water.

HOOK WORM DISEASE.

Synonyms: Ankylostomiasis. Uncinariasis. Tunnel Anemia.

Definition: Hook worm disease is disease produced by the various forms of uncinariæ.

Historical Note: Hook worm disease has been known by other names for a long time. It was not until 1901 that it became of medical importance. In that year Stiles published his studies showing that the hook worm was very prevalent in the southern part of the United States among the poorer classes. The Rockefeller Sanitary Commission, in 1909, estimated that twenty millions of the inhabitants were infected.

Description: The hook worm is from a third to a half inch in length and of the diameter of a hair. Its habitat is the upper part of the small intestine. The head has two pairs of hook-shaped teeth. The eggs about 70 μ by 36 μ in size. They can be found in the feces.

Etiology: The main source of infection seems to be through the skin, usually through the feet. The worms may also be ingested. According to Looss they pass through the veins to the heart, thence to the lungs, bronchi, trachea, esophagus, and digestive organs.

Morbid Anatomy and Pathology: The worms are found chiefly in the jejunum. There may be ecchymoses of the mucous membrane where they are attached. The feces may be stained with blood. Besides their blood-sucking habits, it is supposed that they throw off a detrimental secretion.

Symptoms: The characteristic symptom of hook worm disease is a progressive anemia. This causes marked pallor and wasting. Children are apt to be undergrown and ill nourished. The victim is mentally dull and physically incapable. The vitality is sapped.

There may be gastric disturbance and diarrhea. The patient may have an abnormal appetite, and be a dirt eater.

The anemia is of the secondary type. The red cells may be as low as two millions, and the hemoglobin from 25 to 50 per cent. Eosinophilia is present.

At the site of infection, mostly between the toes, there is first a vesicular eruption which in a few days pustulates, and then dries up.

Diagnosis: The characteristic anemia with mental apathy and physical inactivity should arouse suspicion. The stools should be examined for the eggs.

Prognosis: The prognosis depends on the treatment.

Treatment: To prevent the disease improved sanitation where it is prevalent is necessary.

The individual should avoid going barefooted.

Thymol is said to be a specific. A purgative is given at night —Epsom salts is recommended by the United States Public Health Service. The next morning at six and again at eight, from seven to thirty grains of *thymol* in capsules are given, according to the age of the patient. At ten another purgative is taken. The treatment is repeated once a week until the patient is cured.

Oil and alcohol must be avoided during treatment.

FILARIASIS.

Synonym: Guinea Worm.

Definition: Filariasis is disease produced by filariæ.

Description: The filaria is a long slender worm, it may be as much as six feet long and less than a tenth inch thick.

There are other smaller filariæ.

Morbid Anatomy and Pathology: Filariæ travel through the tissues to the feet and legs, the head reaching just beneath the epidermis. At this point a bulla appears. When broken a small ulcer forms with a small opening in the center. If water is poured on this the uterus which is the full length of the worm empties itself. When empty the worm leaves the part through this opening.

One form of filaria is found only at night.

They may lodge in the urethra.

Apropos of this, in Hakluyt's English Voyages, appears an account of a trip made by one Anthony Jenkinson from Mosco to Boghar in 1558. In speaking of Boghar, Jenkinson says: "There is a little river running through the middest of the said Citie, but the water thereof is most unholsome, for it breedeth sometimes in men that drinke thereof, and especially in them that be not there borne, a worme of an ell long, which lyeth commonly in the legge betwixt the flesh and the skinne, and is pluckt out about the ancle with great art and cunning, the Surgeons being much practised therein, and if shee breake in plucking out, the partie dieth, and every day she commeth out about an inch, which is rolled up, and so worketh till she be all out."

Etiology: The mosquito is an intermediate host. The embryos are found only during the night.

Symptoms: There may be none. In some cases they produce elephantiasis, in others chyle like urine.

Diagnosis: The diagnosis can be made in suspected cases by causing the worm to empty itself by applying water.

Treatment: There seems to be no form of treatment.

Treatment: Treatment consists in causing the worm to empty itself by applying water, and by carefully rolling the worm on a stick as it comes out. The whole process takes several days.

SECTION IV.

Constitutional Diseases

ARTHRITIS DEFORMANS.

(Greek, *αρθρον*, a joint.)

Synonyms: Rheumatoid Arthritis. Rheumatic Gout.

Definition: Arthritis deformans is a chronic disease of the joints characterized by changes in the cartilages and synovial membranes, peri-articular formation of bone and great deformity.

Historical Note: There are several diseases that are somewhat alike, clinically, but which are now considered to be different pathologically. The remains found in the tombs of ancient Egypt antedating 1300 B. C. show that arthritis deformans existed even then. In 1803 Heberden distinguished between the various gouty and rheumatic conditions. Virchow in 1859 called this particular disease arthritis deformans.

Etiology: Arthritis deformans has been variously ascribed to infection and to changes in metabolism. In 1913 Fuller presented a series of cases occurring in men that he had cured by operating on the seminal vesicles. His theory was that the arthritic condition was caused by a focus of chronic inflammation, probably gonorrheal, in the seminal vesicles. Operation removed the focus and the patients recovered. It is probable that other germs elsewhere in the body may cause the trouble.

Heredity seems to play a part. Exposure to cold and damp and mental strain are supposed to be predisposing factors.

Men and women are both affected. Some observers report a majority of one sex, some a majority of the other. I have seen more women than men sick with it. Most of my patients have been middle aged.

Morbid Anatomy and Pathology: The changes normally originate in the articular cartilages. There is fibrillation, the articular

cartilages are worn away, and finally the ends of the bones become affected. There may be a proliferation of cartilage and periosteal cells causing a true ankylosis. Sometimes there is atrophy of the ends of the bones.

Mild cases may show nothing but Heberden's nodes—swellings of the finger and toe joints. These cases are most often in women. In severe cases there may be an almost complete ankylosis of all the joints. These are usually associated with tremendous swelling of the knees and feet, and with general muscular atrophy.

Arthritis deformans does not have the uratic deposits in the joints that gout has. Chronic rheumatism does not have the structural changes in the cartilages.

Symptoms: The earliest symptom of arthritis deformans is the development of Heberden's nodes. These are slight swellings on the ends of the distal phalanges of the fingers. Sometimes they appear on the toes also. At times they will be quite tender and painful. At other times they cause no discomfort. They seem to be more swollen and prominent when tender.

As the disease progresses the swellings cause more and more deformity of the joints and the fingers become stiff and deflected to the ulnar side.

In other cases the onset is not unlike that of rheumatic fever, except that arthritis deformans usually begins in the small joints and rheumatism in the large. Most of the cases have acute exacerbations, when the affected joints become tender, there is fever and increased frequency of pulse.

If unchecked one joint after another becomes affected, including those of the jaw and spinal column. The arms and legs become permanently flexed, the fingers and toes deflected to one side, and in extreme cases the patient becomes like a statue, immovable, and unable to move any part of himself. The feet and knees become tremendously swollen, trophic changes occur, and the skin may break down and ulcerate. I have had several such under my care at the Metropolitan Hospital.

These extreme cases always present general muscular atrophy and great emaciation. There is also profound anemia. The mind remains clear.

Mild cases may go on for years without much deformity except of the hands. Others may progress to helplessness in a few months.

Still's disease: G. F. Still has described a form of chronic arthritis in children. He says: "The disease may be defined as a chronic progressive enlargement of the joints, associated with enlargement of the lymphatic glands and spleen." It occurs more often in girls, and usually before the second dentition.

Complications and Sequelæ: In arthritis deformans the organs of the body are not affected. The disease is limited almost entirely to the condition of the joints and the associated muscular atrophy.

Diagnosis: Gout begins in the toes, and is often limited to them. Men are much more subject to it than women. The reverse is true of arthritis deformans, and the fingers are usually affected first.

Chronic rheumatism attacks the larger joints first and rarely if ever attacks the spine.

Tuberculosis usually limits itself to a certain joint and is not general like arthritis deformans.

The x-ray will show the peculiar joint changes in arthritis deformans.

Prognosis: The prognosis as regards life is good. As regards cure it is not good. If a focus of infection can be discovered and removed, the outlook is favorable. Most cases are very chronic. The patients die of some intercurrent disease.

Treatment: The patient should be put under the best of hygienic surroundings. The diet must be full and nourishing. A change of climate to a warm and equable temperature may be advantageous.

Massage, electricity, baths, hot air treatments, may all be of help. Rest is essential.

If a focus of infection can be found it should be removed. It is well to bear in mind Fuller's theory, referred to above, that in men a chronic inflammation of the seminal vesicles may be the source of the trouble, and operation may relieve it.

Homœopathic remedies may do much, especially in the mild cases.

Belladonna acts speedily in cases where the finger joints become swollen and painful. It is almost specific.

Bryonia may sometimes serve in cases that cannot stand the slightest touch to the affected joint or the slightest motion.

Arbutus andrachne is said to be useful for arthritis of the larger joints.

Sulphur terebinthinatum is another remedy that may be tried.

If the case has progressed to ankylosis remedies can do little except to ameliorate any acute symptoms that may arise.

CHRONIC ARTICULAR RHEUMATISM.

Definition: A chronic affection of the joints characterized by thickening of the fibrous parts of the joints and wasting of the neighboring muscles from disuse.

Etiology: Rarely cases result from acute rheumatism. In the vast majority of cases, however, it is chronic from the start. It comes on late in life in those exposed to cold and damp.

Morbid Anatomy and Pathology: The fibrous elements of the joints are much affected, interfering with motion. The joints are sometimes enlarged. The neighboring muscles waste away and there may be some neuritis.

Symptoms: There is stiffness and pain in the joints, usually a number of them. The pain and stiffness are aggravated in damp weather. They are apt to become swollen and painful then. With rest the joints become more stiff. On beginning to move about they are apt to be more painful. With continued motion the joints limber up.

Osler says there is frequently an associated sclerosis of the valves of the heart.

The disease is very chronic.

Complications and Sequelæ: There may be some resulting deformity.

Diagnosis: The disease affects the larger joints.

Prognosis: Life is not shortened. The disease is very chronic.

Treatment: The general treatment is the same as that for arthritis deformans.

Rhus toxicodendron is an excellent remedy, better after moving about.

MUSCULAR RHEUMATISM.

Synonyms: Myositis. Myalgia. Myodynia.

Definition: Muscular rheumatism is the name applied to pain in the muscles and in the associated fascia and periosteum.

It is usually limited to one group of muscles. Strümpell speaks of a "polymyositis," described by some German writers, where the muscular system generally is affected.

The leading symptom is pain and it is not always possible to say whether the pain is in the muscles or in the nerves of the part affected.

Historical Note: Wilson, in Pepper's System of Medicine, considers the term muscular rheumatism to be a misnomer because the condition is not rheumatic. He says Inman, of Liverpool, is responsible for the term "myalgia" and that that is much more accurate than the other terms. On account of the character of the pains, the aggravation by motion, the soreness, it seems to me as though muscular rheumatism is a good term to keep. Many times it is impossible to say whether in a particular case the soreness is really in the muscles or nerves.

Etiology: Exposure to cold and wet is the most frequent exciting cause. Men, on account of their occupations, are more subject to it than women. It frequently follows exposure to a draught. It may follow sudden chilling after exertion. Persons with a rheumatic or gouty tendency seem to be more prone to it. One attack predisposes to another.

Symptoms: Pain of the affected part is the leading symptom. It may commence as a slight stiffness, augmenting for several hours, or it may begin quite suddenly and be severe from the beginning. The affected muscles become "set." Any attempt at moving them is extremely painful and sometimes impossible on that account. They are tender to touch, as a rule, if not throughout their whole extent, at least in certain spots. The trouble lasts from a few hours to a day or two. In exceptional cases it may become chronic.

The pain may be an ache, or it may be sharp and severe. The slightest motion makes it worse. Even a jar of the body aggravates it. A patient had to take a thirty-five mile railroad

journey once when suffering from torticollis and thought the trip would kill him.

The disease takes different names according to the muscles affected. The forms I have met with most frequently are:—

Torticollis, or wry neck, affects the sterno-cleido-mastoid muscles. The head is turned to one side and held in that position.

Lumbago, is muscular rheumatism of the muscles in the lumbar region. The patient may be unable to move. I had one patient who was seized with an attack in a hotel, while visiting New York. She was gotten to a chair where she sat tilted against the wall from six at night till noon the next day because she could not move.

Omalgia, rheumatism of the deltoid is another common form. It is impossible to move the arm at the shoulder joint.

Pleurodynia, pain in the side of the chest, aggravated by moving, deep breathing and coughing, is another form.

Other muscles or groups of muscles may be attacked.

Diagnosis: The diagnosis, as a rule, is not difficult. In lumbago and pleurodynia careful physical examination will show the pain to be located in the external muscles and not in the internal organs.

Prognosis: The prognosis is favorable.

Treatment: Persons who know themselves to be subject to muscular rheumatism should avoid draughts or sudden chilling of the body when heated. They should accustom themselves to cold baths.

Moderation in eating and drinking is essential.

For the attack massage is sometimes of service. Electricity in various forms may help. The vibrator will sometimes give relief. Hot applications are frequently beneficial. Strapping the chest in pleurodynia gives relief. Strümpell recommends the salicylates.

Cimicifuga is the remedy par excellence for muscular rheumatism. It is of special service in torticollis.

Sanguinaria is indicated if the trouble is in the deltoid. It is said to be indicated for the right side particularly. It has cured attacks in the left deltoid in some of my cases.

Bryonia may be indicated in cases where the pain is mostly in the back.

Ranunculus is indicated in pleurodynia.

Rhus toxicodendron is indicated if the pain is low in the back and extends down the thighs and legs. Worse at night and lying down.

Colchicum is indicated if the pain is in the back, shoulders and abdomen.

GOUT.

(Latin, *gutta,* a drop, a dropping of morbid matter from the blood into the joints.)

Synonym: Podagra.

Definition: Gout is supposed to be a disorder of metabolism. It is characterized by attacks of acute arthritis and by various constitutional symptoms. There are deposits of urate of soda in the joints.

A great many vague symptoms that people have are accounted for by what is called the gouty diathesis or lithemia.

Historical Note: This disease was written about by the old Greek writers. Our modern ideas date from the time of Sydenham who himself suffered from it and wrote about it in 1683. Wollaston in 1797 showed the deposits in various parts of the body to be uric acid. Garrod in 1848 showed that there was an excess of uric acid in the blood.

Etiology: Purin bodies formed during digestion are the source of uric acid. If they are not properly eliminated uric acid accumulates in the blood and gout results.

Improper food and drink is supposed to be the main cause of gout. Red meats and malt liquors are said to be the chief offenders.

Chronic lead poisoning is another important cause of gout.

Any age may be attacked, but the vast majority of cases occur in those at the prime of life. Men are more often affected than women. In those subject to it an acute attack may be brought on by a dietetic or alcoholic excess.

Heredity seems to play a part.

Morbid Anatomy and Pathology: In acute cases the affected joints are inflamed, swollen and red. The first joint of the big toe is most often involved. Then the ankles, knees and joints of the hands and wrists in that order.

In chronic cases chalky deposits are found. These may go on till the joint becomes immobile. Sometimes ulceration occurs.

The blood is said to contain an excess of uric acid.

Associated lesions often found are chronic interstitial nephritis and arterio-sclerosis.

Symptoms: The attack of *acute gout* usually comes on suddenly. The patient wakes with the affected joint—most often the great toe—swollen, hot, tender and red. The pain is exquisite. Any jarring or moving adds to the pain. There may be chilliness, there always is more or less fever, up to 102° F. or higher. After a few hours the pain recedes, and during the day the patient feels comparatively comfortable. The pain may return the next night.

The above symptoms are often preceded by restlessness, insomnia and some indigestion. During the attack the digestive disturbance is more marked. There is loss of appetite, the tongue is coated, and the patient is troubled with nausea.

The urine is scanty, high colored, and of high specific gravity. The bladder is irritated and there is burning on micturition.

The acute attack lasts a week or ten days. After that there may be an interval of many months before a second acute attack. The tendency is for recurring attacks at lessened intervals and of diminishing severity until the condition becomes more or less chronic, with permanent changes and deformities in the joints.

At first one great toe is affected. As the disease tends to chronicity other joints become involved, smaller ones first, then large ones, till many are affected.

Retrocedent or suppressed gout or misplaced gout: In these cases the acute symptoms referable to the joint may suddenly cease and the paroxysm vent itself on some internal organ. There may be severe gastro-intestinal symptoms. There may be severe cerebral symptoms, even delirium or apoplexy. There may be heart symptoms, palpitation, angina pectoris or an acute pericarditis.

Chronic Gout: When gout becomes chronic various joints become permanently affected, beginning with the feet. There are deposits of urate of soda called tophi. These deposits may take place on the cartilages and in the tendons. The affected joints are swollen and deformed. The tophi may also appear in the ears. The skin over them is apt to be thin and shining, and the veins stand out. They may break down and ulcerate.

Lithemia; Uricacidemia; the Lithic Acid Diathesis: This is a chronic condition, a mild form of chronic gout which received a great deal of consideration a few years ago following the studies of Haig. It is caused by an excess of uric acid in the blood.

Perhaps the most frequent exhibition of lithemia is a disturbed digestion. The breath is offensive. The patient is troubled with acid eructations. He is inclined to be constipated.

Then comes palpitation, later possibly arterio-sclerosis or even angina pectoris. There may be hypertrophy of the heart.

The patient is depressed, or he is irritable and morose. He has indefinite sort of headaches. He may become excessively nervous.

The skin is apt to be dry and scaly. The perspiration is offensive. Sometimes the patient has eczema.

The urine is liable to be high colored and of high specific gravity. There is a red sand-like deposit. There may even be gravel and stone in the kidney.

Complications and Sequelæ: The complications and sequelæ of gout have already been mentioned. There is a tendency to the formation of deformed joints in one class of cases. There may be arterio-sclerosis resulting in apoplexy. There may be chronic interstitial nephritis.

Diagnosis: The lithic acid diathesis can be diagnosed only by eliminating all other causes for the various symptoms produced.

In the acute type of the disease, which is comparatively rare in this country, the recurring attacks in the great toe are diagnostic. I have had such cases. In chronic cases with joint deformity the history of the case is essential.

Prognosis: The prognosis for an acute attack of gout is good. Recurrences may be expected. When the disease becomes chronic

the prognosis depends altogether on the symptoms presented by each individual case.

Treatment: The gouty subject should be particular about his diet. In a general way meat should be partaken of sparingly. Milk and cheese may be used to make up any deficiency in the proteids. Alcohol is best omitted from the diet altogether. Coffee should be stopped. Sweets are detrimental.

Fruits and vegetables are permissible, fresh fish and plenty of water.

Individual idiosyncrasies must be studied, and any particular thing that disagrees with a particular patient should be omitted from the dietary.

The special diets prescribed by various authors differ exceedingly in detail. For example, Robin says the patient may eat lean, red meats. He must not eat liver, kidneys, or other internal organs that are used for food. He curtails the use of fats and sugars.

Haig, who has made an exhaustive study of gout on his own person permits the use of bread, toast, biscuits, puddings, milk, cheese, butter and oil. He allows the patient to use all fruits and vegetables excluding peas, beans, lentils, mushrooms and asparagus.

Eggs are allowed and forbidden according to the author.

The gouty patient should live quietly if possible, avoiding great physical and mental exertion. A dry, warm climate is beneficial. Baths and massage and mild exercise are good.

The patient should drink plenty of water. The skin and bowels should be kept in good condition.

Lithiated hydrangea is an excellent remedy in lithemic conditions.

The homœopathic remedy is of great assistance.

Belladonna may be used in acute cases with the hot, swollen, inflamed joint.

Colchicum is indicated in cases with considerable prostration, with the nightly aggravation.

Glonoin has served me in headaches due to a gouty condition.

Calcarea carbonica I believe has a corrective effect on faulty

metabolism, and I use it after acute symptoms have subsided, and in lithemic cases.

Cajuputum is recommended for retrocedent gout.

Many other remedies may be called for.

DIABETES MELLITUS.

(Greek, διαβητης, to pass through; μελί, honey.)

Definition: Diabetes is a nutritional disease characterized by polyuria, sugar in the urine, and loss of flesh and strength.

Historical Note: According to the Papyrus Ebers the ancient Egyptians recognized a disease characterized by polyuria and thirst. Galen about 150, wrote about such a disease. The name "Diabetes" was given by Aretaeus in 150 A. D. The Ayur Veda, written about 500, shows that the Hindus knew the urine in this condition contained sugar. Cingalese writings of the fifteenth century speak of "honey urine." Willis in 1674 said the urine had a sweetish taste. Dobson in 1776 showed this was due to sugar. In 1797 Rollo gave the first good account of diabetes. In 1889-1890, Minkowski and von Mering, experimenting on dogs, found that diabetes developed after complete extirpation of the pancreas. In 1900 Opie declared the pathological lesion in diabetes to be destruction of the islands of Langerhans in the pancreas.

Etiology: Jews and the natives of India and Ceylon are especially susceptible to diabetes. It is more prevalent among the better classes.

Diabetes affects men and women in the proportion of three to two. It usually makes its appearance from the third to the sixth decade.

Heredity is supposed to play a part. Cases of diabetes occur in different members of the same family, or they occur in families with a nervous taint, with insanity, tuberculosis or gout as a family trait.

Diabetes is also said to be caused by over-indulgence in starches and sugars, by the excessive use of beer, and to occur in those who suffer from obesity.

The emotions may cause diabetes. Sometimes it comes from injuries to the head.

Diabetes mellitus may also follow the acute infections, pregnancy, or diabetes insipidus.

Disease of the liver or of the medulla may lead to it.

Morbid Anatomy and Pathology: Examination of a large number of diabetics shows that the islands of Langerhans in the pancreas are diseased or destroyed.

Many cases also show tuberculosis in the lungs.

The heart is often large and is associated with arterio-sclerosis. The blood shows increased specific gravity and contains sugar up to 0.6 per cent. The red cells may run up to 6,800,000; there is usually a mild leucocytosis.

The liver is large and fatty. The kidneys are large and show degeneration of the loops of Henle.

Emaciation is the rule in fatal cases.

Symptoms: Diabetes mellitus is glycosuria plus excessive thirst, polyuria and failure of nutrition. It has been variously classified as acute or chronic, severe or mild.

The acute form is usually severe. It occurs most often in young people, and even in children. The chronic form is milder in its symptoms. It occurs in middle life, or, later, in those who are or have been stout. If the sugar disappears under diet the case is mild.

In any case the onset is insidious. The patient is troubled by an excess of urine, which gets him up nights to void it. He is always thirsty, and usually has a large appetite notwithstanding which he loses weight and grows progressively weaker. The patient tires easily. Then he shows an indisposition to mental or physical effort.

The skin becomes harsh and dry. There is often pruritus, sometimes boils appear. The hair becomes thin and dry, the nails brittle.

The lips are dry. The teeth decay. The breath has a sweetish odor. There is often nausea. Constipation is the rule.

The patient is depressed. The sexual appetite is impaired or lost. Men become impotent. Neuralgias appear.

Impaired vision may be an early symptom.

The temperature is subnormal. The pulse is faster than normal. Faught says the blood pressure is low unless the case is complicated by nephritis or arterio-sclerosis.

The most common symptom is polyuria. The diabetic passes from two or three to eight and even ten quarts of urine a day, and the urine contains sugar. With this excess of urine there may be pain in the lumbar region. The urine may have a sweetish odor. It is said to have a sweetish taste. The specific gravity of the urine is high, it may vary from 1025 to 1050. Very rarely a case will appear where either the quantity of urine is not excessive or the specific gravity is not high. I had a man under treatment in 1914 whose urine was of high specific gravity, but normal in quantity. Sugar is present and averages from 3 to 10 per cent. Cases have been reported where the sugar ran as high as 20 per cent.

There is an increase in the urea and in the ammonia. Acetone and uric acid are found. If nephritis exists albumin is also found.

When the carbohydrates—starches and sugars—are excluded from the diet of a diabetic, the sugar in the urine diminishes or entirely disappears. Most patients can oxidize a certain amount of carbohydrates, so that the point of tolerance must be determined for each individual case. In severe cases sugar may be formed from the albumin of the food, and it may be impossible to stop its appearance in the urine.

Emotional excitement increases the amount of sugar. The acute fevers and physical exertion diminish it.

The sugar may disappear just before death.

There may be temporary glycosuria in persons who have not got diabetes.

Complications and Sequelæ: The skin becomes harsh and dry, and there is usually little perspiration. Pruritus is common, especially of the genitals. The patient is subject to boils, he may have a carbuncle. The hair and nails may come out. Sometimes there is gangrene of one or more toes. This latter condition is associated with arterio-sclerosis. The feet may become edematous, or there may be perforating ulcer of the foot.

There may be cardiac weakness. Arterio-sclerosis is common.

Tuberculosis and diabetes are said to be often associated. I have discovered very few cases of diabetes in my wards at the Metropolitan Hospital. There is occasionally pneumonia followed by gangrene of the lung.

There may be gingivitis or pyorrhea. Owing to the large appetite and heavy eating the stomach becomes dilated. The patient also has a great thirst. Constipation is the rule, although sometimes diarrhea is met with. The stools are apt to contain fat.

The diabetic woman tends to be sterile. If she does conceive she is liable to abort. The diabetic man may become impotent.

In the eyes there is failure of accommodation. Late in the disease there is a tendency to cataract.

In the nervous system there may be headache, or the patient may be dull. Depression is common, and I have known patients to commit suicide. Neuritis and neuralgias appear. There may be paraplegia or hemiplegia. Or there may be a pseudo-tabes.

Coma is frequent at the end. It may appear suddenly, after muscular or emotional effort, or it may follow a delirium.

The lymph glands may be swollen.

Diagnosis: Diabetes mellitus must be differentiated from transient glycosuria by the course of the disease. True diabetes adds thirst, polyuria and wasting of tissues to glycosuria.

Strümpell says the early symptoms of diabetes mellitus are: 1. Languor and debility. 2. Furunculosis. 3. Pruritus pudendæ in women, balanitis in men. 4. Cataract. 5. Sciatica, especially if bilateral. 6. Impotence.

One should be suspicious of diabetes if a patient is fat and nervous, or if he is elderly and has arterio-sclerosis.

If in doubt about the urine the patient may be given a meal rich in starch. If no sugar appears in the urine the case is not diabetes.

Prognosis: The prognosis in diabetes mellitus is not good, it almost always ends fatally. A few cases die in a few weeks or months, others last for years, ten, twenty or more. The patient may be apparently cured but the disease nearly always breaks out again.

Treatment: The diabetic must be put in the best possible hygienic surroundings. Mild exercise, out of doors and massage are indicated. Baths may be given, cold sponges or douches.

Climatic treatment is of value, a low altitude with a mild climate is the best. Certain places in southern California enjoy a favorable reputation for prolonging the lives of diabetics.

For the annoying pruritus a wash of a saturated solution of borax may give relief.

For the intolerable thirst the patient may use lemon juice, cracked ice or may sip hot water.

In cases of coma large enemas of bicarbonate of soda, a pint to the ounce, have sometimes been successfully used.

Camphor or ether may be used as a stimulant.

Diet is our main standby in diabetes. The carbohydrates must be cut down as much as possible because their free ingestion adds to the glycosuria. Each patient is a law unto himself in this, some can take more than others without harm. Allow as much carbohydrates as your patient will stand, because some may be necessary to keep up his general condition.

Authorities differ in the details of their lists. Saunby recommends:

Breakfast, crust of fresh roll with butter, fat bacon, eggs, tea or coffee with cream but no sugar.

Lunch, meat, green vegetables, cheese, crust of bread and butter, claret or whiskey diluted if necessary.

Tea, diabetic bread, tea with cream.

Dinner, bread, meat soup, cooked meat, green vegetables, cheese, butter, claret or whiskey, coffee.

Keep this diet up for a time, then add milk. Or, as a trial, potatoes may be given.

Levulose powder or saccharin may be used as a sweetener.

Raw apples may be eaten.

Ralfe says to avoid milk, liver, mollusks, all kinds of bread, toast, farinaceous vegetables, as potatoes, artichokes, rice, oatmeal, corn flour, sago, tapioca, arrowroot, turnips, carrots, parsnips, peas, beans, beets, asparagus, tomatoes, celery, sea kale, endive, radishes, lettuce, fruits, jams, syrups, sugars, sweet pickles, cocoa, chocolates, liqueurs, sweet wines.

Ralfe allows meat, fish, poultry, game, bacon, ham, eggs, gluten or bran bread, green vegetables, cabbage, turnip tops, water cresses, mustard, sauer kraut, green part of lettuce, sorrel, mushrooms, cheese, nuts, except chestnuts.

Von Noorden recommends an oatmeal diet. He uses 250 grams of oatmeal, an equal quantity of butter, and the whites of six eggs. The oatmeal must be thoroughly cooked and the butter and eggs mixed with it. The mass is divided into four meals.

The meats are restricted by Sajous and others.

Strümpell recommends opium or laudanum as a remedy. He says it lessens the annoying thirst. It sometimes causes decided diminution in the amount of urine and sugar excreted. It is further indicated when there is general restlessness or sleeplessness. He gives four to eight grains of opium in the twenty-four hours. In severe cases he sometimes uses laudanum, from sixty to one hundred drops divided into three doses daily.

Free use of the alkaline mineral waters is also of value.

Halbert speaks favorably of the use of trypsogen, a preparation prepared by G. W. Carnrick Co. It is put up in tablet form. Each tablet contains the sugar-oxidizing enzymes of the islands of Langerhans (from calves and lambs), with trypsin, ptyalin, amylopsin, combined, 5 grains; bromide of gold, 1-100 grain; bromide of arsenic, 1-200 grain. The usual dose is two tablets immediately after each meal. This may be increased from time to time up to six or eight tablets at a dose till there is dizziness or diarrhea, then stopped. It may be renewed after a day or two with the original dosage.

Halbert also recommends as a homœopathic remedy *syzygium* IX. It causes disappearance of sugar in the urine.

Other homœopathic remedies are:

Uranium nitricum is indicated in typical cases of diabetes with polyuria, glycosuria, excessive thirst. It is also useful in impotency.

Argentum metallicum, urine sweetish and profuse. Feet edematous. Sexual weakness. Better open air.

Phosphoric acid, indifferent, sad, profuse urine with sugar,

pain in back and kidneys. I have used this remedy with satisfaction.

Terebinth, inability to concentrate, glycosuria, eructations.

These are the principal ones, although many others may be indicated in individual cases.

DIABETES INSIPIDUS.

Definition: Diabetes insipidus is a chronic affection characterized by the passage of large quantities of normal urine of low specific gravity.

Historical Note: In 1674 Willis distinguished between the two forms of diabetes.

Etiology: The disease is rare. It most often occurs in young men. Drinking large quantities of any liquid may originate it. Occasionally the infectious fevers are followed by it. Heredity seems to play a part sometimes.

The emotions may originate a case.

There is a certain spot in the fourth ventricle irritation of which has experimentally caused diabetes insipidus. Tumors, cerebral syphilis and hemorrhage in that region have caused it. Basilar meningitis may cause it.

Diabetes insipidus may precede or follow diabetes mellitus.

Morbid Anatomy and Pathology: There are no special pathological lesions in diabetes insipidus. Sometimes the kidneys are found to be large and hyperemic, and the bladder dilated and with thickened walls.

Symptoms: The leading symptom of diabetes insipidus is the great quantity of urine passed each twenty-four hours, it may be as much as ten quarts. It is pale in color, may be turbid, of faintly acid or neutral reaction, and of low specific gravity. The solids are relatively low, but actually increased for the twenty-four hours. There is no sugar.

The patient is very thirsty, intensely so. The skin and mucous membranes are dry. The digestion is good at first but becomes impaired. There is apt to be alternating diarrhea and constipation. Late there is wasting.

There is insomnia. There may be headaches and pain in the lumbar region.

There may be pruritus and impotency.

The temperature is subnormal. If fever appears the output of urine is decreased.

Complications and Sequelæ: Diabetes insipidus sometimes runs into diabetes mellitus.

Diagnosis: Diabetes insipidus must not be mistaken for chronic interstitial nephritis. The urine of the latter is increased in quantity and of low specific gravity, but casts can usually be found. There are also other systemic conditions that are found in nephritis.

The absence of sugar in the urine differentiates diabetes insipidus from diabetes mellitus.

Treatment: If the underlying cause can be found it must be treated.

The intake of liquids should be reduced.

Ergot and nitroglycerine have been recommended.

Strümpell advises valerian, one to one and a half drams daily in powder or infusion.

The homœopathic remedies useful in diabetes mellitus may also be useful in this form. Raue says each case will have to be individualized, and that the indicated remedy may not have "profuse urination and insatiable thirst" in a marked degree.

RICKETS.

(Greek, ῥαχις, spine. Rickets is from Old English, *wrikken*, to twist awry.)

Synonym: Rhachitis.

Definition: Rickets is a constitutional disease of early childhood characterized by defective nutrition of the bones.

Historical Note: Glisson was the first to describe the disease in 1650. He suggested the name rhachitis.

Etiology: Rickets occurs most often in poorly nourished children, living in poverty and under bad hygienic conditions. It occurs also in those infants artificially fed on a faulty diet, one deficient in fats and proteids.

The disease appears early in life and is at its worst at the age of two to three years.

Morbid Anatomy and Pathology: The change in the nutrition of the bones is the most important thing. There is delay in ossification, atrophy and hyperplasia (Cheadle).

The periosteum is excessively vascular and cannot be removed from the bone without taking particles of bone with it. The bones are soft, due to a deficiency of lime, and can be cut with a knife. Increased absorption of the bones takes place inside.

The two layers separating the epiphyseal cartilage from the shaft of the bone, the proliferative and the ossific, instead of being distinct, become merged into one another.

The bones while soft become more or less deformed, due to pressure—bow-legs, etc. When ossification finally takes place the bones are large and heavy and remain permanently deformed.

The ligaments and muscles are also soft and flabby. The patients are subject to catarrh. The teeth are late in appearing. The child is backward physically.

Sometimes the liver and spleen are enlarged.

Symptoms: In a case of well marked rickets the head is large and square in shape. The frontal prominences are marked. The bones of the head themselves may be soft and the fontanelles not as small or closed as they should be. The sternum is unusually prominent, while there is a series of prominences at the junction of the ribs and cartilages—the so-called "Rhachitic rosary." If the child has sat up much there may be kyphosis. If it has tried to walk there is either knock-knee or more often bow-leg. The ends of the long bones are thickened, particularly the distal ends of the radius, tibia and less often of the femur. In well marked cases the thorax is narrowed laterally and the pelvis becomes deformed.

The child may appear well nourished, fat and flabby. More often it is inclined to be rather thin, but with a protuberant abdomen.

These changes take place slowly. They are preceded by restlessness, a general soreness, wanting to be left alone, and some slight fever. With these there is more or less digestive disturbance. The child becomes weak and irritable. There is a tendency to excessive sweating, particularly about the head while asleep.

He does not learn to walk early and is generally backward. The teeth are late in appearing, and when they do come are poor in quality and tend to early decay.

Osler lays stress on the presence of a systolic murmur that may be heard over the anterior fontanelle or the temporal region.

A tendency to convulsions is likewise put down as a symptom of rickets. Laryngismus and tetany are also included.

Catarrhal conditions of the mucous membranes are common.

Anemia is the rule.

Complications and Sequelæ: Besides the nervous and other symptoms mentioned, the rhachitic patient is subject to bronchitis and broncho-pneumonia or to gastro-intestinal catarrhs.

Green stick fractures and complete fractures easily result from slight trauma.

Diagnosis: The typical case is easy of diagnosis. In hydrocephalus the child does not hold the head up, and is mentally deficient. Rickets is differentiated from paralysis; though he does not walk he can use his legs.

Prognosis: The disease is chronic. It is not fatal except through its complications, respiratory, convulsions, spasm of larynx, though it does leave various deformities and many patients are dwarfed. When the disease comes to an end the fontanelles close and the patient begins to walk.

Contraction of the pelvis in women may cause trouble later in case of child birth.

Treatment: To avoid development of rickets the infant should be fed at the breast.

After weaning avoid too much farinaceous food. Give milk, raw or modified. Add juice of raw beef and yolk of egg to the diet.

The patient should be frequently bathed. The clothing should be adjusted to the temperature. The child should be kept out of doors as much as possible.

Standing or walking or even sitting should not be permitted while the bones are soft.

Cod liver oil is of great value in these cases. It should be given in small doses, one teaspoonful once or twice a day, after meals.

As a remedy Jacobi recommends *phosphorus* in the form of elixir phosphori, six to ten drops three times a day.

There are many homœopathic remedies that may be of value.

Calcarea carbonica is useful in children who are late in cutting their teeth. They do not learn to walk. The fontanelles delay in closing. They may be fat but they are backward.

Calcarea phosphorica, the bones break easily and reunite slowly. The condyles may be swollen. The child cannot hold up its head.

Kali phosphoricum is another remedy that may be useful when atrophy is present.

Silicea has profuse sweat of the head, the body dry.

OSTEOMALACEA.

(Greek, ὀστεον, bone; μαλακος, soft.)

Synonym: Mollities Ossium.

Definition: Osteomalacea is a gradual softening and bending of the bones.

Historical Note: An Arabian physician, Gschusius, who wrote in the sixth century, is the first to write of osteomalacea so far as known. Rickets and osteomalacea were considered one until the work of Labstein about 1850.

Etiology: Osteomalacea occurs more often in women than in men. The great majority of cases occur in adult women during pregnancy—less often in women who have never borne children. Only rarely does it occur in men. The cause is unknown.

It is very common along the Rhine and in Westphalia, in eastern Flanders and northern Italy.

Morbid Anatomy and Pathology: The essential change is the disappearance of the lime salts, leaving the bones soft, similar to the change that takes place when a bone is treated with hydrochloric acid. The bones can be bent or dented with the fingers. The process takes place from within outward. The marrow is at first hyperemic, with exudations of blood. Later it becomes yellow and degenerates. The process goes on until the cortex is a mere shell. The periosteum when removed reveals a rough and uneven surface.

Symptoms: The disease is of slow development. The first thing noticed is pain in the sacrum, the nape of the neck or the back and thighs. The parts are tender. The patient is weak. Then a difficulty in walking is experienced. Strümpell says, "The gait is either uncertain and slow or characterized by short, painful steps, the lower limb and pelvis being jerked forward as in one piece." Eventually walking becomes impossible and the patient bedridden.

A curvature of some of the bones is noticed, the natural curves of the spinal column become more marked and the patient is shorter in stature. The chest and the pelvis become compressed, laterally, the sternum and symphysis being pushed forward. The promontory of the sacrum is pushed down and forward also. The bones of the extremities are also sometimes affected, and there may be fracture. The bones of the head and face are rarely attacked.

The internal organs are unaffected except by pressure. In consequence of compression of the chest pulmonary congestion is frequent, and catarrhal pneumonia may kill the patient.

Complications and Sequelæ: The deformed pelvis interferes with parturition. The deformed chest may cause dyspnea.

Diagnosis: A well marked case is easily diagnosed. Early in the disease there may be paralysis, and the symptoms may be ascribed to disease of the cord or to hysteria.

Prognosis: An occasional case gets well. Others will develop symptoms during successive pregnancies and remain at a standstill between. The majority run a course of two or three years, and die either from general debility or from some lung complication.

Treatment: The patient should be placed under the best possible hygienic surroundings.

Pregnancy should be avoided.

Ovariotomy has been followed by cure and should be thought of.

Strümpell recommends *phosphorus* in small doses.

There is no homœopathic cure of this condition reported so far as I know.

Calcarea carbonica, with its symptoms of malnutrition, might be of help.

Calcarea phosphorica, phosphorus, silicea or other remedies mentioned under rickets might serve here also.

SCROFULA.

Synonyms: Tuberculosis of the Lymphatic System. Tuberculous Adenitis.

Definition: Scrofula is tuberculosis of the lymphatic system.

Historical Note: Scrofula was formerly considered a distinct disease. It is only since Koch discovered the tubercle bacillus that the true tuberculous nature of the disease has been known.

Etiology: Predisposing causes are catarrhal inflammation of mucous membranes, measles, whooping cough, enlarged tonsils.

Scrofula is more common in children, though it may be met with at all ages.

Morbid Anatomy and Pathology: The lymphatic glands are enlarged. (1) They may tend to heal. 2. They may tend to suppurate. Nearly always the pus is sterile. 3. The unhealed glands tend to become tubercular.

Symptoms: The scrofulous child is usually rather thin and with a clear white skin through which the veins show plainly. Or another type may be short and thick set with a coarse, dry and scaly or oily skin and thick nose.

In either class the glands of the neck or axilla may become enlarged. After staying this way for an indefinite time they may become smaller and finally disappear. In other cases they become so large as to be disfiguring; they are sometimes troublesome and have to be removed. Others break down and suppurate spontaneously.

Many of these patients mature early. But they seem to be very susceptible to disease, and if they are not carried off by some illness they age early.

Some of them develop pulmonary tuberculosis. Quite a number of patients in my service at the Metropolitan presented evidences of scrofula in the shape of scars from swollen glands.

The cervical glands alone may be affected in poorly nourished children. Submaxillary swelling appears first, larger on one side

than on the other. Then the glands on both sides of the clavicle and in the axilla become swollen, sometimes a few isolated glands may be all, sometimes a chain or group of them.

The bronchial and mediastinal glands may be affected. The swelling may cause symptoms of pressure on the great vessels or nerves. Sometimes the swollen glands break into the bronchial tubes, or they may break into certain organs that are nearby.

When the mesenteric glands are affected the patient becomes anemic, the belly is large, the extremities and muscular tissues waste away.

There may be tuberculosis also of the serous membranes occurring through the lymphatic system.

The pleuræ may be affected, very often coming on insidiously and presenting sooner or later physical signs of the various forms of pleurisy. This may be associated with pulmonary tuberculosis. I have found the two conditions often associated in my service at the Metropolitan Hospital, especially among the men.

Tuberculosis of the pleura and of the pericardium also often go together.

Tuberculosis of the peritoneum occurs most often as a complication of pulmonary tuberculosis. There may be a localized exudate, sometimes ascites, pigmented skin and enlarged mesenteric glands. The temperature may be sub-normal, or there may be fever. The condition of the patient may simulate typhoid.

In so-called scrofulous subjects the bones and joints may also become affected. The child is backward in physical development. There are apt to be deformities of various kinds. The hip is perhaps the joint most often affected with tuberculosis. This eventually causes a stiff joint, the thigh and leg on the affected side do not grow to the same size as the other. Next to the hip the spine seems to be most often affected, causing various deformed curvatures. These two classes of cases form the greater part of our orthopedic service at the Tuberculosis Infirmary of the Metropolitan Hospital.

Complications and Sequelæ: Pulmonary tuberculosis may appear later in the scrofulous subject. Premature old age is a common sequel. Coryza and eczema may be complications. Deformities result in bone cases.

Diagnosis: Tuberculosis of the lymphatic system is usually found only in children. As a rule, enlargement of the submaxillary gland of one side is the first indication. The gland tends to suppurate.

Prognosis: In tubercular adenitis the prognosis is doubtful. In many cases, after a few glands have suppurated and broken down, the patients regain their health. Others run into pulmonary tuberculosis.

If the serous membranes become involved the prognosis is bad.

Treatment: Tuberculous adenitis is not a surgical disease. One cannot remove all of the lymphatic glands. Of course, single glands that suppurate and break down will have to be treated surgically. Good results have also been claimed from opening the abdomen in tuberculous peritonitis.

The main thing for all of these cases is general hygienic treatment. The patients should spend most of the time in the open air. They should be well fed up. Rest is essential.

The lymphatic and bone diseases thrive particularly well at the Tuberculosis Infirmary of the Metropolitan Hospital. The results of the open air treatment there will compare favorably with those gotten elsewhere. After hearing the reports of the Sea Breeze Hospital for these cases at Coney Island, reports presented at a meeting of the National Association for the Study and Prevention of Tuberculosis, I suggested to the then Commissioner of Public Charities, the Hon. Robert W. Hebberd, the propriety of a children's ward at the Metropolitan. We are indebted to him for its establishment.

Cod liver oil is useful in this class of cases, a teaspoonful once or twice a day after meals.

Inunctions of iodoform sometimes seem to do good.

My own preference is for the indicated homœopathic remedy. Our materia medica is rich in this field. I will name only a few.

Calcarea iodide is useful in cases with enlarged abdomen, especially if the skin or mucous membranes show associated lesions.

Baryta carbonica, the glands are swollen and indurated, there is coryza and cough.

Calcarea phosphorica in spinal cases particularly. It aids nutrition of the bones.

Iodium in cases of lymphatic tuberculosis with great emaciation.

Calcarea carbonica is a great remedy in this condition as in all forms of malnutrition. Its pathogenesis includes nearly all possible scrofulous conditions.

Silicea is useful in swelling and suppuration of the glands. The patient eats well but is not well nourished.

MARASMUS.

Synonym: Infantile Atrophy.

Definition: Marasmus is literally wasting. As I use the term marasmus here I mean infantile atrophy without demonstrable disease.

Etiology: We may have wasting with tuberculosis, in congenital syphilis and in other conditions. In such cases marasmus is merely a symptom. The underlying disease must be treated.

We may have a true marasmus, or infantile atrophy, due to malnutrition, without any other underlying disease.

It occurs in infants and is due to improper feeding, or to bad hygienic care or to faulty metabolism. The child may suffer from over care as well as from under care. One bad case that came to me was a child two years old who had had nothing to eat except sterilized milk, and nothing to drink except boiled water. The parents were in good circumstances, but the mother suffered from bacteriophobia.

Morbid Anatomy and Pathology: Other than the atrophy there is no special pathological lesion.

Symptoms: The principal symptom is the emaciation. There is a general atrophy of the soft tissues. The skin becomes dry and wrinkled. The face looks old. The extremities are very thin. The abdomen is sunken.

The patient is apt to be cross and irritable. There may be vomiting or diarrhea. Sometimes the appetite is poor, sometimes voracious.

Temperature, pulse and respiration are normal, as a rule.

Complications and Sequelæ: Sometimes, not always, anemia is associated with marasmus.

Diagnosis: A diagnosis of marasmus can be made only after the elimination of underlying diseases.

If the wasting is due to starvation, correcting the diet will promptly cure it.

Rotch says it may be necessary to use the tuberculin reaction to differentiate simple marasmus from general tuberculosis.

Prognosis: The prognosis is always doubtful. Some cases recover after a time, others die.

Treatment: The most essential thing in treatment is to study the case and then correct the diet. Many forms of milk modification may have to be tried before the proper food is found. Personally, I have a preference for pure non-sterilized milk diluted with varying quantities of water according to the age of the infant. An infant at birth would require milk one part to water three or four parts. An infant a year old should be able to digest undiluted milk.

I have also found somatose dissolved in water an excellent food in some cases. Somatose, a teaspoonful to a cup of water. When first put together the somatose forms a sticky mass, but if allowed to stand for half an hour it finally becomes completely dissolved. Stirring will not hasten the process. This solution can be used alone in desperate cases. In others it can be added to the milk.

Inunctions of olive oil, or of cocoa butter, may be used.

The most valuable homœopathic remedy is *calcarea carbonica* given once or twice a day. The thirtieth to two hundredth potency is best. It should always be tried first. A case at the Flower Hospital in September, 1914, had been fed on various formulas for a month without benefit. Improvement began at once after giving *calcarea.*

Other remedies are of lesser importance although sometimes indicated.

OBESITY.

Synonyms: Polysarcia Adiposa. Lipomatosis Universalis.

Definition: An excessive deposit of fat in the tissues.

Etiology: Some patients seem to inherit a tendency to be fat and are always that way.

Other patients take on fat as they approach middle life.

The condition is probably due to faulty metabolism.

Morbid Anatomy and Pathology: There is an increase of fat

throughout the tissues of the body, not only an excess where fat appears normally, but there is an infiltration where fat does not ordinarily appear. In extreme cases there may be fatty degeneration of the heart with consequent weakening, and the train of evils which follow a weak heart.

Symptoms: A person may be very fat and yet otherwise be in good health. She may object to the fat because of the appearance.

When the internal organs become involved the symptoms may be many. We get shortness of breath on exertion due to a weak heart. There may be plethora, there may be anemia.

There may be profuse sweating under the breasts and in the groins. This causes irritation and intertrigo, sometimes eczema.

The sexual desire is often lessened. In women there may be uterine disturbance.

Complications and Sequelæ: Corpulent people appear to be more prone to apoplexy. Pulmonary congestion and anemia may result from the weak heart.

Diagnosis is self evident.

Prognosis: It is hard to say anything regarding prognosis, except that in a general way fat people do not seem to live to a great age.

Treatment: Many stout people are not heavy eaters, and yet diet seems to be the most satisfactory method of treatment.

There is one diet that I have not seen mentioned in the books, but which I have used successfully in several young and vigorous persons whose excretory organs were in good condition. That is an exclusive diet of beef. By that I mean just exactly what I say—beef and nothing else, no bread, no vegetables, nothing absolutely to eat but beef. The beef may be cooked or raw, as the patient prefers; roast beef or broiled steak or chopped beef. No restriction is placed on the quantity. Water is allowed *ad libitum*—no other drink is permitted. I have reduced patients twenty-five pounds in two weeks by this method and without any apparent injury. I should not recommend this diet in persons with diseased heart or kidneys, nor would I continue it for more than two weeks at a time.

The Banting system of diet is also rich in proteids and should be used only for persons with sound kidneys for that reason. According to Hutchinson the Banting diet is as follows:

Breakfast: 4 to 5 oz. of beef, mutton, kidneys, broiled fish, bacon, or any cold meat except pork, a cup of plain tea and a little biscuit or 1 oz. of toast.

Dinner: 5 to 6 oz. of any lean meat or fish, any vegetable except potatoes, 1 oz. dry toast, fruit, any kind of poultry or game.

Tea: 2 to 3 oz. of fruit, a rusk or two, and a cup of plain tea.

Supper: 3 to 4 oz. of meat or fish.

To this Banting added claret, madeira or sherry. It would seem to me best to leave that out. I agree with Anders that these cases do better without alcohol.

The Banting system gives about 1100 calories per diem, whereas a normal diet contains about 2500.

The Oertel system cuts down the fats and limits the amount of fluid.

Ebstein gives more fat and excludes sugars, sweets of all kinds, potatoes in every form. He also limits the amount of bread to 3 or 3½ ounces. His diet is as follows:

Breakfast: Tea without milk or sugar, 2 oz. of bread with plenty of butter.

Dinner: Soup, 4½ to 5½ oz. of meat with sauce, green vegetables, fresh fruit.

Tea: Same as breakfast.

Supper: Tea, one egg, fat roast meat or ham, smoked fish, 1 oz. of bread with butter, a little cheese, fresh fruit.

This gives about 1300 calories.

Von Noorden gives small meals every two hours.

Water may be used freely.

Tea or coffee without sugar are permissible.

Drugs have little action on obesity.

I have used the *phytolacca* berry unsuccessfully.

I have used *iodothyrin* without effect other than to produce tachycardia.

SCURVY.

Synonym: Scorbutus.

Definition: A constitutional disease characterized by great debility, with anemia, mental depression, a spongy condition of the gums, and a tendency to hemorrhage.

Historical Note: Scurvy is supposed to have existed very early in the world's history. The first definite descriptions of it did not appear till the thirteenth century. It occurred in armies in besieged towns and in ships on long voyages. Owing to the unsanitary condition of the times it often prevailed in urban communities. In 1798 James Lind and Sir Gilbert Blane improved the dietary in the British navy and added lime water to it. That was the beginning of a method of prophylaxis that has finally resulted in almost extinguishing the disease.

Etiology: Scurvy is due to a lack of vegetable matter in the dietary. Other food may be sufficient. As predisposing factors unhygienic conditions, excessive work or worry, exposure to cold and wet, or debilitating disease, seem to play a part. All persons exposed to these conditions may not be attacked, however. Any age and either sex may suffer.

The disease has also been considered infectious.

Morbid Anatomy and Pathology: In a person dead of scurvy decomposition sets in rapidly. Ecchymoses are found in various parts of the body. Effusions of blood and of plastic material are found in the subcutaneous tissues and between bones and their epiphyses. There is fatty degeneration of the muscles.

The heart is small and flabby and may or may not be filled with blood. The mouth shows stomatitis.

Symptoms: Scurvy is of insidious onset. The patient feels tired, he suffers from shortness of breath and from palpitation. The skin becomes dry and rough. It is cooler than normal. Petechiæ appear on the limbs. The patient is weak and dizzy when he tries to walk. These symptoms may be weeks in developing.

The patient is depressed. He has pains and aches in the joints and muscles.

The appetite is erratic. At times it may be good, at other times

there may be disgust for food, or the patient may crave unusual things.

At first the mouth shows no special change. The gums are pale, slightly tender, and the patient has a bad taste. Later, the gums become darker, inflamed, swollen and tender. They become spongy and bleed easily. These changes are most marked between the teeth, where teeth are absent they are not so apt to occur. Exceptionally necrosis of the gums may take place. Ulcerative stomatitis may occur, making eating of solid food impossible.

Ecchymoses appear in the skin, especially on the legs, sometimes on the trunk and arms. They rarely appear on the face. The mucous membranes are rarely affected.

In severe cases there may be actual extravasations of blood in the subcutaneous tissues severe enough to form blebs. These may break down leaving ulcers.

Other hemorrhages may occur, notably epistaxis, on slight provocation. As the disease progresses anemia becomes marked.

Fever does not occur. The pulse is slow and weak and may be intermittent. The patient wastes away.

Complications and Sequelæ: Pneumonia, both lobular and lobar, frequently complicates the disease. Pleurisy and pericarditis sometimes occur.

Diagnosis: A history of improper feeding—a lack of vegetable food—with the spongy gums and hemorrhages in the skin, the mental depression and debility, should make the diagnosis easy. Other hemorrhagic conditions lack some of these elements.

Prognosis: The prognosis in mild cases is good provided the diet can be properly regulated.

Treatment: The hygienic conditions should be improved and the dietary corrected. Fresh soups and vegetable juices may be used if the patient cannot chew solid food. Lime and lemon juices are good correctives. Plenty of vegetable food should be given, especially the succulent vegetables.

Homœopathic remedies may be needed in bad cases.

China may be used in excessive debility with hemorrhages from the mouth and intestines. There is ringing in the ears, the face is pale. There is a desire for sour things. Emaciation.

Arsenic is called for with the arsenic restlessness from exhaustion and thirst for small quantities of water.

Cistus Canadensis is called for if the gums and mouth are in a particularly offensive condition. There is extreme sensitiveness to cold.

Kreosote, there is bleeding from gums, nose, uterus. The patient is despondent.

Mercurius, the gums are sore, there is stomatitis.

BARLOW'S DISEASE.

Synonym: Infantile Scurvy.

Definition: A peculiar form of scurvy occurring in infants.

Historical Note: The condition was described by Cheadle and others. Barlow made a careful study of the subject and it now bears his name.

Etiology: Infantile scurvy is caused by artificial feeding. Usually malted milk and condensed milk, when used exclusively, seem to be the most frequent offenders, although many other prepared foods have been used in other cases, and quite rarely a child nursed at the breast may be affected. The majority of cases develop under twelve months.

Morbid Anatomy and Pathology: The lower limbs are most often affected. The hemorrhage is subperiosteal and mostly in the long bones. The epiphyses may separate. Sometimes the upper extremities and vertebræ are affected. There are extravasations about the joints. Occasional hemorrhage occurs in the various organs of the body.

Symptoms: The patients become anemic, there is loss of appetite, and there is some gastro-intestinal disturbance. As the disease progresses swelling appears near the joints. There may be a little tenderness but no fluctuation. So long as the little patient is let alone he seems to suffer no pain. He keeps perfectly quiet to avoid pain. There may be hemorrhages in the skin. If there are no teeth the gums are not affected. If a tooth shows hemorrhage may occur in the gums. The sternum may appear to be sunken in as though from a blow. Hemorrhage sometimes occurs in the orbit producing a proptosis of the eyeball.

The fever, if any, is irregular, and is usually 100° to 103° F.

Complications and Sequelæ: Barlow's disease is sometimes associated with rickets.

Diagnosis: The disease must be differentiated from purpura, which is not characteristically a joint affection; from rheumatism; from rickets; from poliomyelitis, which develops suddenly.

Prognosis: The prognosis is good if the food is properly regulated. Otherwise it may go on to a fatal issue.

Treatment: A change of food to a properly modified fresh milk and the addition of orange juice to the dietary will usually cure the condition.

Drugs are rarely indicated. Occasionally a homœopathic prescription may help some. (See remedies mentioned under scurvy.)

SECTION V.

Poisonings and Intoxications.

ALCOHOLISM.

Definition: Alcoholism is the name given to the effects of the abuse of alcohol on the mental and physical make-up of the individual.

Historical Note: There seems to be an inherent craving in the human economy for a stimulant, and alcohol in some form has been used from time immemorial to satisfy that craving. Alcohol has been used from the beginning of history as a beverage and as a medicine. The effects of its use have of late years been exhaustively studied, but with the most conflicting conclusions by different observers. The action of alcohol is so different on different persons that a sharp distinction cannot be made as to what constitutes use and what constitutes abuse. The opinion is gradually becoming prevalent that the less alcohol one uses the better.

Etiology: The cause of alcoholism is the excessive use or the abuse of alcohol. Persons unaccustomed to its use may be overcome by a comparatively small quantity and develop (1) *acute alcoholism.* Persons who use it habitually may gradually develop (2) *chronic alcoholism.* In either case a larger quantity than usual may cause acute symptoms even to delirium, (3) *delirium tremens* or *mania-a-potu.*

Persons of a neurotic temperament seem to be more prone to the alcohol habit than are others. The taste for alcohol is also said to be hereditary. My limited experience does not quite bear that out, as I have known children of drunkards who were most rabid total abstainers, and children of total abstainers who were drunkards.

Morbid Anatomy and Pathology: In *acute alcoholism* we may find an acute gastritis after death. There may be alcohol in the central nervous system and in the liver.

In *chronic alcoholism* there is always a chronic gastritis. In the liver are commonly found fatty and atrophic changes with an increase in the connective tissue. Parenchymatous and interstitial changes take place in the other internal organs, notably the kidneys. I have found fatty liver and enlarged kidneys with non-adherent capsules in the majority of cases.

Wet brain is common, that is, the veins are distended and there is an excess of fluid in the ventricles and at the base. The brain may be edematous.

Symptoms: (1) *Acute alcoholism* or *intoxication.* The symptoms are best described by noting the effects of the ingestion of a considerable quantity of alcohol on an individual.

A small quantity of alcohol acts on the cerebro-spinal system, giving a sensation of warmth by causing a relaxation of the terminal capillaries and a consequent flush of blood to the body surface. There is no actual rise in temperature, the flush of external heat is at the expense of the internal organs. Large doses of alcohol cause an actual lowering of the temperature. The cardiac force and arterial pressure are increased by the physiological dose. The functional activity of the brain is augmented. There is a feeling of mental and physical exhilaration followed by one of comfort and repose. Lack of inhibition follows. There is lessened ability to work. If the ingestion of alcohol is continued to toxic doses this disturbance of the system becomes more and more marked; the exhilaration is changed to intoxication, there is loss of co-ordination; there is loss of muscular power, the subject sees everything double, there is sleeplessness, delirium, anesthesia, and when the full limit is reached, collapse.

Alcohol is a true poison, and a person who has taken a full toxic dose is in an alarming condition. The temperature is subnormal, the body is bathed in a cold perspiration, the pulse may be so weak as not to be felt, it may be very slow or very fast, the breathing is labored and stertorous, the pupils may be contracted or dilated, they do not react to light, the lips are blue, the insensibility is profound. Sometimes acute delirium and convulsions ensue. Either condition may terminate in recovery or in death.

The amount of urine is increased, the amount of urea de-

creased. Frequently in cases that do not progress to collapse nausea and vomiting set in, and there is an acute gastritis due to the direct chemical action of the alcohol.

(2) *Chronic alcoholism.* In this condition there is a chronic gastritis. Alcohol in small quantity stimulates the mouths of the gastric glands causing an increase of the flow of the various juices. If the use of alcohol is habitual there is eventually set up a catarrhal condition of the stomach due to over-stimulation with consequent faulty digestion. Again, alcohol in quantity precipitates pepsin and impairs the utility of the gastric juice, thus giving another source of alcoholic dyspepsia.

Alcohol passes rapidly from the stomach into the portal circulation. Here it stimulates the hepatic cells, and in time causes fatty and atrophic changes with an increase in the connective tissue. This produces cirrhosis of the liver. In spirit drinkers the liver may shrink in size. In malt liquor drinkers the liver may become enlarged.

The kidney structure becomes involved in a somewhat similar manner producing chronic nephritis.

With this change in the great secreting and eliminating organs the patient becomes more or less physically unfit to do his work.

The will power becomes dulled and the nervous stamina gives out. The membranes of the brain become thickened. Sustained mental effort becomes impaired or impossible. The character becomes changed. The patient is irritable. He becomes untruthful and unreliable.

(3) *Delirium tremens* or *mania-a-potu.* In temperate persons who consume a large quantity of alcohol within a short time, but more often in habitual drinkers who take an excessive quantity within a short time, there is sometimes induced a condition known as delirium tremens, or mania-a-potu. As a rule, there are premonitory symptoms for a day or two when the patient is in a highly nervous condition. He cannot keep quiet, is restless and sleepless, he starts and jumps at little things, talks to himself, and is uncertain in his ideas. He is partly conscious, with lapses into imaginary states. Then he begins to see and hear altogether imaginary things. The delirium takes on peculiar character-

istics. Hallucinations are common and varied. The patient either goes through some long imaginary experience as a connected whole; or, more often, he will see monstrous animals, or imagine vermin of some sort are attacking him, and try to fight them off. One patient of mine was distressed by a big black horse that insisted on sitting on his chest. Another suddenly knocked down a big policeman and tore off his uniform because "the Lord Jesus Christ told him to take the buttons off that policeman's uniform," and so on.

At times the patient is completely disoriented. It may be possible to get an occasional rational idea from him, or to change the current of his thoughts. He is in a state of psychic suggestibility. His hallucinations are apt to be curiously mixed with actualities.

There is tremor. The protruded tongue will show fine fibrillary motions. The fingers will tremble when extended.

In these cases the patients should frequently be examined physically, because many of them develop a pneumonia while in delirium, a complication that is of bad prognostic omen.

Pneumonia, or other acute inflammatory conditions, or severe injuries, in a habitual user of alcohol, may precipitate an attack of delirium tremens.

Alcoholic insanity. The alcohol habitue may develop a true permanent insanity. In this there is loss of memory, disorientation and hallucinations. There may be periods of depression or excitement. In extreme cases there is complete dementia.

Complications and Sequelæ: Any disease may co-exist with alcoholism. Tuberculosis and pneumonia are of frequent occurrence in chronic alcoholics. Gastric catarrh, cirrhosis of the liver and nephritis have been mentioned above. Multiple neuritis may occur.

Diagnosis: Some cases of *acute alcoholism* are easy to diagnose. Others are extremely difficult. If the patient is unconscious when seen he should always be given the benefit of the doubt. An acute alcoholic, one simply in a drunken stupor, will come out of it after a few hours' rest. But a drunken man may pass into uremic coma, or he may have a stroke of apoplexy come on, or

he may have fallen and fractured his skull and thus caused a cerebral hemorrhage. All these possibilities must be taken into account before passing final judgment.

Chronic alcoholism is diagnosed largely from the history of the case. If the patient tries to hide the cause of his condition the diagnosis may not be easy at first. Hardly any other disease has the peculiar shifty mental make-up of the alcoholic however, and if that is detected, together with a history of impaired digestion, enlarged or contracted liver, and nephritis, alcohol as a cause is a legitimate surmise.

Delirium tremens may be mistaken for delirium from other disease, and vice versa. Other causes must be sought for to make sure.

Prognosis: In *acute alcoholism,* if the condition is not too profound, the prognosis is good.

In *chronic alcoholism* the prognosis is always questionable. A few patients are cured. Many are patched up for a short time, but the majority go on or have relapse after relapse, until some complication carries them off.

In *delirium tremens* the prognosis of a given attack depends almost entirely on (1) the treatment, (2) whether or no pneumonia or some other complication supervenes. If the patient does not die the delirium passes off in a few days.

Treatment: Abstinence from alcohol is the only sure prophylactic against any form of alcoholism. Some persons can stand, apparently without detriment, a large amount of alcohol, and can use it habitually for a long time.

In the process of becoming intoxicated, *acute alcoholism,* at some point the inhibitory power is lost; then the patient don't care, he is a creature of circumstance and will go on drinking till overcome.

The *chronic alcoholic* becomes such insidiously. He takes his drink with more or less regularity until all at once he discovers he cannot do without his accustomed stimulant.

Delirium tremens is the direct result of an overdose, and may appear suddenly.

In *acute alcoholism* the use of alcohol must be stopped at once.

In acute cases it may be necessary to wash out the stomach. Rest in bed for a few hours or a day or two is usually all that is necessary.

The patient may be given a little vichy, perhaps, to settle the stomach. Black coffee may be used freely. Milk, milk and vichy, milk toast, soft boiled eggs may be given.

After sobering up from a debauch a victim will often have an aversion to alcohol lasting for a considerable time.

Chronic alcoholism is best treated in an institution. Alcohol must be withdrawn absolutely. The patient must be fed upon a good nourishing diet. Massage or gentle exercise should be prescribed. Something must be suggested to occupy the patient's mind.

Coffee in moderate amount may be used. Sugar, either as such or in the form of some wholesome candy, may be used to allay the craving for alcohol.

Nux vomica is the most frequently used drug for either acute or chronic alcoholics.

Delirium tremens. Alcohol must be absolutely prohibited. The patient is best left in a padded room where he cannot hurt himself, with a bed to lie on on the floor. Restraint in bed is dangerous. My experience, which in hospital practice has been large, teaches me that this one factor, a padded room versus restraint in bed, often means the difference between recovery and death from exhaustion.

Unless doped with some powerful drug, like morphine or the bromides, the patient may remain wakeful and talkative and restless for twenty-four or thirty-six hours or even longer. Heavy drugging is detrimental because it adds to the general depression of the vital forces.

Tying in bed aggravates the intense restlessness, and the patient will endeavor to get away, thus adding to his physical exhaustion. In a padded room he may keep constantly moving, but he is not using his strength to the same excessive degree. He will finally drop into a restful sleep.

Warm baths are of great service in the various forms of alcoholism.

Strong *black coffee* given *ad libitum* has been of great service in numbers of cases of delirium tremens at the Flower Hospital. Coffee is an antidote to alcohol. It is a stimulant. The delirium of coffee, like the delirium of alcohol, is very loquacious.

Avena sativa tincture given in thirty minim doses in water every two or three hours will frequently brace up a patient who is on the verge of delirium tremens. He is restless, nervous, with tremulous tongue and tremulous fingers. *Avena* does not help the delirium, it is of service only before the delirium appears.

Capsicum is an excellent remedy for delirium tremens. The patient is delirious, sleepless. He is thirsty, but water makes him nervous. There is a bad odor to the breath. Dram doses may be given in milk.

Gelsemium tincture in one to five drop doses is recommended as a sedative by Pettey.

Cimicifuga tincture, in drop doses, was Dr. J. T. O'Connor's favorite prescription for delirium tremens at the old Ward's Island Homœopathic Hospital.

Nux vomica is a useful drug for the nervous and gastric symptoms.

Hyoscyamus may be given for the peculiar mental hallucinations. The patient is suspicious.

Stramonium is sometimes indicated in a loquacious delirium. The patient wants company.

TOBACCO POISONING.

Definition: Tobacco poisoning results from the use of tobacco. It may be acute or chronic.

Historical Note: Tobacco was introduced to the civilized world in the sixteenth century. Tobacco is native to America.

Etiology: Tobacco is occasionally used as a medicine. It may be used in enemas, or the leaves may be used as poultices. Such use sometimes causes acute poisoning.

Tobacco is widely used for smoking, less widely for chewing and as snuff. Cases of acute poisoning sometimes result from that. More often cases of chronic poisoning arise from such use.

The poisonous principle is nicotine, one of the alkaloids of tobacco.

Symptoms: Tobacco is a very powerful poison, next to prussic acid the most powerful and quickest acting known. Death has been known to occur within three minutes after its use. The patient dies of respiratory paralysis.

All are familiar with the symptoms of the first smoke. There is nausea and vomiting, cold and clammy skin, feeble pulse, in short, collapse.

Chronic poisoning causes palpitation, tachycardia and irregularity of heart's action, sometimes angina pectoris. In some it causes loss of sleep. On the other hand, I have known the smoking of an excessive number of strong cigars to put a man into what was like a drunken stupor that had to be slept out.

Smoking too much at one time will cause an increase in the quantity of urine.

Other effects of nicotinism are gastric derangements and chronic catarrhal conditions of the nose and throat.

Excessive continued use may cause ataxic symptoms. Tobacco amblyopia is another serious chronic effect. There may be a highly nervous or depressed mental condition, due solely to the excessive use of tobacco.

Prognosis: The prognosis is good if tobacco is given up.

Treatment: Stop the use of tobacco, and in acute cases give stimulants.

The late Dr. T. F. Allen used to say that the habitual use of tobacco created a craving for alcohol, and that the two antidoted each other. It seems to me as though the first statement is true only in part, for many smokers do not drink. Alcohol is an antidote to tobacco. Strong coffee I believe aggravates the symptoms of tobacco.

Treat chronic cases symptomatically. According to Hering the following homœopathic antidotes may be used:

Ipecac for the vomiting from tobacco.

Arsenic for the bad effects of chewing.

Nux vomica for the gastric symptoms and bad taste after too much smoking.

Phosphorus for the palpitation.

Sepia for the neuralgia, dyspepsia and chronic nervousness.

Lycopodium for the impotence.

Plantago major is said by Hering and by Boericke to cause an aversion to and cure the craving for tobacco.

OPIUM POISONING AND MORPHINISM.

Definition: Poisoning from the use of opium or some of its compounds. The chronic form is most often due to the use of morphine in this country.

Historical Note: Opium was in use as a medicine before the Christian era. Its use as a habit, for its stimulating and pleasureable effects, is supposed to date from the sixteenth or seventeenth centuries.

Etiology: In Eastern countries opium smoking is the preferable form of use. In this country opium smoking is chiefly found among the lowest outcasts of society. The preferable form of the vice here is morphinism, and by far the majority of these patients have begun the use of morphine for the relief of pain.

All forms of the vice entail the use of an increasing quantity until the dose sometimes becomes enormous. The largest dose with which I have personally been familiar was sixty grains of morphine a day, taken hypodermatically, by a physician. The enormous size of this dose may be appreciated when it is remembered that ⅛ of a grain has occasionally proven fatal, and that ¼ of a grain is considered the limit of safety in the ordinary individual.

Morbid Anatomy and Pathology: In acute cases the various organs are sometimes congested. In chronic cases there may be a catarrhal condition of the digestive tract. The tissues are anemic. Rigor mortis is said to be of brief duration. Decomposition sets in early.

Symptoms: In *acute poisoning* the patient in a short time lapses into unconsciousness. The stupor becomes profound. The pupils are minutely contracted. There is stertorous breathing.

In *chronic cases* the effect of the accustomed dose is to cause a gentle stimulation, and then a dreamy mental condition. A patient addicted to the use of morphine develops gastric catarrh with indigestion. The bowels become very constipated. The

patient develops a peculiar pallor of the skin. He becomes anemic and thin. There is headache, vertigo and insomnia. His mental and physical stamina are lost. He develops a tremor. Mentally he becomes irritable, hysterical and totally unreliable. He will lie to gain his ends. The whole moral nature becomes changed for the worse. He is hysterical.

The sexual function is lost in men. Amenorrhea, and in case of pregnancy miscarriage result in women.

Sometimes delirium tremens results, in all respects like that due to alcoholism. The patient may become maniacal and do injury to others, at other times he is apathetic.

Complications and Sequelæ: The complications and sequelæ have been noted in the symptomatology. The gastric symptoms are the most frequent.

Diagnosis: The diagnosis may be difficult because the vice is nearly always a secret one. If the history is known the diagnosis is easy. In some of the wretched derelicts that I have seen in the Metropolitan Hospital the craving for morphine makes the victims beg for it. Exposure of the body has in several instances shown a hundred or more marks where the hypodermic needle had been inserted.

Prognosis: In acute opium or morphine poisoning vigorous treatment will usually save the patient.

In the chronic form the prognosis is good if the patient can be kept under strict surveillance for a sufficient length of time. This may mean months or even years. The case I speak of where the patient injected sixty grains of morphine a day required two full years to complete what has proved to be a permanent cure.

Treatment: The only sure prophylaxis is to avoid the use of opium or morphine for any purpose. To prevent formation of the morphine habit, if a physician finds it necessary to use morphine in a case he should always administer it himself, and under no circumstances allow the patient or family to do it.

In acute poisoning the patient must be aroused at all hazards, and large quantities of black coffee given until the effect of the drug wears off. Wine, or spirits, may also be used.

The chronic morphine patient must be placed under the best

possible surroundings. He is best treated in an institution where he may be under the constant surveillance of persons who cannot be bribed or threatened into giving him morphine. My personal preference is to withdraw all morphine at once.

In some cases the resulting distress is so great that the dosage may have to be diminished gradually. Beside the great unsatisfied craving when morphine is stopped, the patient may become weak, he may have vomiting and diarrhea, he may become delirious. All these symptoms usually subside in a few days.

In chronic poisoning, beside feeding up the patient, the various symptoms may call for black coffee or alcohol.

Pettey claims that morphinism and other drug diseases are a sort of toxemia. His first line of attack then is to eliminate the drug from the system. He gives purgatives, as follows:

℞. Calomel,
Powdered extract of cascara....aa gr. x.
Ipecac .....gr. j.
Strychnine nitrate .....gr. 1/4.
Atropine sulphate .....gr. 1/50.
M. and make four capsules.
Sig.—One every two hours until four are given.

Eight hours later give castor oil or citrate of magnesia.

After that it may be necessary to use morphine again, but in less than the usual dose.

After forty-eight hours repeat the purgatives.

When the morphine craving returns he gives *scopolamine* 1/200 hypodermatically.

Other symptoms are met as they arise.

Homœopathic antidotes are *belladonna, ipecac* and *nux vomica.*

COCAINE POISONING.

Definition: The effects of the use of cocaine.

Historical Note: Cocaine is the active principle of *erythroxylon coca,* a plant indigenous to South America. The natives chew the leaves, and it is said to give them great physical endurance. Cocaine was isolated from coca by Niemann about 1860. It has been used in various ways as a local anesthetic since about 1880.

During the past few years it has attained considerable importance because of the increasing number of habitual users who have broken down mentally and physically.

Uses: Cocaine is a valuable local anesthetic. Used in two to four per cent. solution on mucous membranes, as in the eye, it will in a few moments produce local anesthesia. Injected into the tissues it causes anesthesia of the surrounding parts. Injection into the spinal canal causes anesthesia of the parts below without loss of consciousness.

Chronic Poisoning: In this country the hypodermatic use of cocaine is prevalent among the dissolute. Occasionally its use as a spray in the nose, or its use as a drink in coca wine, becomes a habit. Persons who use it that way develop a drug habit and must use constantly increasing doses as does the morphine habitue. The cases that I have seen have been at the Metropolitan Hospital, usually among the lowest of prostitutes, who are also addicted to the use of morphine and of alcohol.

Pettey says that in the south coca is used in various forms in soda fountain drinks.

Symptoms: *Acute poisoning.* Under proper administration the action of cocaine is purely local and passes off after a few hours.

In case of an overdose—and an occasional patient will have an idiosyncrasy against it—we get *acute poisoning.* There is first an excitation, then a depression. The pulse is increased in frequency and lessened in strength. Respiration becomes shallow. The patient is pale, breaks out into a profuse perspiration, and is anxious. He may develop a violent delirium. He has pains in various parts of the body. He may have convulsions, or may become rigid.

Chronic poisoning. The drug is taken for its stimulating effects and for the sense of well being and lack of fatigue that it produces. As these effects are transient the doses must be repeated. As tolerance is established the doses must be increased.

The appetite fails, the digestion is upset, and the patient becomes emaciated. The face becomes sallow and the eyes sunken. There is tremulousness. With the physical deterioration there is

also mental decay. The patient suffers from insomnia. He has hallucinations and delusions. He has sensations of creeping vermin over the skin. There is loss of memory. The only object in life is to get cocaine and then more cocaine. Finally he becomes a mental and physical wreck. All moral sense is lost, he will resort to any extremity to satisfy his craving for the drug.

Prognosis: The acute effects do not last long. The patient either recovers in a short time or dies. If he recovers tingling may persist for a day or two.

In chronic cases the prognosis is unsatisfactory.

Treatment: Stimulants must be used in acute cases, alcohol or ammonia or oxygen. Chloroform may be given for convulsions.

Boericke mentions *gelsemium* as an antidote.

The only chance for absolute cure for cocaine habitues is in an institution.

Pettey advises elimination by purgatives as in the morphine habit. In his experience the users of cocaine alone are not numerous, most cocaine users are morphine users as well. He stops the cocaine at once, continuing the morphine for a time. Then after some months attacks the morphine habit.

Gelsemium may be used to counteract cocaine.

ETHER POISONING.

Definition: The effects of inhaling or drinking ether.

Historical Note: Ether drinking, as a habit, has been in vogue in Ireland since 1846. It is almost unknown in this country.

Etiology: From one to two drams are drunk at a time, and the user can experience the pleasure of three or four intoxications in a day. The effect does not last more than an hour or two.

Morbid Anatomy and Pathology: Not known.

Symptoms: The effects come on quickly. Small doses produce exhilaration. The patients laugh and shout and sing. Larger doses produce violent excitement.

Chronic drinkers suffer from indigestion. They are weak and debilitated. There is wasting. Patients lose all moral sense.

They will lie or steal to get their drug. They are pugnacious and hysterical.

Treatment: Treatment of habitual use of ether consists in reform of the habits.

For an overdose Bartholow mentions *quinine, strychnine* and oxygen as antidotes.

Ether Asphyxiation: The danger from an ether anesthesia lies in respiratory failure. If that occurs stop the ether, invert the patient, and perform artificial respiration. Keep the patient warm. Amyl nitrite inhalations may be of service.

PARALDEHYDE POISONING.

Paraldehyde is an ethereal compound. It is said to be a constituent of various cough mixtures and other quieting nostrums. It is a hypnotic.

Symptoms: Sufferers from paraldehyde poisoning present symptoms somewhat like those of alcoholism. There is mental unreliability, sometimes hallucinations, loss of moral tone, and emaciation and anemia. The heart is irregular and weak. The patient's digestion is impaired. The patient has an excessive appetite. There is much gas. The patient is constipated.

Prognosis: The prognosis is usually good.

Treatment: The treatment is similar to that for alcoholism. Potter says it should be conducted in an inebriate asylum.

CHLOROFORM POISONING.

Definition: The symptoms induced by inhalation of chloroform.

Etiology: Habitual users take chloroform by inhalation, taking a few whiffs now and again. A tolerance is never established, the dose must always remain the same.

Morbid Anatomy and Pathology: Nephritis has been found in death from chloroform. The heart, liver and kidneys have also shown fatty degeneration.

Symptoms: To the habitual user of chloroform the whiffs give a pleasant sensation. Fatal cases present vomiting. The pulse is

rapid. Delirium develops and may change to coma and then death. The pupils are dilated, the reflexes are lost.

As in other drug habits, the victims become unreliable. They grow thin. There is weakness and tremor and disturbed moral and mental disposition.

Treatment: For acute poisoning artificial respiration should be carried on. Plenty of fresh air should be admitted to the room.

Oxygen inhalation may be tried.

For the chloroform users institutional care is best.

ILLUMINATING GAS POISONING.

Definition: Poisoning by inhalation of illuminating gas.

Etiology: Illuminating gas is said to owe its toxic properties to the carbonic oxide contained. Sometimes it is taken with suicidal intent. At other times it is inhaled accidentally because of a leak in the gas connection, or because a burner has blown out. In my service at the Flower Hospital I frequently have gas poisoning cases brought in.

Morbid anatomy and Pathology: Carbonic oxide unites with the hemoglobin of the blood rendering it incapable of carrying oxygen to the tissues. After death the red color of the blood is retained. The organs of the body are abnormally red.

Symptoms: The symptoms come on gradually. If the poisoning is accidental, as is so often the case, and is due to leakage from the gas fixtures after the patient has fallen asleep, he may pass from sleep to unconsciousness. Sometimes embarrassed respiration will waken him and he will make an effort to get to the air, only to be finally overpowered on the way to the window or door.

Other cases begin with a sense of fullness and throbbing in the head. There is giddiness and headache. There is ringing in the ears and spots before the eyes. There may be nausea and vomiting. Coma comes on, sometimes convulsions. Respiration is embarrassed. The skin takes on a dusky hue.

If the patient comes out of it he may be dazed for a time. Occasionally insanity results.

Complications and Sequelæ: Sometimes pneumonia follows.

Diagnosis: The diagnosis is self evident from the surroundings.

Prognosis: The prognosis is doubtful. It has been good in the majority of cases I have seen because they have been discovered in time. If not removed from the gas death will result.

Treatment: The first requisite is removal from the gas laden atmosphere to a place where fresh air is plentiful. Then some form of artificial respiration must be carried out for a considerable length of time, either until the patient is able to breathe naturally, or as long as there seems to be a chance of resuscitation.

Venesection may be required in desperate cases with injection of normal salt solution.

Usually these measures, some or all of them, will suffice.

Injections of strychnine may be given.

After symptoms and complications must be met as they arise.

GASOLINE POISONING.

Definition: Poisoning by inhaling the fumes of gasoline.

Etiology: Gasoline for cleaning fabrics may overpower the user. Gasoline in quantity for automobiles may do the same thing. A patient in my ward at the Flower Hospital in 1910 was the driver of a truck that transported gasoline in quantity to garages.

Symptoms: The patient becomes giddy, perhaps nauseated. There is loss of voluntary motion, and the patient may lapse into unconsciousness.

Prognosis: The prognosis is good if discovered in time.

Treatment: Removal of the patient from the fumes of gasoline is the first requisite. Then comes plenty of fresh air. Artificial respiration must be done if necessary. Rest in bed.

Stimulate if necessary.

Meet any other symptoms as they arise.

BROMISM.

Definition: The effects of continued use of bromine, usually in the form of bromide of potassium. Other bromides may produce it.

Historical Note: Bromide of potassium seems to have come largely into use, especially for epilepsy, along about 1860 or a little later.

Etiology: The condition known as bromism is caused by the long continued use of the bromides.

Symptoms: There is a gradual impairment of the mental faculties. The patient becomes dull and stupid. There is headache and confusion of mind in some. In others there are hallucinations. There is lessened sensibility of the skin and mucous membrane. The gait is uncertain and sometimes there is lack of co-ordination. There is a pallor of the skin and often acne of the face and back. The breath has a peculiar fetor.

Digestion becomes impaired and the patient becomes thin. Sexual feeling and power are lost.

Prognosis: If the use of bromides is stopped the patient may recover.

Treatment: Arsenic given with bromides delays bromism. Strong coffee with meals prevents bromism.

If the patient is very susceptible bromide of sodium, which is less toxic than the potassium salt, may be used.

Tonics may be given.

IODISM.

Synonym: Iodine poisoning.

Etiology: Occasionally cases of acute iodine poisoning result from its use in treating a case.

Chronic cases may result from the long continued use of iodide of potassium.

Symptoms: Large doses of iodine may produce collapse. There is rapid, weak pulse, with pallor, cyanosis, and dyspnea.

There is vomiting. Albumin appears in the urine, which becomes scant. There may be symptoms of coryza and sore throat. There is severe frontal headache.

In chronic cases the digestive tract becomes upset. The patient has symptoms of gastric catarrh. There is a catarrhal condition of the eyes, nose and throat. Acne is quite common. Strümpell says there may be an erythema nodosum. The patient has headache and may be nervous and irritable.

Treatment: In acute cases give white of egg. For the collapse use alcohol.

In chronic cases diminish or stop the doses of iodide of potash.

Hepar antidotes the effects of iodide of potash.

Antimonium tartaricum, arsenicum album, camphor, coffea, hepar, sulphur, antidote iodine.

IODOFORM POISONING.

Historical Note: Iodoform was discovered in 1822. It is extensively used in surgery.

Etiology: Some cases of poisoning have followed the use of iodoform as a dressing for wounds.

Symptoms: The patient has headache, vertigo, he is restless and cannot sleep. He may have mania and delusions of persecution. The temperature rises to 104° F. or more. The pulse becomes weak and rapid.

In severe cases there may be convulsions, coma or collapse.

Diagnosis: The diagnosis is made from the history.

Prognosis: Some patients have an idiosyncrasy against iodoform. They may die quickly.

In others less susceptible the symptoms will disappear promptly on suspension of the remedy.

Treatment: Iodoform should not be used too lavishly. Immediately any bad symptoms result from its use the remedy should be discontinued.

Stimulants should be used. Copious drinks of water should be given.

Bartholow recommends small doses of opium tincture frequently repeated.

The homœopathic remedies to use are *arsenicum album, camphora, coffea, hepar,* and so on.

CARBOLIC ACID POISONING.

This includes poisoning by lysol and other carbolic acid preparations.

Etiology: The poisoning may be intentional or accidental.

Morbid Anatomy and Pathology: Carbolic acid applied to tissues forms first a white coating. If the action is continued this eschar later becomes brownish. The blood becomes dark and coagulates slowly.

Symptoms: There is first a local effect when carbolic acid touches the skin or mucous membranes. The part is whitened. If continued an eschar is formed.

The symptoms produced are vertigo, contracted pupils. Sometimes the patient becomes rapidly unconscious. The pulse becomes rapid, respiration becomes stertorous. The urine is dark greenish in color. It contains albumin and sometimes blood. Carbolic acid is eliminated by the kidneys.

Treatment: Alcohol pure is a direct antidote to carbolic acid if applied quickly wherever the acid has been, either internally or externally. Vinegar may be used if nothing stronger is at hand. Vinegar was Dr. Edmund Carleton's antidote.

Atropine is said by Bartholow to be the antidote for the systemic effects of carbolic acid.

Arsenicum album may be given later.

ATROPINE POISONING.

Atropine is an alkaloid of belladonna. Both are used in medicine.

Etiology: Atropine poisoning may be accidental or intentional.

Symptoms: Excess of belladonna or atropine causes a dryness of the mouth and throat, with great thirst. The pupils are dilated. There is dizziness and headache and hallucinations. Convulsions may occur. There may be inco-ordination of the muscles. The pulse is full and bounding, the face flushed, there may be a general redness of the skin.

Diagnosis: The history of the case is a great help in diagnosis.

Prognosis: If death occurs, it is caused by respiratory paralysis.

Treatment: Pilocarpine and physostigma are physiological antidotes.

Morphine or opium may prevent paralysis of respiration.

After large doses coffee used freely may be of service.

Camphor, coffea, hepar, hyoscyamus, opium, in potency, are useful for the after effects.

DIGITALIS POISONING.

Etiology: Digitalis poisoning results from too large and too frequently repeated doses of digitalis.

Symptoms: Disturbance of digestion is the first symptom of too much digitalis. There is nausea and vomiting, sometimes diarrhea. The pulse becomes excessively slow. There is dyspnea. The patient is drowsy. The extremities become cold and the patient goes into collapse.

Diagnosis: The diagnosis can be made when the above symptoms occur in a patient taking digitalis.

Prognosis: The prognosis is doubtful. Death often occurs.

Treatment: Emetics are indicated after an overdose.

Black coffee may be given freely. Milk or vinegar may be used.

Ether and camphor may be useful as stimulants.

Nux vomica or *opium* may be given in potency for the after effects.

STRYCHNINE POISONING.

Etiology: Too much strychnine is often given medicinally. Sometimes strychnine is taken with suicidal intent.

Symptoms: In mild cases of overdosing various muscular twitchings occur. There is sometimes tympanitis from paralysis of the bowel. I have seen this accident happen in typhoid fever where strychnine had been used as a heart stimulant.

In more severe cases the twitchings go on to convulsions. There may be trismus and opisthotonos. All reflexes are exaggerated. The pulse becomes very rapid and weak.

Diagnosis: The diagnosis may be made from the history.

Prognosis: In severe cases the prognosis is bad.

Treatment: Emetics or the stomach pump may be used after an overdose.

Alcohol or black coffee may be given freely. Castor oil is useful.

Chloroform may be necessary to control the convulsions.

For the after effects *chamomilla, cocculus* or *pulsatilla* may be indicated.

LEAD POISONING.

Synonym: Plumbism.

Definition: Lead poisoning is the result of using lead and getting it into the system.

Historical Note: Lead poisoning has been known for centuries.

Etiology: Workers in lead, as painters, are subject to lead poisoning. It may be inhaled or it may enter the system by way of the digestive tract from soiled hands. Epidemics have occurred where the drinking water was contaminated with lead. Contaminated beer has also caused it. It occurs in pottery workers. In pregnant women it produces abortion.

Morbid Anatomy and Pathology: The effect of lead is seen in the brain, the peripheral nerves, the liver and kidneys particularly. It causes interstitial changes and parenchymatous degeneration. The tissues are pale. The arteries are sclerosed.

Symptoms: The most common symptom of lead poisoning is colic about the navel due to gastro-enteritis. This may come on quite suddenly in one who has been a long time exposed. I have seen it in men who had been painters for fifteen or twenty years without previous attacks. I have seen it in others after exposure of only a few weeks. As a rule, constipation accompanies it. Anemia is frequent. A blue line appears on the border of the gums, especially if the teeth are not well cared for.

Wrist drop occurs with paralysis of the forearm and hand. It may be on only one side, it is usually on both. Occasionally a permanent deformity results from the contractions.

Cramps sometimes occur.

In extreme cases there may be delirium, convulsions or coma. There is choked disc.

Lead poisoning causes chronic interstitial nephritis and arteriosclerosis with slow pulse. There is loss of weight.

The urine contains albumin, blood, casts, etc.

Complications and Sequelæ: The kidney and gastric conditions noted above and some paralyses are the most frequent complications and sequelæ.

Diagnosis: In workers in lead like painters, typesetters,

plumbers, etc., the diagnosis is not very difficult because we have the history of the patient's occupation. In cases occurring from breathing emanations from lead wall papers or drinking contaminated water the condition may be mistaken for something else because attention has not been directed to lead as a cause.

Prognosis: In a few very violent acute cases death takes place in a short time. I lost a patient in my service at the Flower Hospital in 1913. If actual tissue changes have taken place the ultimate outlook is not good. Convalescence is slow. The occupation will have to be changed to prevent relapses.

Treatment: Those who work at trades where lead has to be handled should be careful to cleanse the hands and particularly the finger nails before eating. In lead works respirators are needed. In other cases search will have to be made to find the source of the trouble so that it may be eliminated.

In acute cases the patients will have to be put to bed. The stomach pump or emetics and purgatives will be required. Sulphate of soda or magnesia acts as an antidote. White of egg and milk must be given.

In most chronic cases the constipation requires enemas. Baths must be given.

The old school relies on iodide of potassium as the remedy—5 to 10 grains three times a day.

Hering gives as antidotes to be used symptomatically *alum, opium, petroleum, nux vomica, platinum, antimonium crudum, cocculus, zinc.*

ARSENIC POISONING.

Definition: Arsenic poisoning may be acute or chronic. Arsenic is an irritant poison in large doses. In continued small doses it may cause chronic gastro-enteric disturbance, weakness, emaciation, etc.

Historical Note: Arsenic was used by the ancients in the arts and as a medicine. It was once in vogue as a slow poison, given secretly, with criminal intent.

Etiology: We may have acute arsenic poisoning due to taking a large dose of arsenic. We may have chronic arsenic poisoning

due to inhaling arsenic from wall papers containing arsenic; from working on artificial flowers or other things containing arsenic; from taking arsenic as a medicine (Fowler's solution), or from eating or drinking something contaminated by arsenic. An epidemic of chronic arsenic poisoning occurred in a town in England a few years ago that was traced to arsenic contaminated beer.

Morbid Anatomy and Pathology: Arsenic is an irritant poison. In acute poisoning it attacks the mucous membrane of the entire digestive tract, spending most of its force on the stomach and beginning of the intestine. It causes thickening, inflammation and even ulceration. In chronic poisoning the whole intestinal tract is involved. The blood is fluid, changes may or may not show in the liver and kidneys.

Symptoms: In acute poisoning the patient has an intense gastro-enteritis with nausea, vomiting and diarrhea. There is severe abdominal pain. The patient has the distressed look that goes with choleraic symptoms. There is faintness, twitching, cyanosis, collapse. The urine is much diminished and there is usually more or less constant desire to pass it.

In those who have been taking arsenic as a medicine—usually in the form of Fowler's solution—there is puffiness under the eyes that becomes noticeable. The digestion is disturbed. Anemia sets in if the medicine is continued.

In the chronic form we get all of the above symptoms in less acute form. There may be various eruptions on the skin. Finally a multiple neuritis develops. The patient becomes emaciated and goes into a typhoid condition.

Complications and Sequelæ are included in the symptomatology.

Diagnosis: In acute cases the action of an irritant poison is self-evident and the diagnosis usually easy. In chronic cases it may be difficult to account for the general breakdown. If in doubt arsenic should be looked for in the urine, as it is eliminated by the kidneys.

Prognosis: Death may occur in a few hours in the acute form. In the chronic form the prognosis is usually good once the source of the trouble is discovered and the cause removed.

Treatment: The use of arsenic must be stopped if the poisoning has come from medicine or from cosmetics or from one's trade. In obscure cases the patient's habits and surroundings must be most carefully gone into so that the source of the trouble can be removed.

In acute cases the stomach tube must be used to be followed by the free use of eggs and milk.

In chronic cases the patient must be built up with proper food, eggs and milk.

The symptoms must be met as they arise.

Sesquioxide of iron is a physiological antidote to arsenic. Lime water or emetics of sulphate of zinc may be given.

Hering gives *camphor, cinchona, ferrum, hepar, iodine, ipecac, nux vomica, tabacum, veratrum* as antidotes for small doses. These will be useful in the chronic poisoning.

MERCURIALISM.

Synonym: Mercury Poisoning.

Definition: Acute poisoning may be due to taking a large dose of mercury by accident or with suicidal intent. Chronic poisoning may be due to too long continued use of mercury as a medicine or to occupations where mercury is used, as in rubber making, etc.

Historical Note: Intentional constitutional mercurialization was once in vogue for certain diseases. Now it is done only in rare cases of syphilis, where the need for heroic action seems imperative. Practically all cases are now accidental—either from the medicinal use of mercury, or from its use in the arts and trades.

Etiology: Mercurialism may occur in those who use mercury in the arts. Mercury is volatilized at ordinary temperatures and the vapor is poisonous. Long continued use of mercury as a medicine may cause it. Anders says women and children are more susceptible than men.

Morbid Anatomy and Pathology: There appears to be no special pathological change, other than the gastro-enteritis, and kidney inflammation. The mouth and teeth may be affected.

Symptoms: *Acute Poisoning:* Corrosive sublimate, bichloride of mercury, is the most frequent cause of this form. It may be taken accidentally, or with suicidal intent.

The mucous membrane from the mouth to the intestines is corroded. There is a metallic taste. There may be vomiting. There is diarrhea with tenesmus. The urine is greatly diminished, there may even be complete suppression.

One case in my service at the Flower Hospital in 1905 came in with the following symptoms:—

The tongue and mouth were sore. The pupils dilated. The face flushed. The pulse was weak. The respirations were labored. There was a metallic taste and great thirst. A diarrhea, of green liquid stools, with cramps and great tenesmus. The next day the above symptoms continued, the stools every hour or oftener, the rectum and anus were excoriated. The pulse was 120, the temperature 102° F. Patient was up and about in nine days.

Another case gradually sank into a sort of cataleptic state. She could be roused to take nourishment, but for days lay in this apparently unconscious condition.

A third case occurred in my service in August, 1914. There was complete suppression of urine and frequent bloody stools. The patient died on the sixth day.

Chronic Poisoning: Mercury given in any form—by mouth by injection, by inunction, by inhalation— may cause mercurialization.

Stomatitis is one of the first symptoms of mercurialization. There is fetor of the breath. The gums ulcerate. The teeth become loosened. The hair falls out, the nails become brittle.

A fine tremor develops, which may become coarser. Convulsions and paralyses similar to those seen in hysteria may ensue Albuminuria and anasarca may occur.

Obscure cases of mercurialization may be due to amalgam fillings in the teeth. A number of such cases have been reported in the homœopathic literature. There is a question about some of them, but others are undoubtedly authentic. Some of the symptoms are stomatitis, pyorrhea, swollen glands in various parts of the body, and other symptoms enumerated above. Re-

moval of the amalgam fillings or rubber plates containing mercury is followed by relief. Dr. E. N. Chaney, of Los Angeles, reported several such cases in the *North American Journal of Homœopathy* for December, 1913.

Complications and Sequelæ: The complications and sequelæ appear in the symptomatology.

Diagnosis: The diagnosis must be made from the history.

Prognosis: The prognosis, as a rule, is good when the offending cause is removed in chronic cases. Many acute cases die.

Treatment: *Acute Poisoning:* Emetics come first, then give the patient quantities of milk or white of egg.

In the third case noted above Dr. Ralph A. Stewart decapsulated one kidney. It was much swollen and hyperemic. The patient passed an ounce of urine in the next twenty-four hours. He died sixty hours after operation.

Calendula and water may be used as a mouth wash.

Silicea or *hepar* in potency should be used.

Chronic Poisoning: If working in mercury this must be stopped. If the intoxication is due to the medicinal use of mercury this also must be stopped.

The patient must be put under the best hygienic surroundings. A nourishing diet must be instituted.

Chlorate of potash may be used for the stomatitis.

Iodide of potash is the mainstay of the old school.

Hering mentions ***hepar, kali iodide, nitric acid, aurum, carbo vegetabilis, cinchona, ferrum, belladonna,*** as antidotes.

PHOSPHORUS POISONING.

Definition: Phosphorus poisoning, the symptoms caused by inhalation of phosphorus fumes, or by the ingestion of phosphorus.

Etiology: The most frequent source of phosphorus poisoning is in match making. Matches are sometimes eaten with suicidal intent.

Morbid Anatomy and Pathology: The blood is dark and there are ecchymoses in the internal organs. The skin is jaundiced. Fatty degeneration is found in the heart, liver, kidneys and muscles.

There may be bone necrosis in chronic cases.

Symptoms: There is at first vomiting. A patient of mine, suicidal, at the Flower Hospital, vomited blood. After a day or two jaundice appears. There is abdominal pain. There may be diarrhea with cramps. The liver is enlarged. The pulse is rapid, the respirations increase, there is some fever. My patient had a pulse of 140, respirations 32, temperature 100° F. Ecchymoses appear in severe cases, and hemorrhages from the nose, kidneys, and even from the uterus. The urine is of low specific gravity, and contains albumin, sometimes blood. The patient is weak.

In chronic cases there may be necrosis of the bones. The lower jaw is first affected.

Complications and Sequelæ: Necrosis of the bones may occur.

Diagnosis: The diagnosis is made from the history.

Prognosis: The prognosis is always grave. My case recovered.

Treatment: The stomach must be emptied in acute cases.

The best antidote is oil of turpentine in mucilage.

Oily substances are inimical in acute phosphorus poisoning.

The homœopathic antidotes to all cases are ***nux vomica, coffea, terebinth.***

ERGOT POISONING.

Chronic ergot poisoning is not found often in this country.

The acute form is sometimes met with due to an overdose of the drug.

Etiology: Diseased grain eaten by the poor in certain parts of Europe causes chronic ergot poisoning.

Morbid Anatomy and Pathology: Ergot may cause gangrene of the extremities. Sometimes it causes changes in the posterior columns of the spinal cord similar to those of tabes. Ergot slows the heart and causes contraction of the arteries.

Symptoms: *Acute poisoning.* There are disturbances of digestion with vomiting and vertigo. There is slow pulse with weak heart. The patient becomes drowsy and may die.

Chronic poisoning. The digestive disturbances appear as in the acute form, but of lesser degree. The heart is weak.

Paresthesia is marked. There may be change in the men-

tality, even to convulsions. There may be change in the gait, incoördination like in locomotor ataxia.

Other cases present dry gangrene of the hands and feet. There is a line of demarcation, and sloughing of the part affected.

Treatment: In acute ergot poisoning emetics are indicated.

Ether, camphor, or black coffee may be used as antidotes.

In the chronic form the treatment must be symptomatic.

Camphor and *opium* may be used.

FOOD POISONING.

Definition: Symptoms of poisoning sometimes follow the ingestion of food. The bad effects may be due to: 1. Various infectious bacteria. 2. Changes in the food, as development of ptomaines, and putrefaction. 3. Idiosyncrasy against certain kinds of food on the part of certain individuals.

Etiology: Eating of the offending food causes the symptoms.

Morbid Anatomy and Pathology: This varies, of course, with the symptoms.

In poison from meats and fish there is gastro-enteritis. In fatal cases, often hemorrhagic in character, the spleen and kidneys may also be involved.

Ergot poisoning sometimes causes gangrene—sometimes causes changes in the posterior columns of the spinal cord similar to those found in tabes dorsalis.

Mushrooms cause fatty changes in various organs similar to those produced by phosphorus.

Symptoms: (1) Infectious germs in food eaten, if they set up any disturbance, cause some particular disease.

(2) Tainted meats and fish, sometimes ice cream, cause more or less severe stomach and intestinal symptoms. It usually takes a few hours—sometimes 24 or 36—for the symptoms to develop. There is nausea, vomiting, diarrhea. Ice cream caused these symptoms in 28 doctors and nurses at the Metropolitan Hospital, Sunday, Feb. 12, 1911. If the case goes on for a day or two, there is dizziness, wakefulness, even delirium, and changes in the pupils. The heart becomes rapid and weak. There may be collapse.

(3) Idiosyncrasy against certain foods like shell-fish, strawberries, etc., in some individuals causes mild gastro-intestinal symptoms. In others there is urticaria.

Complications and Sequelæ are as mentioned above.

Diagnosis: In the sudden onset of nausea, vomiting and diarrhea we have a group of symptoms that point to something eaten as the cause. This is especially true if a number of persons who have partaken of the same food are attacked at the same time.

Appendicitis may begin in a similar manner, a few hours will serve to develop a localized tenderness.

Prognosis: The prognosis must be guarded until the patient is well on the way to recovery. Relapses are frequent with meat poisoning.

Treatment: Prophylaxis consists in avoiding contaminated food.

The stomach may have to be emptied. A cathartic may help to remove the offending material. The patient should be in bed—and frequently is unable to be otherwise.

Tonics, as strychnine, are recommended by the old school.

The indicated homœopathic remedy should be used in all cases.

Ipecac is always to be thought of where there is nausea, vomiting and purging.

Pulsatilla is a remedy that follows *ipecac* well. It is especially useful for diarrhea after the vomiting has stopped.

Arsenicum album is useful in the general collapsed condition. There is incessant vomiting of all food and drink. There is restlessness.

Many other remedies may be thought of.

BERI-BERI.

Beri-beri is an Eastern word. Its etymology is unknown. Gould says it is of Cingalese origin and means weakness.

Synonym: Kakke (Japanese).

Definition: The most concise definition of beri-beri is Osler's: "An endemic and epidemic multiple neuritis of unknown eti-

ology, occurring in tropical and subtropical countries, characterized by motor and sensory paralysis and anasarca."

Historical Note: Beri-beri is mentioned in the oldest extant medical treatise, a Chinese work dating back to 2697 B. C. Other Chinese and Japanese writers have described the disease for the last two thousand years. The disease is called Kakke in Japan. Its modern study has practically dated from the time Japan was opened up to the world.

Etiology: Beri-beri is possibly an infectious disease, because a case introduced into a community frequently is followed by others. It prevails mostly along the seacoast and navigable rivers. It occurs only in tropical and subtropical countries, and more extensively in the hot and wet seasons than in cold or dry seasons. It is found most frequently among the Chinese, Japanese and Malays, although white races are not altogether exempt. It seems to be caused by unsanitary conditions and is apt to appear in persons on shipboard, or in crowded quarters.

The essential cause is unknown. Nitrogen starvation has been advocated. In the Japanese navy Takaki improved the diet and the disease rapidly diminished. Ingestion of fish and rice have been accused of being causative factors.

Manson and Daniels incline to the belief that pediculi, or possibly some other ectozoa, are responsible for its transmission.

During the last few years Japanese and American army surgeons in the far East have concluded that the use of polished rice causes beri-beri. The disease prevails mostly among rice-eating peoples. Feeding with unpolished rice has cured cases. Experimentally, beri-beri has been produced in animals by feeding them on polished rice, and they have been cured by feeding them on unpolished rice. The husk or shell of the rice seems to contain some element necessary to the nutrition of men or animals fed on a rice diet. The sale of polished rice is now forbidden in the Philippines.

Morbid Anatomy and Pathology: Rigor mortis develops early. The superficial veins and heart are filled with blood. The heart

is enlarged. The subcutaneous tissue and lungs are edematous. The muscles are atrophied.

The characteristic lesion, however, is a degeneration of the peripheral nerves to be observed only with the microscope.

Symptoms: Herzog gives three forms of beri-beri, besides mild cases that present malaise, weakness of the legs, and increased heart action on slight exertion. These symptoms may disappear, or may run into the severer forms.

1. *Acute pernicious beri-beri* begins, as a rule, suddenly. There is dyspnea and oppression in the chest. The least motion causes the heart to beat rapidly. There is vomiting. The legs are weak. The patient succumbs in a few hours to heart failure.

2. The *wet or edematous* form is preceded by malaise. There is loss of appetite. The heart's action is easily increased. The calves of the legs are tender on pressure. The patient has difficulty in walking, it is hard for him to lift his feet. There is edema of the legs and feet, and the skin pits on pressure.

3. In the *dry or atrophic form* the patient may have a slight edema of the legs and feet for a short time. This is followed by atrophy and contraction of the leg muscles.

In all forms there is disturbance of sensation. This occurs first in the lower extremities. In severe cases it may also occur in the upper extremities. There may be hyperesthesia, or there may be an approach to anesthesia. In severe cases the foot turns out as the muscles atrophy. We may have foot drop. If the upper extremities become involved we have similar disturbances of sensation in the arms, with possibly wrist drop.

Electrical reaction is diminished or lost. The nerves of the larynx are frequently affected and we have hoarseness or even aphonia. There may be absence of knee jerk.

The heart is enlarged, the apex is displaced to the right and upward. The slightest motion causes palpitation. There may be a systolic murmur. The second sound over the pulmonary valve is accentuated.

The urine is usually diminished.

In fatal cases death usually occurs from paralysis of the pneumogastric nerve.

Relapses are frequent. One attack predisposes to another.

Complications and Sequelæ: Dysentery and tuberculosis are the most frequent complications.

The heart is irritable and weak for a long time after recovery.

Sometimes deformities are left.

Diagnosis: Beri-beri is marked by the edema without albumin in the urine, the easily accelerated heart's action, pain in the muscles of the calf of the leg and atrophy of the muscles.

It must be distinguished chiefly from other forms of neuritis.

Prognosis: The prognosis is always doubtful owing to the constant possibility of heart failure. Relapses are frequent, and one attack predisposes to another. An increase in the flow of urine is a good sign.

The mortality, under good conditions, may be as low as two to six per cent. In some epidemics it has been as high as fifty per cent. or more.

Treatment: On general principles the patient should be isolated as far as possible. All clothing, bed clothing and utensils should be sterilized. Overcrowding should be avoided.

The patient should be put on a liberal diet. If a rice eater, only unpolished rice should be allowed.

The patient should be put to bed and kept there.

The heart may need stimulating, but *digitalis* is said not to be good.

Amyl nitrite, or *nitroglycerine* may be used for imminent heart failure.

Electricity may be used.

Various remedies will suggest themselves to the homœopathic physician. *Arsenic* or *apis* would be good drugs for the edema. *Strychnia phosphorica* for the nerves.

Elaterium is mentioned by Boericke. .

PELLAGRA.

(Italian, *pelle,* skin; *agra,* rough.)

Synonym: Italian Leprosy.

Definition: A disease presenting recurring attacks of an

eczematous like eruption on the hands, of gastro-enteric disturbance, and finally of loss of mentality.

Historical Note: Pellagra has been known in Italy since about 1740. As early as 1776 Venice passed laws against the use of spoiled corn, on the ground that spoiled corn in some way caused the disease. At present the disease is so widespread throughout Italy that twenty-two special hospitals for its treatment exist. Special diet kitchens have been established by the government to supply good food for the poor.

Pellagra also exists to a lesser degree in other parts of southern and eastern Europe.

An occasional sporadic case had been reported in the United States, half a dozen perhaps, up to 1908, when a paper was presented by J. W. Babcock showing that it existed in the State Hospital for the Insane at Columbia, South Carolina. Dr. Babcock concluded from his investigations that pellagra had probably existed in this hospital for nearly twenty years. Since his report was issued, attention has been called to the disease and several thousand cases have been reported, mostly from the southern and middle western States.

Etiology: The cause of pellagra is supposed to be the ingestion of spoiled corn. Just how or why is not known. Corn is eaten in large quantities in the United States, yet until 1908 there had not been enough cases of pellagra to call attention to the disease. The disease is marked by recurrent attacks of the eruption on the hands, and has some of the other characteristics of an infectious disease. May 15, 1910, the newspapers reported that Sambon, of England, had found that pellagra was transmitted by a species of gnat, the "similium repans."

The weak and poorly nourished are more susceptible. Dr. Minnie E. Dunlap, of the State Hospital at Lexington, Kentucky, in the *Medical Century* for August, 1912, says her patients ranged in age from 20 to 82 years.

Morbid Anatomy and Pathology: The pathological changes are found mostly in the nervous system. There is degeneration of the posterior columns of the spinal cord, of the cervical and upper part of the dorsal region, and the lateral columns in the

middle and lower parts of the dorsal region. Pigmentation of the anterior horns and of various organs occurs. The brain shows general wasting.

Symptoms: The cutaneous symptoms give the disease its name, *pelle,* skin; *agra,* rough. (1) *The dry type.* It appears first on the backs of the hands somewhat like sun-burn. Sometimes it appears also on the dorsum of the feet in those who go barefooted. It has a distinct outline and is always absolutely symmetrical. The affected skin becomes roughened and scaly, in two to three days, and sometimes fissured, and then after two or three weeks exfoliates. It usually appears first in the fall of the year, disappears after a time, and then reappears in the spring.

(2) The *wet type.* In severe cases bullæ may form, which break down and exude serum, pus, or blood, leaving a raw, ulcerating surface. The eruption may come and go through several successive years.

Dunlap says the wet type makes its appearance as a bright, dark red erythema, resembling a scald, with the same distinct symmetrical outline. In the course of twenty-four or forty-eight hours the outer layers of the dermis begin to dry and crack, and to ooze serum, pus and blood from the fissures. The eruption area is usually confined to the posterior cervical region, the hands, feet, elbows and in a small per cent. of cases to the perineal and perianal regions. About half of the cases die. The others run along four to six weeks and then desquamate and apparently recover. Most of them recur in two or three months. The absolute symmetry, Dunlap believes, points to lesions of the central nervous system.

Associated with the skin symptoms are marked digestive disturbances. There is anorexia, sometimes nausea and vomiting, together with morning diarrhea. The diarrhea may be uncontrollable and lead to death. There are also stomatitis, frequently going on to ulceration, accompanied by distressing salivation.

With the skin and digestive symptoms the patient is nervous and depressed. As the attacks recur the mental condition becomes worse and worse, until complete loss of mind results. There is loss of judgment, confusion and disorientation. There

may be depression or mania. The patient has delusions and hallucinations.

The spinal cord becomes affected and we have a gait simulating that of spastic paraplegia. Various paralyses result. Finally we have a complete physical, mental and nervous wreck.

Dunlap says: "The foul odor from pellagra is distinctive, and is described by the nurses as stifling. An odor which clings to the patient after a full bath and free use of deodorants. It emanates from breath, perspiration, exudation from fissures, from urine and feces, and is a fac-simile of the so-called insane odor."

There are two forms of the disease, acute and chronic. The acute runs its course in a few weeks and is usually fatal. The chronic comes on in the fall, subsides, resumes in the spring. This may rarely end in final recovery.

Complications and Sequelæ: The patient usually becomes hopelessly insane.

Diagnosis: The diagnosis where the disease is prevalent is not particularly difficult. The triad of symptoms: (1) The eruption on the backs of the hands, and sometimes of the feet; (2) the stomatitis, gastric and enteric symptoms; (3) the mental and nervous symptoms, are characteristic. This combination where the disease was not known to be prevalent might not be recognized as an entity and would be apt to cause confusion.

Pellagra has been called the disease with the three "Ds"—Dermatitis, Digestive disturbance, Dementia.

Prognosis: The prognosis is almost always bad, the patients usually becoming hopelessly insane and dying as the result of the profound mouth and gastro-intestinal disturbances.

Treatment: The treatment so far advocated has been wholly prophylactic, based on the supposition that spoiled corn in some way caused the disease. In Italy, the country most afflicted, there are twenty-two special institutions for the care of pellagra patients. As preventives, the government has assumed drastic measures in regulating the storage of corn and the grinding of meal from it. Places where the people are taught to prepare other food properly, and even where other food is given free, have been established by the government.

If the report proves true, that a gnat transmits the disease, other methods of prevention will have to be devised. Of course, the present supervision of the government can be maintained because it is all of value in improving the hygienic and dietetic condition of the poorest people.

The Thompson-McFadden Pellagra Commission, after two years' work, reported in 1914 that they believed the disease to be infectious. They had found no evidence that eating of corn caused it, or that gnats transmitted it. Rest, hygiene, and a liberal diet were all they had to recommend in the way of treatment.

The medicinal treatment is, under ordinary circumstances, a failure.

Dr. Dunlap reports results with *sulphur* 6x.

Arsenicum would seem to cover many of the symptoms. Dr. Dunlap has been disappointed in it.

Secale cornutum is another remedy that might be thought of.

Also *brassica napus* and *agrostema*.

WOOD ALCOHOL POISONING.

Definition: The effects produced by drinking or inhaling wood alcohol (methyl alcohol) or Columbian spirits.

Symptoms: The drinking of wood alcohol, as is well known, causes blindness and death. In 1914 H. H. Tyson and M. J. Schoenberg, of New York, were able to show that inhalation produced the same results. The subject is of importance because two million workers are employed where wood alcohol is used. Chronic poisoning frequently results eventually causing blindness.

Treatment: Is entirely prophylactic. Free ventilation and short working hours are necessary where wood alcohol is used.

SECTION VI.

Diseases of the Blood.

ANEMIA.

(Greek, αν, negative, αιμα, blood.)

Definition: Anemia means, in ordinary usage, not only diminution in the total quantity of blood, but also deterioration in quality.

It may be primary.

It may be secondary.

Primary anemia occurs in two forms: 1. *Chlorosis.* 2. *Idiopathic* or *Pernicious Anemia.*

CHLOROSIS.

(Greek, χλωρος, greenish yellow.)

Synonym: Green Sickness.

Definition: Chlorosis occurs in young girls. It is characterized by marked relative diminution of the hemoglobin of the blood.

Historical Note: Duncan, in 1867, was the first to show that the hemoglobin was much less than the percentage of decrease of the number of red corpuscles.

Etiology: The essential cause of chlorosis is unknown. It is most common in girls from 14 to 20, who are overworked and live under poor hygienic surroundings. Girls of the better class are not exempt.

Morbid Anatomy and Pathology: The liver and spleen are sometimes enlarged.

Blood: The principal pathological change is in the blood. A given drop of blood is paler than normal. The color index is low, the hemoglobin may be but 40 per cent. or less. There is also a decrease in the red cells, but not so marked—to 80 per cent. or thereabouts. The corpuscles are irregular in shape—

poikilocytosis. The amount of iron is diminished. There may be a slight leucocytosis. In severe cases there is lymphocytosis.

Symptoms: The general contour of the patient is not changed; the subcutaneous fat is retained, or even increased.

There is a peculiar greenish tinge to the complexion. Dermographism may be present.

The patient is apt to be short of breath on exertion. She has attacks of palpitation. The pulse is full and soft. A systolic murmur can usually be heard at the apex. Over the cervical veins one can hear the so-called "bruit de diable" with the stethoscope. There is said to be a tendency to thrombosis.

The patient is nervous, she may be hysterical. She cries over nothing, she is irritable. She cannot keep still. She may have headaches.

Amenorrhea, more often dysmenorrhea, is present. One of my patients complained of pain in the breasts and abdomen before, during and after menstruation.

Chlorotic girls are apt to be catarrhal subjects.

The appetite is capricious, there is frequently a longing for sour things. There is hyperacidity, mild indigestion, and constipation.

There may be slight fever at times.

Complications and Sequelæ have been mentioned.

Diagnosis: The diagnosis is usually easy. The patient is usually a well-nourished young woman with the peculiar greenish complexion, with very white or bluish sclerotics.

A drop of blood should be examined for the color index which is below normal.

Incipient tuberculosis and heart disease may be differentiated by the physical signs.

Bright's disease may be differentiated by the urinary findings.

Prognosis: The prognosis is good.

Treatment: The general hygiene must be improved as much as possible.

Iron is usually recommended; when used it is best given in the form of Blaud's pills.

Pulsatilla is the remedy that I have found most useful. It

covers the anemia, the indefinite nervous symptoms, the catarrhal symptoms, and the menstrual symptoms.

Calcarea carbonica is another remedy that is sometimes of value. The patient is "fat, fair, and flabby." The patient craves indigestible things.

Arsenicum album may be used for patients who tire very easily.

Ferrum, iron, in potency, may be used.

Natrum muriaticum and *calcarea phosphorica* may also be thought of.

PERNICIOUS ANEMIA.

Synonyms: Idiopathic Anemia. Progressive Anemia.

Definition: "A peculiar disease of the blood resulting from defective hematogenesis and excessive hematolysis, characterized by severe and usually fatal anemia, peculiar morphological changes in the red cells, and by characteristic changes in the bone marrow."

Historical Note: Cases of this disease were reported early in the last century. Channing, of Boston, in 1849, gave accurate clinical descriptions of it. Addison, in 1855, was the first to carefully differentiate pernicious anemia from other forms. Since that time many observers have reported their findings, gradually adding to knowledge of the subject. Cohnheim, in 1876, was the first to report on the changes that take place in the bone marrow.

Etiology: Pernicious anemia is more common in men than in women. It occurs during middle age. In women it may appear during or following pregnancy.

Occasional cases are due to intestinal parasites, to syphilis, to tuberculosis, to malaria, and to other very severe infections.

Morbid Anatomy and Pathology: There is a slight lemon color to the skin. Hemorrhages in the skin and serous membranes are common. The muscular tissue is intensely red. The heart is large and flabby. The internal organs are fatty. The liver may be enlarged and contain iron in excess.

The essential lesion is the change in the bone marrow, which shows a large proportion of megaloblasts and gigantoblasts.

Blood: The hemoglobin is reduced, but relatively increased. The red cells are very much reduced, they may be as low as 500,000. They are enlarged, vary in size and shape, and are nucleated. There is poikilocytosis; megalocytes and megaloblasts are present. The leucocytes may be normal.

Symptoms: Pernicious anemia may follow gastro-intestinal diseases, severe infections, or mental shock or worry.

It may develop without any known cause. Addison, in his description, says:—

"It makes its appearance in so slow and insidious a manner that the patient can hardly fix a date to the earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness in attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless, the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitations are produced by the most trifling exertion or emotion; some slight edema is probably perceived about the ankles; the debility becomes extreme—the patient can no longer rise from bed; the mind occasionally wanders; he falls into a prostrate and half torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking contrast to the failure and exhaustion observable in every other respect."

Hemic murmurs are constant. The arterial pulsation and palpitation may be annoying. There is air hunger—anemic dyspnea.

Hemorrhages into the skin and mucous membranes are common. There may be epistaxis and retinal hemorrhage.

Gastro-intestinal symptoms are common. There may be nausea, vomiting and diarrhea.

Some cases run an irregular fever.

The urine is of low specific gravity, usually dark or reddish in color.

Diagnosis: Pernicious anemia is characterized by the diminished number of red cells with the relative increase in hemoglobin. The blood is characteristic.

Prognosis: The prognosis is usually bad, the disease progressing to a fatal issue. Some cases are marked by apparent recovery, but relapse after a few weeks or months.

Treatment: Rest is essential. The underlying causes, if found, must be removed.

Fowler's solution sometimes helps. Begin with one drop, three times a day, and increase the dose till ten drops are given three times a day. If puffiness under the eyes appears diminish the dose.

Arsenicum album may be tried. It has excessive debility.

China may be of use in cases due to infections.

Phosphorus seems to cover the hemorrhagic conditions and the fatty changes in the liver.

Iron is said to be useless in pernicious anemia.

Splenectomy has been performed as a last resort in a few cases with at least temporary benefit. Coleman and Hartwell reported a case in 1914.

SECONDARY ANEMIA.

Definition: Secondary anemia is anemia due to loss of blood or vital fluids.

Etiology: Great loss of blood due to traumatism, childbirth or rupture of an artery from disease.

It may be due to prolonged lactation.

It may be due to excessive dissipation.

Anemia may result from disease, from lack of sufficient nourishment, from chronic lead poisoning or other chronic poisonings.

Blood: The hemoglobin and red cells are reduced according to

the severity of the case. The leucocytes are also proportionately reduced.

Prognosis: The prognosis is good if the cause is removed. The red cells increase very rapidly. They return to normal more quickly than the hemoglobin.

Treatment: The cause must be removed. Then the patient must be placed under good hygienic conditions, with nourishing food, plenty of fresh air, mental and physical rest.

In these cases iron is useful in the form of Blaud's pills.

China may be given in low potency. It is especially useful after hemorrhage or loss of vital fluids.

Arsenicum and *nux vomica* are useful following acute diseases.

LEUKEMIA.

(Greek, λευκός, white, αιμα, blood.)

Synonym: Leucocythemia.

Definition: Leukemia is a disease of the blood-making organs, characterized by an abnormal increase of the number of white corpuscles, together with enlargement of the spleen, lymphatics, and disease of the medullary substance of the bone.

There are different forms described, according to whether the spleen, lymphatics, or medullary substance of the bones show the greatest changes. These forms are essentially one disease, though they have been called *splenic leukemia, splenomedullary leukemia, myelemia, myelogenous leukemia, lymphatic leukemia.*

Historical Note: Leukemia was at one time considered to be a suppurative disease. In 1841 Craigie and Bennett both reported cases with excess of white cells in the blood, but which they supposed to be pus cells. Each considered the enlarged spleen to play some part in the disease. Each declared no suppurating foci discoverable. Bennett later, 1851, accurately described the disease. Virchow, during the same decade, named the condition "weisses blut"—white blood or leukemia. He showed the connection of the lymphatics with leukemia. Neumann in 1869, first described the lesions of the marrow.

Etiology: Leukemia may appear at any age and in either sex.

It is more frequent in middle life. It occurs in the proportion of two males to one female.

Heredity appears to play a part.

Trauma, especially of the spleen or bones, has acted as an exciting cause.

Malaria has been found to have formerly occurred in about one-third of the cases.

Pregnancy sometimes acts as an exciting cause.

Morbid Anatomy and Pathology: The most constant change is enlarged spleen, a chronic hyperplasia. The organ is firmer than normal. There may be hemorrhagic infarcts.

The bone marrow is changed. It is light in color. This also undergoes a hyperplasia.

The lymph nodes are enlarged.

Blood: The quantity of blood is normal. It is paler than usual. It coagulates slowly. The red cells are diminished to 3,000,000, or less, even to 1,000,000. There is poikilocytosis. Normoblasts are numerous. The hemoglobin is reduced.

The white cells are enormously increased. In mild cases they run as high as 200,000, while in severe cases they may run as high as 1,000,000. The proportion of polynuclear neutrophiles is much increased. The eosinophiles and lymphocytes likewise. Neutrophile myelocytes are pathognomonic.

The findings may vary considerably from time to time.

In a case in my service at the Flower Hospital, in 1909, the hemoglobin showed 60 per cent. The red cells 2,800,000, white cells 248,000. Polyneutrophiles 49 per cent., myelocytes 31 per cent., lymphocytes 13 per cent., eosinophiles 7 per cent. Poikilocytosis was marked. Normoblasts and megaloblasts were both present.

In another fatal case the red cells dropped to 800,000, while the whites were 200,000.

Symptoms: The onset of leukemia is usually insidious. The patient has the disease well-marked when first seen.

There may be pallor of the skin. Epistaxis is frequent, other hemorrhages sometimes occur. There may be retinal hemorrhage. Purpuric spots may appear.

There is slight dyspnea. The pulse is rapid and soft. There is headache with dizziness and faintness. There may be tinnitus, even deafness.

The abdomen is enlarged. Examination will disclose a much enlarged spleen. Its growth is gradual, but it becomes distinctly palpable. Frequently it is tender, and the patient complains of pain in it. The lymphatic glands also are usually enlarged.

In men priapism is a troublesome feature. Strümpell says that is sometimes the first symptom.

In a case presenting the above symptoms the blood should be examined. If the blood findings are as already described the case is clear.

Complications and Sequelæ: Hemorrhages from different parts may cause complications.

Diagnosis: The blood findings are diagnostic. Occasionally some intercurrent infection may change the character of the blood to a certain extent.

Prognosis: The prognosis is bad. It is usually fatal in two to three years.

Treatment: Rest, fresh air and a nourishing diet are all indicated.

At Johns Hopkins some cases of leukemia were recently found in benzol workers. Since then a number of cures have been reported by the use of benzol as a remedy. Dr. J. P. Cobb, of Chicago, reported a case in 1914, at the American Institute of Homœopathy.

Arsenic, in large doses, is recommended. *Iron* is also said to do good.

Homœopathically, *arsenicum album* seems to cover most of the symptoms.

Picric acid has priapism and great exhaustion as marked symptoms.

PSEUDO-LEUKEMIA.

(Greek, ψευδης, false.)

Synonyms: Hodgkin's Disease. Adenia. Lymphadenoma.

Definition: Pseudo-leukemia, or Hodgkin's Disease, is a pro-

gressive hyperplasia of lymph glands, with anemia, and secondary lymphoid growths in the spleen, liver, etc.

Historical Note: Although similar cases had been described before, Hodgkin, in 1832, was the first to clearly describe cases of the disease, which now bears his name. According to Ewing, Wunderlich, in 1858, gave the first complete description of the malady and noted its idiopathic origin.

Etiology: The disease has sometimes been supposed to be of infectious origin. Tuberculosis is the cause of the majority of cases according to Ewing. Osler says that this is not proven. Syphilis and the pyogenic organisms may cause it.

It occurs mostly in young persons, and males are more often affected than females.

Morbid Anatomy and Pathology: The lymph nodes are enlarged, hard and firm. At first isolated and movable, they later become fused and may form a large mass. In one case of mine at the Flower Hospital the masses in the cervical region were so huge that the face was almost pyramid in shape. The swollen glands, on section, are white, smooth, and of varying consistency. They form first chains, then groups. The cervical glands are affected first, then the axillary, then the retroperitoneal, then other glands. Ulceration is rare. The spleen is usually very much enlarged.

Blood: The hemoglobin is reduced. The red cells grow less as the disease progresses. The leucocytes are increased, but rarely go above 50,000 or 60,000. Other changes are not characteristic.

Symptoms: The characteristic of the disease is the enlarged lymph nodes, they are palpable, even visible, in the neck, axilla and groin. In my case the mass in the neck on both sides was enormous. The tumors, by pressure, may cause dyspnea and cough, when they surround the trachea. There may be pain about the heart with palpitation. Hemic murmurs are common.

The spleen is usually enlarged.

There may be fever. There is usually headache, giddiness, tinnitus; there may even be delirium.

Bronzed spots may appear on the skin, as in my case. Ulceration is said sometimes to take place through the skin.

Diagnosis: Tubercular glands and sarcoma may have to be differentiated by the microscope. In pseudo-leukemia there is a proliferation of the endothelial and reticular cells.

Tubercular adenitis gives the skin reaction to tuberculosis.

Prognosis: Pseudo-leukemia is usually very chronic, lasting several years. It is nearly always fatal. A sudden exacerbation may carry the patient off in a short time.

Treatment: Early in the disease excision may stop the progress of the disease.

Arsenic in the form of Fowler's solution is recommended by old school authorities.

My case was benefited for a time with *phytolacca.*

Arsenicum iodide, phosphorus and *iodium* are mentioned by Boericke.

PURPURA.

(Greek, πορφύρα ; Latin, *purpura,* purple.)

Definition: Purpura is a disease characterized by hemorrhages into the skin and mucous membranes. The appearance of the skin after such hemorrhages have occurred gives the disease its name.

Introductory Note: Purpuric spots are sometimes secondary to certain other diseases, as in severe forms of the eruptive fevers. In such cases it is probably an indication of a deterioration of the blood. Purpuric spots also occur at times in advanced cases of Bright's disease. They are said to occur in phthisis, although in the many thousands of tuberculous patients that I have seen I have never met them. I have seen them in rheumatism and pneumonia. Some of the snake poisons and some drugs also produce these hemorrhagic extravasations. Poorly nourished persons who have been the victims of fleas, pediculosis or other vermin may have purpuric spots as a result of the bites.

Etiology: The exact cause of purpura is not known. The disease may appear suddenly in persons who seem to be perfectly healthy, it may appear in those who are anemic and ill-nourished. All ages and both sexes may be affected, although it is said that women are more often attacked than men, and that the disease is found with greater frequency between the ages of twenty and

thirty. As purpura sometimes follows severe mental shocks it is supposed that the vaso-motor system controls the hemorrhages.

The affection is not common. Personally, I have seen but a few cases, all of them hospital cases except two. At the Metropolitan Hospital of 57,242 patients treated from 1889 to 1898 inclusive, but two were cases of purpura. Even the purpuric spots found in the aged and decrepid were rare.

Notwithstanding cases of purpura are so rare the disease is not difficult to recognize if a case has once been seen. The spontaneous appearance of the extravasations is very alarming to the uninitiated.

Morbid Anatomy and Pathology: The pathology of purpura is obscure. The extravasations are spontaneous; by some they are supposed to be a simple diapedesis of the red corpuscles. Whether due primarily to change in the blood or in the blood vessels is not definitely known. Ziegler says that in spontaneous hemorrhages the endothelial lining of the capillaries allows the corpuscles to pass through, that the fault is in the vessels, not in the blood.

On account of its resemblance to scurvy (it has been called land scurvy), and also because of the appearance of purpuric spots in the course of, or following, the eruptive fevers, in blood poisoning and in other infectious diseases, purpura has been classed by some as a germ disease. No specific organism has yet been found.

Symptoms: The eruption of purpura is a spontaneous extravasation of blood into the connective tissue of the skin, forming ecchymosed spots exactly similar to those found after contusions, the ordinary black and blue spots. Very slight injuries to the skin of purpuric patients will produce extensive ecchymoses. This may be of medico-legal importance, for patients suffering from purpura have endeavored to have the disease form the basis of a suit for both civil and criminal proceedings. The eruption is usually smooth, rarely elevated. As a rule, it is neither sore to the touch nor does it produce any subjective symptoms. When the spots first appear they may be either bright red or they may be more of a purple color. They appear suddenly in successive crops and gradually fade away. They do not coalesce but retain their

original size. The ecchymoses vary in size from pin points to extravasations, as large as the hand or larger, and are irregular in outlines. They may be raised like urticaria. They are apt to be large wherever there is pressure. One of my patients at the Metropolitan Hospital, a bed patient, had larger ecchymoses on the back at the shoulders and buttocks, presumably from the weight of the body. Being a true ecchymosis purpura does not disappear on pressure. When the spots begin to disappear the color first grows darker, then turns to a dirty yellow, and finally disappears altogether. Many of the larger spots fade from the center to the periphery.

In the severer forms of the disease the ecchymoses appear on the mucous membranes as well as on the skin. I have seen them on the hard and soft palate, on the gums, and on the glans penis. There may be free hemorrhage from the mucous membranes in some cases. Hemorrhages have also occurred in the serous cavities.

Varieties: Purpura may be divided into three forms: (1) *Purpura simplex;* (2) *Purpura hemorrhagica,* and (3) *Purpura rheumatica.* These conditions merge into each other so that it is sometimes difficult to exactly assign a given case.

1. In *purpura simplex* the extravasations of blood are limited to the skin. There are no systemic disturbances or they are very slight, as lassitude and some loss of appetite.

2. *Purpura hemorrhagica,* the *morbus maculoses Werlhofii* of the Germans, is much more severe than the preceding form. It is distinguished by extravasations into the mucous membranes as well as into the skin. Hemorrhages may occur from the various organs, and we may have hematemesis, hemoptysis or hematuria. Albumin may be found in the urine of some cases. This form of the disease usually presents more or less constitutional disturbance; there is fever, sometimes delirium. Anemia usually becomes a marked symptom. Occasionally a spot will break down and ulcerate.

3. *Purpura rheumatica, peliosis rheumatica,* or *Schönlein's disease,* is a form of purpura distinguished by symptoms of acute rheumatism. There is swelling of the joints, pain in the joints,

fever sometimes followed by endocarditis. I have seen three cases of this form, one in my service at the Metropolitan Hospital in 1898, one in my service at the Flower Hospital in 1911. This last patient had hemorrhages in the mucous membranes of the mouth with superficial ulcers on the tongue and lips. The third case was a girl of seventeen, who had had acute articular rheumatism three times. She developed purpura rheumatica in February, 1914. She has been my patient for fifteen years.

Blood. Examination of the blood in purpura shows a progressive anemia. There is diminution of the red corpuscles, sometimes to below a million. There is a diminished hemoglobin. Usually there is a slight leucocytosis.

Complications and Sequelæ: Anemia is usual. Hemorrhages may occur from the surfaces of the body, from the lungs, and from the stomach, or, as in one of my cases, from the nose and from the kidneys (hematuria). Edema was also a marked symptom in one case, especially of the face, scrotum and penis. It came and went; muscular pains were present.

Diagnosis: The disease most closely resembling purpura is scorbutus. A previous history of lack of proper food, the spongy gums, and the development of the case should make a diagnosis of scorbutus clear.

Some of the erythematous skin diseases may show eruptions similar to those of purpura, but the subsequent course of the disease will prevent any error being made.

The purpura eruption appears in successive crops, the fresh spots of to-day looking a little faded to-morrow and disappearing entirely after a few days.

Prognosis: In patients suffering from other diseases, with a few purpuric spots here and there, the prognosis depends on the original disease.

In the graver forms of purpura as a disease entity the prognosis is doubtful. One case that I saw as an interne at Ward's Island died. My other cases have all recovered.

Treatment: The patient with severe purpura should be kept in bed. The diet should be light and nourishing.

The old school text-books mention sulphuric acid. Osler rec-

ommends Fowler's solution of arsenic pushed till its physiological effects are seen. Ergot is also recommended.

Homœopathically my preference is for *arnica*. It acts directly on the vaso-motor system, thus controlling the capillary vessels. It produces ecchymoses into the skin and mucous membranes.

Apis is the remedy if local edemas are present. It finally cured a very severe case at the Metropolitan when prescribed symptomatically. It is not referred to by the authorities at my disposal.

Mercurius solubilis cured my Flower Hospital case with ulcerative stomatitis.

Among other drugs that may be thought of are *crotalus, hamamelis, phosphorus.*

The totality of the symptoms must be prescribed for, not the ecchymoses alone. These latter tend to disappear anyway.

HEMOPHILIA.

Synonyms: Bleeder. Hemorrhagic Diathesis.

Definition: A condition in which the blood does not properly coagulate, usually congenital.

Historical Note: Bleeders have been recognized in all ages. Osler says American physicians were the first to accurately study hemophilia and to recognize its hereditary character. The disease has been exhaustively studied during the last century in this country and abroad. Sir Almroth E. Wright, in Allbutt's System of Medicine, has an extensive article on the subject.

Etiology: There are several curious facts connected with hemophilia. First, it always occurs in males (Wright). Other authorities state that the proportion affected between the sexes is about 12 males to 1 female. Second, the disease occurs in families known as "bleeder families" and mostly among the male offspring of mothers who belong to such families. "Bleeder" fathers and normal mothers rarely transmit it. Normal fathers and mothers from "bleeder" families do transmit it. Some of the children may not develop it.

Statistics are on record showing cases appearing through as

many as seven generations, and extending over a period of two hundred years.

The disease practically always manifests itself very early in life, more than ninety per cent. of the cases show before the end of the second year.

Other than this the true cause of the disease is unknown.

Morbid Anatomy and Pathology: Wright believes the main fault to lie in the blood. He has shown that whereas in normal persons the blood coagulates in about five minutes, in bleeders it varies from ten to seventy minutes. He likewise shows that the number of leucocytes is subnormal, especially the polymorphonuclear leucocytes.

There is nothing further known of the pathology.

Symptoms: The most characteristic symptom is severe hemorrhage on the slightest provocation. Strümpell questions if hemorrhages ever occur without some external cause, although he acknowledges that it is not always possible to find the cause.

Another factor is the difficulty of stopping the hemorrhage when it has once begun. It is sometimes impossible and the patient bleeds to death. I lost one infant, twenty-four hours old, from hemorrhage around the base of the cord, not through it. Another mild case was a young woman who bled for two days after extraction of a tooth. The hemorrhage finally ceased.

Wright says frequently the first sign that a child is to be a bleeder is the appearance of hematoma.

The circumcision of Hebrew children reveals the presence of hemophilia early when it occurs in that race.

Articular effusions are of comparative frequency in bleeders.

Spontaneous (?) hemorrhages occur from the mucous membranes, of which the most common is epistaxis. The bleeding may continue for hours or days, causing the patient to have a profound anemia.

Slight traumatisms start the bleeding. Surgical operations are out of the question, for the patient is liable to bleed to death. Early dental caries is common. Some patients have a depraved appetite—a systemic cry for *calcium*. Wright devotes a special paragraph to women of "bleeder" families—he denies the exist-

ence of true hemophilia in females. He says they never show hematoma, and rarely suffer from immoderate hemorrhages, nor do they lose an excessive amount of blood at the menstrual flow or after confinement. Exceptions occur, however, but they are rare. They are exceptionally fertile. The blood of bleeder women shows the characteristic delayed coagulability and diminished number of leucocytes.

Diagnosis: The diagnosis depends on: 1, The family history; 2. repeated uncontrollable hemorrhages either without known cause or on excessively slight traumatisms; 3. the ecchymoses usually have a slight swelling.

Prognosis: Many cases die early in life. There is always the possibility of bleeding to death. As the patient grows older, however, the coagulability of the blood increases and many patients live to an advanced age.

Treatment: Calcium lactate in 15 to 20 grain doses has been recommended. If operation is necessary, dessicated thyroid extract, 3 grains after meals, may be given for a day or two beforehand.

Fresh blood serum, 10 to 20 c.c., may be injected directly into the blood in desperate cases. Human blood is preferable. Horse serum or, barring that, diphtheria antitoxin may be used.

I saw all these things tried, together with nasal tampons on a man with epistaxis, and notwithstanding he bled to death.

Of homœopathic remedies, *ipecac* may be used for bright hemorrhages.

Hamamelis applied directly to the gums seemed to have a hemostatic effect in my patient who bled from the gums.

SECTION VII.

Diseases of the Heart and Blood Vessels.

PERICARDITIS.

(Greek, περι, around; καρδια, the heart.)

Definition: Inflammation of the pericardium.

Etiology: Like endocarditis, pericarditis is most often secondary to some other condition, although rarely it may be primary.

It most often follows or is a complication of acute articular rheumatism or scarlet fever. Less often it may be associated with other infectious diseases. It may occur in septic conditions. It may form a complication in gout, Bright's disease or tuberculosis. It may be an extension of a pleurisy or pneumonia.

It may be traumatic when it becomes a surgical condition.

It may occur at any age, but is most often found in young and middle aged adults.

Bacteriology: In the purulent form pus organisms are found. In other cases tubercle bacilli, pneumococci or whatever may have caused the condition are present.

Morbid Anatomy and Pathology: There are various forms of pericarditis. 1. Dry pericarditis. 2. Pericarditis with effusion—sero-fibrinous, fibrinous, purulent or hemorrhagic.

In the beginning we have a dry pericarditis;—the layers may be found to be more or less adherent, or there may be a certain amount of fibrinous material between them.

Later we have pericarditis with effusion; the enclosed liquid may be considerable in quantity and sero-fibrinous, purulent or hemorrhagic. The hemorrhagic form is found chiefly in tuberculosis.

In cases with much effusion the pericardial layers become much thickened. The myocardium also is frequently affected. Endocarditis is often found.

Symptoms: The ordinary form of pericarditis may have no symptoms and may be unsuspected. In other cases there is pain, sometimes very acute, and tenderness over the lower part of the sternum.

Frequently there is an irregular fever of 102° or 103° F.

Dyspnea is a more or less pronounced symptom. This may become very severe.

The pulse becomes irregular and hard.

The face is somewhat cyanotic and the breathing rapid and irregular.

These symptoms should call attention to the chest.

Physical Signs: *Inspection.* In the first stage, before effusion takes place, inspection reveals nothing.

If there is much effusion the precordium may bulge and the interspaces be filled out. The left lung is encroached upon so that expansion on that side is diminished.

Palpation. A friction rub is sometimes felt before the stage of effusion. This may still be felt at the base of the heart even after effusion takes place. The apex beat becomes feeble or lost.

Percussion reveals an enlarged area of cardiac dulness due to the effusion. It may be roughly pyramidal in shape with the apex upward. It extends to the right fifth intercostal spaces (Rotch's sign).

In large effusions the area of dulness may vary according to the position of the body.

Auscultation. The friction rub heard with the heart beat is pathognomonic. It may be accentuated by having the patient hold his breath and using a little pressure on the stethoscope. After the stage of effusion the friction sound is lost; it persists longest at the base. Then we get weak heart sounds or none at all, but with the increased area of cardiac dulness.

Complications and Sequelæ: Occasionally acute pericarditis becomes chronic. In cases with effusion the fluid may be absorbed leaving adherent pericardia. In severe cases the myocardium becomes affected and we finally get all the phenomena of failing compensation.

Diagnosis: The diagnosis must be made from the physical signs.

Prognosis: The prognosis is usually good if uncomplicated.

Treatment: Absolute rest in bed is the first and most essential thing.

The ice bag is recommended.

Strümpell advises *digitalis* in weak pulse.

The indicated homœopathic remedy should be used.

Apis is useful in cases of effusion.

Aconite is useful in dry pericarditis, especially if the peculiar fear of death of that remedy is present.

Bryonia is to be thought of if the friction sounds are prominent.

Spigelia if there is much precordial pain.

Other remedies are *colchicum, arsenic,* etc.

Other Forms of Pericarditis.

Adhesive pericarditis: This may be the final result of simple pericarditis. It may exist without symptoms, being discovered only post-mortem.

In other cases there may be hypertrophy and dilatation of the heart. In a few cases chronic peritonitis, perihepatitis and perisplenitis may co-exist.

The leading *physical sign* is a retraction of some part of the chest wall at systole. This may be seen in front at the seventh or eighth rib, at the left toward the back between the eleventh and twelfth ribs.

Hydropericardium: Dropsy of the pericardium found usually in cases associated with dropsy elsewhere.

Hemopericardium: Blood in the pericardium. Usually the result of aneurism, sometimes of injury.

Pneumopericardium: Due to accident; runs into pyopneumothorax, etc.

ACUTE ENDOCARDITIS.

(Greek, ἔνδον, within; καρδια, the heart.)

Definition: Acute endocarditis is an acute inflammation of the lining membrane of the heart. It usually attacks the valves.

NOTE.—This section and the one on *chronic endocarditis* should be read together.

Etiology: In the great majority of cases endocarditis is secondary to some other condition, particularly acute rheumatic fever. It may be a complication in any of the infectious diseases. Scarlet fever is the most frequent offender, perhaps, next to rheumatic fever. Tonsilitis, now looked upon as a rheumatic affection, may also be associated with endocarditis. Influenza, pneumonia, tuberculosis, chorea (a rheumatic affection), more rarely measles and the other infections cause it. A most malignant type of endocarditis occasionally follows gonorrhea. Septic conditions, as erysipelas, purpura hemorrhagica, and puerperal fever, may be complicated with endocarditis.

Osler says the endocarditis of pneumonia is particularly malignant.

An acute endocarditis associated with any of the above conditions may be grafted on a chronic condition.

Bacteriology: According to Strümpell the staphylococcus is frequently found, less often the streptococcus, and rarely the specific organism of the associated infection, as the pneumococcus or the gonococcus. The gonococcus was found in one of my Metropolitan Hospital cases.

Morbid Anatomy and Pathology: There are two forms of endocarditis: (1) *Verrucosa,* the formation of nodules, and (2), *ulcerosa,* ulceration of the tissues. This latter may be called *malignant* or *infectious.* The two forms may run into each other. The valves of the left side of the heart are most often affected—the mitral and the aortic.

The nodules may be very small, or they may be quite large. They are usually covered with a layer of fibrin and leucocytes. The ulcers may form on the edges of the valves only, or the valves may be completely perforated.

The diseased tissue in either case may break off forming emboli. These may lodge in any organ of the body—kidneys, spleen, intestines, brain. Thus we may have infarct of any of these organs. The infarcts may go on to abscess formation.

Symptoms: The symptomatology of acute endocarditis is not a definite entity. The diagnosis can be made only from the physical signs. Many cases are so mild as to escape detection

otherwise. In septic conditions or in infectious diseases the heart must be closely observed. At the onset of endocarditis there may be a slight rise of temperature, or there may be, usually is, an irregularity of pulse. Any slight exertion may cause an acceleration of the pulse.

In the *malignant form* the patient runs quite a high fever, as a rule. This may be intermittent, it may be septic, it may be continuous. There is profuse sweating. The patient may become delirious, or he may sink into coma. In cases that are prolonged there may be loss of weight and general debility extending over weeks. There may be metastatic abscesses and hemorrhages into the skin and organs.

Embolic processes may develop suddenly in various parts of the body. Hemiplegia may occur. I had one such case at the Flower Hospital.

Physical Signs: The *physical signs* may be somewhat uncertain. The apex may be to the left. I have learned to look with dread on a heart where the sounds are separated by equal pauses, that is, the interval between the second and first sounds is as short as that between the first and second. This bodes ill in the majority of cases.

Murmurs may or may not be heard. The most common is a soft blowing sound at the apex of limited area of transmission. There may be accentuation of the second aortic sound.

Complications and Sequelæ: Acute endocarditis most often runs into chronic endocarditis with permanent valvular changes.

There may be embolic processes, the results varying with the part affected.

Diagnosis: In the acute infections endocarditis if severe enough to produce symptoms may usually be diagnosed by the physical signs. If the heart dulness is increased to the left it may be taken for granted that endocarditis exists even without an audible murmur.

In the malignant or ulcerative type the diagnosis may be very difficult. It has been confused with typhoid fever, miliary tuberculosis, meningitis and various other conditions. A clear history of the case will help to clear it up. The presence of

embolism or hemorrhage or both will be additional evidence in favor of malignant endocarditis. The type of fever is septic and may range from normal to 103° or 105° F. even.

I have seen a number of fatal cases of malignant endocarditis where the fever was markedly septic, there were chills and night sweats, and emaciation. Hemorrhagic infarcts into the lungs, as evidenced by cough and hemoptysis; infarct into the spleen, as evidenced by sudden acute pain in the spleen; infarct into kidneys, as evidenced by pain like renal colic, and possibly hematuria. The diagnosis in most of them was verified at the autopsy. In other cases autopsy was refused.

Prognosis: The immediate prognosis of simple endocarditis is good. It tends to run into the chronic form. Cases may recover. Severe forms are always doubtful as to the outcome. The septic form is almost invariably fatal.

Treatment: The first requisite is absolute rest.

Application of an ice bag sometimes does good.

In cases of impending heart failure stimulation may be desirable. Strümpell recommends *digitalis, strophanthus* or *camphor.* Anders says *digitalis* should not be used if the myocardium is affected.

Camphor is one of the best of heart stimulants.

Aconite is a most excellent remedy in the beginning of endocarditis. It has an anxious fear of death.

Spigelia is a remedy mentioned especially by Raue. This follows *aconite* acceptably.

Iodium, if *spigelia* fails. Particularly useful in the endocarditis of rheumatism.

Aurum in rheumatic conditions.

Cactus grandiflorus has a feeling of constriction about the heart.

Spongia—cannot lie down, pain about heart.

CHRONIC ENDOCARDITIS.

Synonym: Valvular Disease of the Heart.

Definition: A permanent change in the structure of the valves of the heart is known as chronic endocarditis.

[NOTE. This section and the one on *acute endocarditis* should be read together.]

Etiology: The most frequent cause of chronic valvular lesions is an antecedent acute endocarditis. An occasional case is supposed to be chronic from the start. The majority of cases will give a history of rheumatic fever or of some other infectious disease in the more or less remote past. The valvular trouble probably dates from that time, though the heart symptoms may not have been pronounced enough to attract attention until months or years afterwards.

Other causes of chronic endocarditis are arteriosclerosis, gout, syphilis and chronic alcoholism. In women it sometimes dates from childbirth.

Again we often find valvular lesions in association with chronic nephritis. In some of these cases the heart lesion seems to develop first, in others the kidney lesion seems to develop first. I agree with Strümpell that "it is not always easy to determine whether both conditions are related in the way of cause and effect, or whether they are both secondary, and both alike the result of some third unfavorable influence."

Finally physical exertion by constant overstraining may produce valvular lesions, or some sudden great effort may produce them.

All ages may be affected. Most of the cases occur or originate in early adult or middle life.

Morbid Anatomy and Pathology: There may be vegetations or other deposits on the valves, or there may be shortening of the chordæ tendinæ, in either of which conditions the valves cannot close properly. This is called *insufficiency*. The blood flows backward through the imperfectly closed valves—*regurgitation*.

On the other hand, there may be deposits or adhesions, which keep the valves from opening fully, so that the blood is forced through a narrowed opening—*stenosis*.

We may have both *insufficiency* and *stenosis* in the same valve. As a rule, *stenosis* rarely occurs alone. *Insufficiency* frequently does.

As a result of these conditions the heart has to make a greater

effort to keep up the circulation. This causes hypertrophy of the heart muscle. This is known as *"compensation."*

When the heart finally reaches the stage where it can no longer overcome the extra burden we have the stage of *"failing compensation."* This causes stasis of the blood current, and as a result imperfect aeration.

It is now necessary to discuss each valve separately.

1. Mitral Regurgitation: *Synonyms:* Mitral Insufficiency. Mitral Incompetency.

The mitral valve fails to close properly because of contraction of its edges, or of shortening of the chordæ tendinæ, or rarely of adhesion to the walls of the ventricle.

This is the most common of all the valvular lesions. In such a heart contraction of the left ventricle forces some of the blood back through the impaired mitral valve into the left auricle. As a consequence greater force must be exerted to send a sufficient amount of blood into the aorta. The back pressure into the left auricle causes dilatation and possibly hypertrophy of its walls. The extra work required of the left ventricle causes some dilatation of the cavity and always hypertrophy of its walls.

Later on the back pressure in the pulmonary circulation may throw extra work on the right ventricle and thus cause hypertrophy of that side of the heart.

Still later the circulation throughout the body becomes impaired. Venous stasis sets in. The patient becomes cyanotic. Edema of the face and extremities occurs, passive congestion of the various organs occurs, then general edema, and we have all the symptoms of a failing heart.

Physical Signs: *Inspection* shows an increased heart impulse in well developed mitral regurgitation. The apex beat is seen low and to the left. There may be an epigastric impulse as well.

Palpation will reveal the same condition. Sometimes a systolic thrill can be felt.

The pulse shows no distinctive characteristics.

Percussion at first gives an increased area of heart dulness to the left. Later it may give it to the right as well.

Auscultation usually gives a loud blowing sound at the apex

in place of the first sound of the heart—a systolic murmur. Sometimes the first sound is heard with this blow. The second pulmonic sound is usually accentuated. This murmur tends to be transmitted to the left. In some cases it is heard best at the left second intercostal space. I have noticed this in quite a number of cases, especially in association with tuberculosis.

Anders says mitral regurgitation may sometimes be heard when the patient is lying down and not when he is standing.

Late in the disease there is a dilatation of the right ventricle causing a relative tricuspid insufficiency, heard best over the ensiform cartilage.

2. Mitral Stenosis: This is a narrowing of the mitral valve due to thickening or adhesion of the segments. As a consequence the flow of blood into the left ventricle is retarded. It may occur alone, but is often due to or associated with insufficiency.

The first effect of stenosis of the mitral valve is enlargement and hypertrophy of the left auricle. The left ventricle not having the usual amount of blood to pump through becomes smaller than normal unless it has first become hypertrophied on account of a preceding insufficiency. After enlargement and hypertrophy of the left auricle comes a marked hypertrophy of the walls of the right ventricle due to the increased amount of work thrown back on it.

While the heart's action is slow the patient is comparatively comfortable, but when the heart's action becomes rapid the left ventricle does not have time to become properly filled and the pulse is weak. As the walls of the heart do not receive a sufficient supply of blood its action becomes extremely irregular.

Later in the disease the systemic disturbances are much like those of late mitral regurgitation. We are not so apt, however, to have general edema.

Physical Signs: *Inspection* shows a slightly increased area of and unusually prominent heart action. There is marked epigastric pulsation, and there is jugular pulsation. The apex beat is not displaced.

Palpation gives the increased area of the heart's action. It may be felt to the right of the sternum. There is also usually

felt a diastolic thrill that is pathognomonic. Strümpell says it may be felt at the apex. Anders says it is best felt over the "third and fourth (less frequently the fifth) interspaces." The radial pulse is small and may be irregular.

Percussion gives an increased area of heart dulness to the right, sometimes to a considerable distance beyond the sternum. Frequently there is increased dulness to the left as well. There is also an increased area of dulness at the base due to the enlarged auricle.

Auscultation gives a presystolic or diastolic murmur at the apex. It is not transmitted over any great area. It is sometimes heard best a little to the right or to the left of the apex. It is advisable to palpate the heart at the same time in order to be sure that the murmur is diastolic.

In very bad cases the opening in the valve may be so small that there is not force enough to the blood current to produce an audible murmur. The pulmonic second sound is accentuated.

In uncomplicated stenosis of the mitral valve the first sound of the heart at the apex is markedly clear and distinct.

As the disease progresses we may get a mitral regurgitation, and later a tricuspid insufficiency.

Mitral stenosis is one of the severer forms of heart disease. It is subject to exacerbations and remissions, and may be amenable to treatment for a long time.

3. Aortic Regurgitation: *Synonyms:* Aortic Insufficiency; Aortic Incompetency.

Atheroma and syphilis cause this form of heart disease more often than any other.

The aortic valve fails to close properly most often because of contraction of the valves, less often because of adhesions. Sometimes a rupture is caused by violent physical exertion: ***athlete's heart.***

As a result of this condition blood flows back from the aorta through the damaged valve during the diastole of the left ventricle. This causes first a dilatation and later an hypertrophy of the walls of the left ventricle, an enlargement greater than in any other form of heart disease.

This form of valvular disease may not interfere with the general efficiency of the patient for a long time, but when the period of failing compensation finally sets in the patient goes down hill rapidly.

Physical Signs: *Inspection.* Owing to the extreme hypertrophy of the walls of the left ventricle the entire heart area may appear prominent in the chest. The apex beat is seen low and to the left, in the sixth and even in the seventh interspace, and sometimes as far out as the anterior axillary line. The carotids often can be seen to pulsate.

Palpation gives a pronounced firm apex beat. We may also elicit a capillary pulse wave after light pressure on the skin.

Percussion gives a much increased area of heart dulness, especially to the left. The radial pulse is full but short, and quick, and promptly recedes. This can be best made out by elevating the arm, and is known as Corrigan's pulse, or the water-hammer pulse.

Auscultation reveals a long diastolic murmur at the upper part of the sternum, frequently of a musical character. It may sometimes be heard at the apex, but faintly. The systolic sound at the apex is not as clear and distinct as is normal.

Valvular sounds may be heard over the various arteries. Over the femoral can sometimes be heard a double sound. There is no characteristic transmission of this murmur through the carotids.

Failing compensation produces a venous stasis, and as a result cough, dyspnea, sometimes hemoptysis, and later edema.

4. Aortic Stenosis: Pure aortic stenosis is said to be a rare disease.

This is a narrowing of the aortic opening due to a thickening or adhesion of the valves. It interferes with the flow of blood from the left ventricle into the aorta. As a result the left ventricle becomes hypertrophied. Moreover, the amount of blood thrown into the aorta is diminished, consequently the radial pulse is small and the arteries become contracted.

Physical Signs: *Inspection* shows a forcible apex beat moved to the left. Occasionally the apex beat is less forcible than normal.

Palpation also shows the forcible apex beat removed to the left. The pulse is soft and inclined to be slower than usual.

Percussion shows the area of the heart's dulness to be increased towards the left. Emphysema frequently accompanies aortic stenosis. In that case the heart dulness may appear to be less in extent than normal, or it may be masked and lost altogether because covered by lung tissue.

Auscultation gives a harsh systolic murmur at the second right intercostal space or second costal cartilage. It is transmitted to the right and to the carotids. The second sound of the heart at the base is weak.

Aortic stenosis is more common in old people with arteriosclerosis. The heart beats slowly, the pulse sometimes being as low as forty or less per minute.

The case may go on for years. But in severe cases the patient may have attacks of vertigo or syncope, and even epileptiform convulsions. One case had repeated fainting attacks for a month and convulsions once, then died very suddenly four days later.

5. Tricuspid Regurgitation: *Synonyms:* Tricuspid Insufficiency; Tricuspid Incompetency.

Tricuspid regurgitation is extremely rare as a primary condition. It is quite frequent as a secondary condition following disease of the left side of the heart.

It may be caused by shortening or contraction of the valves, or by adhesions. A relative insufficiency occurs when the right ventricle is so dilated that the valves cannot close properly.

Contraction of the right ventricle causes a reflux of blood into the right auricle. As a consequence there is a venous stasis throughout the body. This causes a venous pulse.

As tricuspid regurgitation is usually added on to some pre-existing lesion, the general symptoms already existing are simply exaggerated.

Physical Signs: *Inspection* shows the venous pulsation in the jugulars, but more pronounced on the right side.

Palpation will help to prove this a true venous pulse. Slowly empty the jugular from below up with pressure of the finger. The vein will fill from below during the heart's systole due to the reflux of blood through the damaged valve. The liver may be also carefully palpated for a venous pulse there.

Percussion results depend mostly on the preceding lesion.

Auscultation shows a systolic murmur heard best at the lower end of the sternum.

6. Tricuspid Stenosis: This is a rare condition. It is most often congenital. When acquired it is almost always in association with mitral stenosis.

The diagnostic point to be remembered is that the murmur is diastolic and heard best over the lower end of the sternum.

7. Pulmonary Regurgitation: *Synonyms:* Pulmonary Insufficiency; Pulmonary Incompetency.

This is another rare form of valvular lesion. When it comes it has the same effect on the right side of the heart that aortic regurgitation has on the left.

8. Pulmonary Stenosis: This is most frequently a congenital condition, very rarely indeed an acquired one.

Strümpell says children with pulmonary stenosis rarely live to be over fifteen. They are under sized and under developed for their age. They are cyanotic, the eyes are prominent, and they have clubbed fingers.

Physical Signs: *Inspection.* The cardiac region is prominent.

Palpation also shows this prominence.

Percussion. Dulness extends further to the right than normal.

Auscultation reveals a systolic murmur over the whole heart loudest at the second left intercostal space. The pulmonic second sound is weak.

The patients die usually of failure of compensation. Pulmonary tuberculosis is a frequent complication.

Combined Lesions: It is not unusual to have a double lesion of a given valve, that is, to have both stenosis and regurgitation at the same time. Quite often more than one valve will be affected. Late in the disease, with failing compensation, secondary lesions appear.

Symptoms: *Period of Compensation.* In acquired valvular lesions of the heart, if the onset has been sudden, the amount of extra work thrown on the muscular tissue of the heart causes, first, an acute dilatation of the cavities affected. During this period the patient may suffer from dyspnea and dropsical condi-

tions may appear. If the patient responds to careful treatment, or if the onset of the valvular defect is slow, the walls of the heart gradually become hypertrophied and eventually are able to do the extra work required of them so that the patient is not troubled by his disease. This condition is known as the period of compensation. By care in avoiding unnecessary exertion, and by right living, the patient may go on for years without being annoyed by any heart symptoms.

Failing Compensation. If, however, a fresh attack of acute endocarditis supervene, or if the heart muscle gets worn out, or if the patient undergoes some excessive physical exertion or profound emotional disturbance, the heart loses its ability to cope with this extra work and the period of failing compensation sets in.

In mild cases a little extra exertion may cause an increase in the frequency of the heart beats, possibly some irregularity, and there may be some dyspnea. There may be pain and tenderness.

In severe cases of failing compensation there are many symptoms. The most noticeable one is shortness of breath. This may vary all the way from a slight dyspnea on exertion to a condition where the patient cannot lie down. When he reaches this stage he is apt to have acute asthmatic attacks when he literally gasps for air, when all the voluntary muscles of respiration are brought into play, and the distress is very great. This is true cardiac asthma.

The shortness of breath may be due to the congestion of the lungs, or it may be due to hydrothorax, either of which is due to the impaired circulation.

Edema is another symptom caused by failing compensation. This may occur in any part of the body. It appears first, as a rule, in the feet. As the case progresses it extends up the legs, and there may be ascites. Next to edema of the extremities edema of the lungs is probably the most common. At the end there is sometimes anasarca.

Unpleasant dreams are frequent.

In certain cases there will be disturbed mentality due to the impaired circulation. A young German in my service at the Flower Hospital in September, 1914, became insane in this way.

In a few cases hemiplegia occurs as the result of embolus. I have seen a number such.

Emboli may lodge in other parts of the body besides the brain. A case in my service at the Flower Hospital suddenly developed acute edema in one arm due to this cause. Another private patient had the same accident. Each died very suddenly a few days later, probably from cerebral embolus.

The most common place for this accident to occur is in the lung, producing hemorrhagic infarct. This is evidenced by sudden dyspnea, increased frequency of respiration, cough with bloody sputa, and, if near the surface, the physical signs of a localized consolidation.

The quantity of urine will be diminished, and albumin will often be found in it.

The digestion is impaired. There may be nausea and vomiting towards the end.

Most of the cases of failing compensation that come to the physician come for the dyspnea. Others will come because of pain about the cardiac area or because of pain in the epigastrium. In the latter case the physician must be on his guard else he may mistake the trouble for indigestion. In every such case the heart should be examined.

The **physical signs** of failing compensation are many.

Inspection. The patient may appear more or less cyanosed. The respirations will nearly always be increased and physical exertion will accelerate them.

Exposure of the chest will also frequently reveal difficult breathing. The voluntary muscles are brought into play in severe cases.

Frequently there is an increased frequency or tumultuous action, or both, in the cardiac pulsations.

Palpation gives a weak, rapid and often irregular pulse.

In case of hydrothorax there will be diminished vocal fremitus. In case of infarct of the lung this may be increased.

Over the cardiac area there may be thrills and irregularity in the heart's action.

Percussion reveals a much enlarged area of cardiac dulness.

Hydrothorax and sometimes infarct give dulness over the affected areas.

Auscultation over the lung will give the peculiar crepitation of congestion or edema if either exists. In case of infarct or of hydrothorax there is a diminution or absence of the respiratory murmur.

The heart sounds vary according to the initial lesion early in the case. Later, when the heart is much dilated, all sorts of murmurs may be heard. It is frequently impossible to say which valve was primarily affected, as secondary murmurs are usually present. There is a general break down, and murmurs may be heard at the mitral, aortic and tricuspid valves.

The normal cardiac rhythm is gone. The first and second sounds lose their proper relations. As noted in the section on acute endocarditis, the shortening of the interval between the second and first sounds, and the lengthening of the interval between the first and second sounds, so that the two are nearly alike, is always an alarming symptom to me.

Complications and Sequelæ: In a slowly developing endocarditis there will be a hypertrophy of the walls of the heart to overcome the defect in the valvular system. This may last for years. When the period of failing compensation sets in the various organs become congested and edema sets in on account of the back pressure. If particles of vegetation are thrown off from the valves emboli are found causing different symptoms according to where they lodge. In the brain, hemiplegia; in the lungs, pain, dyspnea, hemoptysis; in the kidneys, pain, hematuria, etc.

Diagnosis: The diagnosis can be made only by consideration of all the symptoms, plus a careful physical examination of the heart.

Prognosis: The prognosis must be made on the sum total of the symptoms. Mitral lesions are less dangerous than aortic lesions. During the stage of compensation the prognosis is good. If the patient has symptoms pointing to failure of the circulation he must avoid severe physical or mental strain. If he cannot avoid these things the prognosis becomes progressively worse.

When compensation has broken down the prognosis is always

in doubt. Careful treatment may relieve an acute breakdown, and the patient may eventually get in fairly good condition again, and be able to go on for months. This may occur two or three times at intervals. But eventually, after compensation has failed once, sooner or later it will fail again and beyond repair, and death will take place unless the patient is carried off by some intercurrent disease.

Treatment: In the acute infectious diseases that predispose to endocarditis the heart should be carefully watched for symptoms of trouble. If endocarditis supervenes, absolute rest must be enjoined, and the case must be symptomatically treated.

A patient who already has a chronic endocarditis, who has no symptoms resulting from it, should be instructed how to care for himself that he may prevent a breakdown. Severe physical and mental effort must be interdicted. He must be taught to live the simple life, and if possible, to live it in an equable climate.

When the break comes, and failing compensation sets in, the first and most important thing is rest, absolute rest—physical and mental. As dyspnea is the most common symptom the patient may need to be propped up in bed rather than to be horizontal.

A nutritious and easily digested diet must be given. Liquids are best kept at a minimum if edema is a marked symptom.

If cardiac distress or pain is marked, or if tachycardia is a marked symptom, application of an ice bag over the cardiac area may be of service a half hour at a time at intervals.

Sometimes local stimulation over the cardiac area, either by mild applications or by friction, will help.

Spondylotherapy, as introduced by Abrams, may be tried. What permanent help it can give I do not know, as I have not as yet had time to try it out. I do know that percussion of the seventh cervical vertebra, using a pleximeter and a percussion hammer, will, after percussing for four or five minutes, noticeably slow the pulse. How long the slowing lasts I cannot say at this writing.

Digitalis leads in the list of drugs when the compensation fails.

The tincture or the infusion may be used, ten drops of the tincture, or a teaspoonful of the infusion. The dose should be given twice a day. It may take a day or two for the effect of the *digitalis* to be manifest. When it is the heart's action is slowed and strengthened and there is an increase in the quantity of urine. It is more useful in cases with marked edema. It sometimes fails.

A few patients cannot take *digitalis* because it produces nausea.

Strophanthus tincture in drop doses, every two hours, is probably next best. I have had most excellent results from it.

Caffeine IX may be used—a tablet every two hours.

Apis is a great homœopathic remedy. I have seen this remedy in potencies from the third to the two-hundredth clear up cases that seemed hopeless—were water-logged, in fact. My own preference is for the lower potencies, but the 200th used by Dr. McKnight when an interne on my service at the Metropolitan Hospital cured one very desperate case. The man recovered enough to leave the hospital and resume his work. He lived several years afterwards.

Apis is a slow acting remedy and does not take hold for several days. One case of cardiac dropsy was relieved within a week.

Aconite will sometimes help a badly acting heart, especially if the *aconite* mental symptoms are present.

I have been unable to get results in valvular lesions with *cratægus,* though I have had good results when the disturbance was purely functional.

Arsenicum album is one of the best remedies for long continued use when compensation is re-established.

Camphor in drop doses every five or ten minutes is one of the best heart stimulants for emergency use in threatened heart failure.

Spigelia relieved pain about the heart in a case of mitral regurgitation with palpitation.

Arnica is useful in cases with pain and precordial distress where the condition has been brought on by hard work.

Many other remedies may be indicated.

MYOCARDIAL DISEASE.

(Greek, μυς, muscle; καρδια, the heart.)

Definition: Myocarditis is inflammation of the muscular walls of the heart. It may be acute or chronic, circumscribed or diffuse.

Etiology: Acute circumscribed myocarditis, or abscess of the heart, is most often due to metastasis by way of the coronary arteries. Less often it is due to extension from a pericarditis or an endocarditis.

The diffuse form is due to the infectious diseases as typhoid, diphtheria, scarlet fever or sepsis.

Chronic myocarditis may also be circumscribed or diffuse. Frequently it is due to an acute endocarditis. In other cases it follows arterio-sclerosis. A frequent form is due to sclerosis of the coronary artery.

Morbid Anatomy and Pathology: In abscess of the heart the broken down areas may be single or multiple, minute or of considerable size.

The diffuse form shows an infiltration of the connective tissue usually with degeneration of the muscle fibres. The chronic form is a sclerosis.

Myocardial Degeneration: The various forms of degeneration may attack the heart muscle. Principal among those are parenchymatous, amyloid, hyaline and fatty. Amyloid and hyaline degenerations are found in association with amyloid degeneration of other organs and are usually associated with each other. Fatty degeneration is sometimes the final result of parenchymatous changes. Fatty infiltration is most often found in persons suffering from obesity.

Etiology: The infectious diseases are most often at the bottom of parenchymatous degeneration of the heart. Gout and syphilis, chronic Bright's disease and anemia are the most frequent causative factors in fatty degeneration. Obesity is the cause of fatty infiltration.

Morbid Anatomy and Pathology: The various degenerations are best differentiated with the microscope. The parenchymatous form shows the fibres to contain minute granules of an albumi-

nous nature. Acetic acid dissolves them. The amyloid form reacts to iodine. The fatty form shows fat granules. In this latter there may be considerable microscopic deposits of fat.

Symptoms: The symptoms of myocardial disease are often very obscure. The history of the case is essential in making a diagnosis.

The patient is apt to suffer from dyspnea on slight exertion. The pulse is apt to be irregular and is easily disturbed by physical exertion or by the emotions. Attacks of palpitation are not infrequent. Symptoms of circulatory stasis develop. There may be periods of precordial anxiety and even angina.

There may be sensations of slight pain or fulness in the region of the heart. The patient may complain of cold feet and hands or cold nose, indicating deficient heart action.

Edema occurs in various parts of the body due to the defective circulation. The urine may be scant for the same reason.

Examination of the heart shows it irregular and weak in action, but valvular lesions are absent in uncomplicated cases. The sounds may not be clear and are usually weak and may vary in intensity. The pulse may be slower than normal, dropping to 60, 50 or even less. Blood pressure is low and may vary considerably from time to time.

There may be more or less chronic enlargement of the heart's area due to either hypertrophy or dilatation or both. In cases with very weak muscles the area of cardiac dulness may increase on exertion. When the walls give way there may be a systolic murmur at the apex.

The defective circulation may cause symptoms of local anemia or congestion in the various organs. Failing mentality may result through disturbed circulation in the brain.

Complications and Sequelæ: Sudden death may be due to thrombosis in the coronary arteries. In other cases hypertrophy occurs, followed sooner or later by dilatation, with cardiac asthma and circulatory stasis.

Diagnosis: The diagnosis is often very difficult. The history of the case is essential in forming an opinion. Irregular, weak heart, without evidence of valvular lesion, makes the diagnosis of myocardial disease probable.

Prognosis: The prognosis is always grave. Many cases present no definite symptoms till the end. Death may be sudden and without warning.

Treatment: At first complete rest is essential. When the heart is a little more regular graduated exercise is of benefit.

The diet should be light and nutritious, largely proteid. Liquids should be curtailed. Alcohol and tobacco must be interdicted.

Arsenic or *digitalis* may be indicated.

HYPERTROPHY OF THE HEART.

(Greek, ὑπερ, excessive; τροφη, nourishment.)

Definition: A thickening of the muscular walls of the heart. Hypertrophy of the heart is not a disease but a symptom.

Etiology: In the section on chronic endocarditis we will find that hypertrophy of the heart is secondary to valvular lesions.

It may be primary, due to long continued over-exertion of the heart. Strümpell puts down habitual over-eating or drinking as a common cause. Then comes habitual over-physical exertion, as in porters and so on. Another factor is frequent or long continued over-action of the heart due to emotional causes or constant mental strain.

Chronic nephritis is a frequent cause.

Morbid Anatomy and Pathology: The hypertrophic heart is larger than normal and may weigh two or three pounds or more. The normal adult heart weighs, on the average, eleven ounces in men, nine ounces in women. The largest hearts I have seen have weighed twenty-two ounces. The muscular tissue is firm and increased in amount.

Symptoms: So long as the hypertrophy is just enough to maintain the circulation at the normal there may be no symptoms. When it becomes excessive there may be a vague feeling of discomfort in the region of the heart. Tenderness and pain may be present.

Later, when the overworked muscle begins to give way, dilatation takes place with all its resultant train of evils as outlined in the chapter on endocarditis—failing compensation.

The **physical signs** of hypertrophy of the heart are:

Inspection. The heart impulse is accentuated and the area enlarged. The apex is displaced low and to the left, as a rule.

Palpation gives the increased force of impulse.

Percussion gives an enlarged area of cardiac dulness. The area extends to the left and downward in left-sided hypertrophy and transversely in right-sided hypertrophy.

Auscultation gives prolonged and accentuated sounds usually.

Complications and Sequelæ: Hypertrophy of the heart is often associated with nephritis. Dilatation always occurs sooner or later.

The end result of hypertrophy is a cardiac breakdown. The muscle finally loses its tone, acute dilatation occurs and failing compensation ends the scene.

Diagnosis: The diagnosis is made on the history and physical signs. As simple hypertrophy produces no special symptoms, the patient's attention is, therefore, not called to the heart until it begins to give way.

Prognosis: The prognosis is good until dilatation sets in when it is doubtful.

Sometimes the heart will recover itself under proper treatment. Sooner or later it gives way finally.

Treatment: Ordinary cases require no special treatment.

Mental and physical rest are indicated if the heart becomes troublesome.

Coffee, tobacco and stimulants should be interdicted.

Aconite is of service when the heart becomes irritable.

Arnica is probably the most useful remedy in cases due to overwork or overworry.

DILATATION OF THE HEART.

The cavities of the heart may be enlarged and the walls remain normal, or be hypertrophied or be thinned. In either case dilatation is usually an end result.

Etiology: Dilatation of the heart is most frequently caused by a long standing valvular lesion. It may sometimes be caused by an acute endocarditis. Severe physical exertion may cause it;

acute dilatation sometimes occurs in athletes as the result of a contest, or it may occur in those unaccustomed to exercise as a result of some sudden physical exertion.

The infectious diseases may cause degeneration of the heart muscle to such an extent that dilatation ensues.

Finally severe emotional shocks may cause dilatation through the nervous system.

Symptoms: The symptoms are those of failing compensation mentioned in the chapter on chronic endocarditis. There is increasing dyspnea. The pulse is erratic, irregular and weak. There may be faintness and nausea.

Physical Signs: The area of heart dulness will be enlarged.

Auscultation shows the sounds to be weak and irregular. Various murmurs may be heard due to the disturbed valve tension.

Diagnosis: Acute dilatation may be suspected if irregular heart's action comes on following some severe physical strain, or great emotional shock. It may be suspected if failing compensation occurs in an old case of heart disease.

Prognosis: The prognosis is always in doubt at the beginning. Some cases recover under treatment.

Treatment: The first and most important thing in treatment is rest, physical and mental.

The drug treatment is the same as mentioned in the various chapters on endocarditis and myocardial diseases.

NERVOUS PALPITATION.

(Latin, *palpo*, to quiver.)

Definition: Palpitation is the name given to the subjective perception of the heart's action. The patient is conscious of his heart.

Etiology: Palpitation is most often a purely nervous phenomenon. The frequency of the heart beat bears no necessary relation to the subjective sensations. As a rule, however, attacks of palpitation are associated with a marked increase in its frequency.

Strong emotional excitement may produce palpitation. Ex-

cessive use of tobacco, coffee, tea, or alcohol will sometimes cause it. I have known a strong cigar to send a normal pulse to 150. Nervous indigestion frequently causes it. Occasionally it is associated with organic disease of the heart.

In an irritable heart, one easily accelerated, exercise may bring it on.

Symptoms: This consists in the patient feeling his own heart beats. Very often the sensation causes him to think he has incurable heart disease, and the attacks cause him great mental distress.

If the patient is conscious of his heart's beat he has palpitation.

Diagnosis: If the heart is examined during an attack and the sounds found to be normal, then the palpitation is a nervous or functional phenomenon. Of course, the rhythm may be disturbed in organic heart disease. That, however, is not nervous palpitation.

Prognosis: The prognosis is good.

Treatment: Any bad habits the patient may have that may tend to cause the trouble should be corrected. For example, the use of tobacco or coffee or alcohol may have to be stopped.

If the patient is worried about his condition he must be reassured.

Aconite is one of the best drugs in this condition. It is especially useful when the patient is frightened by the palpitation.

Cactus grandiflorus, palpitation with a feeling of constriction. Worse lying on left side. This may be used in cases of mitral regurgitation with attacks of palpitation.

Gelsemium. Palpitation when startled, easily startled. On going to bed hands and feet cold. Gas in stomach. Numb feelings. Feels as though she needed a long breath. Headache, dizzy, horrid dreams, tired, faint and depressed.

Cratægus. I have sometimes found this remedy useful in purely nervous palpitation. In the palpitation that occurs in valvular lesions I have not found it serviceable.

Various other remedies may be indicated.

TACHYCARDIA.

(Greek, ταχυς, quick; καρδια, heart.)

Synonym: Rapid Heart.

Definition: Tachycardia is rapid heart beat.

Etiology: Osler says some persons have a normally rapid heart. I do not recall any adult patient in normal condition with a pulse habitually more than 80.

The same things that cause palpitation also cause tachycardia, namely, emotional disturbance, physical exertion, tobacco, coffee, tea and alcohol.

In fever the pulse rate normally increases about ten beats for each degree of temperature. In convalescence from fevers we frequently have left an irritable heart, that is, one that is very easily accelerated by any physical or emotional effort.

Irritation or disease of the medulla or of the pneumogastric nerve may cause tachycardia.

Symptoms: There are no special symptoms other than the rapid action of the heart which comes on in paroxysms, as a rule. In very rare cases the rapidity is persistent.

Strümpell speaks of cases that have an enlarged area of cardiac dulness in which he has been able to make out a temporary dilatation.

Complications and Sequelæ: Occasionally tachycardia ends in heart failure.

Diagnosis: This is made on the rapidity of action without subjective symptoms.

Prognosis: This depends on the cause. If that can be reached something may be done to benefit the condition.

Treatment: Rest is the first requisite. Cold applications may be used over the heart.

The underlying cause requires the principal attention.

BRADYCARDIA.

(Greek, βραδυς, slow; καρδια, heart.)

Synonym: Slow Heart.

Etiology: This is hardly a disease—it is a symptom. Following

many of the acute infections and following childbirth it is not uncommon to find a slow pulse 50 or even less per minute. In catarrhal jaundice we find it. We find it in old people and in arterio-sclerosis. It may occur in other conditions, but the above are the ones most common.

Symptoms: The slow pulse is often the only symptom. Sometimes faintness or dizziness may occur.

Prognosis: The condition is normal after the acute infections. The prognosis is good.

Treatment: In old people it may be necessary to stimulate some. *Gelsemium* is an excellent remedy for old people.

STOKES-ADAMS DISEASE.

Definition: This is a special type of bradycardia most often occurring in old people with arterio-sclerosis.

Etiology: Stokes-Adams disease is supposed to be caused by defective innervation of the bundle of His.

Symptoms: The pulse is very slow, it may be 20 or even less. It is very irregular, and there are long intervals without any beat at all. Besides the slow pulse, there are attacks of giddiness, syncope, even convulsions. The auricular pulsations are visible in the neck. The auricles beat faster than the ventricles.

Prognosis: The prognosis is uncertain.

Treatment: Rest is necessary for the attack. Between times the patient's life should be so regulated as to avoid excitement or exertion.

Stimulation may be necessary during a severe attack.

HEART-BLOCK.

Definition: Heart-block is the name given to the symptoms produced by disease of the auricular-ventricular bundle or bundle of His. Such disease interferes with the normal stimulus conduction from the auricles to the ventricles.

Historical Note: Gaskell, in 1900, was the first to note the action of the heart when the auricular-ventricular bundle was destroyed in cold-blooded animals. Kent and His confirmed these

experiments, and found similar results in experimenting on mammals. Since then certain patients presenting like symptoms have been found to have diseased auricular-ventricular bundles.

Etiology: The symptoms are caused by some lesion of the bundle of His. Digitalis, asphyxia, stimulation of the vagus, may cause symptoms of heart-block.

Morbid Anatomy and Pathology: There may be abscess or arterial occlusion in the auricular-ventricular bundle; or there may be inflammation, degeneration or fibrosis of the bundle.

Normally the stimulus for heart contraction begins at the great veins, passes to the auricles, thence through the auricular-ventricular bundle to the ventricles. When the bundle is diseased in any way normal stimulus conduction is interfered with, and auricles and ventricles act independently of each other.

Symptoms: Heart block may be (1) *partial*, or (2) *complete*, Heart-block, in whole or in part, may be assumed, when the pulse is very slow—40 or less. It is usually regular and is not influenced by emotion or by physical exertion. There may or may not be any evidence of cardiac distress, as ventricles and auricles act independently. Murmurs, if they exist, are apt to come and go: that is, the intensity of the murmur will be greater when the auricular-ventricular beat happens to be in normal sequence, and be less when the beat is irregular.

Diagnosis: The diagnosis may be suspected with the above symptoms. It can be made certain only with the polygraph or electro-cardiograph.

Prognosis: The prognosis is doubtful as heart-block indicates myocardial disease. Many patients, however, live in comparative comfort for years.

Treatment: Rest is the most important thing in the way of treatment.

Digitalis is contraindicated according to Cowan.

Each patient must be treated symptomatically.

AURICULAR FLUTTER.

Definition: In auricular flutter the auricles contract rhythmically, but with great rapidity, even as much as 350 per minute, while the ventricles contract normally or even very slowly.

Historical Note: The first case of this kind was reported by Gibson and Ritchie in 1905. Other cases were later reported by other observers. Jolly and Ritchie named the condition "auricular flutter" in 1910.

Etiology: The cause is not known. The condition is found in hearts that appear to be normal, it is found in hearts with endocardial or myocardial disease.

It is probable that overuse of coffee, alcohol, or tobacco, may cause it; or indeed anything that tends to accelerate the heart's action.

Symptoms: The symptoms are not very definite. They are not unlike those of tachycardia or of palpitation. One patient of Cowan's said his heart felt "like a bird fluttering, with occasional irregular thumps." The pulse was rapid, as in tachycardia. The attacks lasted for from a few minutes to forty-eight hours.

Another patient had an aortic lesion and symptoms of heart failure, dyspnea, edema, etc.

Diagnosis: Accurate diagnosis can be made best from electro-cardiograms. The auricular contractions may run as high as 350 per minute, while ventricular contractions may be 80 or less.

Prognosis: Cowan thinks auricular flutter to be much more common than is now supposed. He believes attacks, if long continued, may lead to heart failure.

Treatment: In a general way the patient should be treated as for failing heart.

McKenzie and others have found full doses of *digitalis* to change auricular flutter to auricular fibrillation. Then by stopping the *digitalis* the auricles get back to a normal rhythm.

AURICULAR FIBRILLATION.

Definition: Auricular fibrillation is where the walls of the auricles contract with great rapidity, even as much as 600 per minute, but in a tremulous or wave-like manner and not rhythmically as in auricular flutter.

Historical Note: According to Cowan, Cushing and Ed-

munds, in 1906, called attention to the fact that the so-called "delirium cordis" or "mitral pulse" was like the pulse found in animals with auricular fibrillation. Other observers, by the use of the electro-cardiograph, found fibrillation to be present.

Etiology: The condition is sometimes found in mitral stenósis.

Delirium cordis, with auricular fibrillation, is frequently found in dying hearts.

Other causes are not fully known.

Symptoms: An irregular heart, together with a rapid and irregular pulse, would seem to indicate fibrillation.

Prognosis: The prognosis is always grave.

Treatment: *Digitalis* may help. Rest and treatment for failing heart is indicated.

ANGINA PECTORIS.

(Latin, *ango,* to strangle; *pectus,* the breast.)

Synonyms: Stenocardia. Neuralgia of the Heart.

Definition: Angina pectoris, breast pang, is a symptom of various conditions of the heart and blood vessels. It is most often associated with occlusion of the coronary arteries or degeneration of the myocardium. It is manifested by mild, severe or agonizing pains in the region of the heart.

Etiology: The symptom occurs most often after middle life. It is vastly more common in men, and frequently the patient has arterio-sclerosis. I have seen it in women. Prolonged mental anxiety, gout, syphilis, alcoholism and the excessive use of tobacco are all put down as causative factors. It appears to be hereditary in some cases.

Morbid Anatomy and Pathology: Occlusion of the coronary arteries is usually found after death.

Symptoms: The attacks come on suddenly. They are usually brought about by physical exertion, by deep emotional disturbance, or by acute flatulent indigestion.

The pain is agonizing, and there is a feeling of impending death. The pain radiates to the left shoulder and down the left arm.

The patient becomes pale and ashen. He may hold his breath, voluntarily. The pulse is apt to become rapid and irregular; in rare cases it may not be disturbed. Not infrequently the patient breaks out in a cold perspiration. The attack may last from a few seconds to half an hour or longer.

Patients sometimes die in the first attack. If they survive, at the end of the attack they pass much clear urine, and often have eructations of gas.

There are mild cases where the pain is not severe but is persistent.

Diagnosis: The diagnosis of angina must be made from the symptoms and after an examination of the heart and blood vessels. It is almost invariably associated with arterio-sclerosis or with valvular disease.

Pseudo angina is a condition found most often in women of a neurasthenic or hysterical tendency. The attacks last longer, are apt to be periodical and are associated with nervous symptoms.

In true angina the patient is immobile and silent. In pseudo angina she is active and emotional.

Prognosis: True angina is often fatal. The prognosis is always grave. Patients die suddenly.

False angina is never fatal.

Treatment: Subjects of angina should live quietly, without physical or mental strain so far as possible.

Ice bags may be used over the heart. Hot foot baths may be tried.

Amyl nitrite may be inhaled during the attack. Two or three drops are sufficient. Perles may be used, breaking one in a handkerchief and inhaling as needed. This drug is a powerful vaso-motor dilator. Personally, I cannot remain in the room after giving amyl nitrite.

Morphine injections may be of service.

Inhalations of chloroform or of ether may be used.

Between the attacks old school authorities recommend nitroglycerine in 1/100 grain doses three times a day.

Iodide of potassium, twenty grains three times a day, is recommended in cases with marked arterio-sclerosis.

Iodide of sodium was recommended by Dr. J. M. Schley some

twenty years ago. In 1900 Dr. George E. Gorham, of Albany, reported several cases relieved by the iodide of sodium in 7½ or 10 grain doses three times a day.

Spigelia has a feeling of suffocation caused by exertion, but without dyspnea. The apex low and to the left. I have verified this drug.

Latrodectus mactans is recommended by Boericke. The pathogenesis is very much like angina.

Oxalic acid is another drug that may be tried.

Glonoine, in potency, may be used.

Gelsemium is useful in pseudo-angina.

ARTERIO-SCLEROSIS.

Synonyms: Atheroma of the Vessels. Endarteritis. Arterio-capillary Fibrosis.

Definition: Arterio-sclerosis is a hardening of the arteries.

Etiology: Arterio-sclerosis is one of the changes that takes place naturally with advancing age.

According to Allbutt, high blood pressure is the most common cause.

If it appears early in life—before forty—some special cause must be found for it.

Long continued physical or mental strain causes it sometimes.

Constant overeating or overindulgence in alcohol causes it. Lead poisoning, gout, syphilis and chronic nephritis all cause it.

Some of the worst cases I have seen have been associated with tuberculosis.

Morbid Anatomy and Pathology: In acquired arterio-sclerosis the arteries are thickened. This is due to chronic over-stretching and a compensatory hypertrophy of the walls.

The senile form of hardening is due to degeneration. There may be spots of necrosis.

The heart may be much enlarged due to its effort to keep up the circulation in the unelastic arteries.

In some cases the veins are affected also.

Symptoms: *Physical Signs:* The disease is evidenced by the hard tortuous arteries. This hardening can be felt. The blood pressure is usually high.

The heart is much hypertrophied. The apex beat is low and to the left. The aortic second sound is accentuated.

If the coronary arteries are specially affected, eventually myocardiac disease results. In such case there may be aneurism of the heart, thrombosis, angina pectoris.

When the kidneys are specially affected there may be interstitial nephritis.

When the arteries of the brain are affected there may be vertigo, headache, insomnia, paralysis.

Sometimes gangrene of the extremities results.

Complications and Sequelæ have been mentioned in the symptomatology.

Diagnosis: The diagnosis is reasonably certain when the arteries are hard and tortuous, the tension is increased and the left ventricle enlarged with accentuated aortic second sound.

Prognosis: The prognosis is doubtful and depends largely on the particular type of disease presented. Patients may live a long time.

Treatment: Quiet and rest are necessary. Interdict alcohol and coffee. The diet must be regulated. The intake of proteids should be reduced. Plenty of water must be given.

Attention must be given to the elimination by the bowels and kidneys.

The iodide of potassium has enjoyed a great reputation in these cases, especially if the disease is due to syphilis.

Glonoine may be useful in cases with headache and symptoms of nephritis.

Baryta carbonica in cases due to old age.

Plumbum iodatum may be tried.

ANEURISM.

(Greek, *ανευρυσμα*, aneurism.)

Definition: An aneurism is a localized dilatation of an artery.

The most important ones to be considered are *aneurisms of the aorta.* They may occur at any part of it. The etiology is the same for any part. The symptomatology is varied according to the location.

Etiology: The things that cause arterio-sclerosis also cause aneurism; namely, syphilis, alcoholism, gout, lead poisoning, overwork. Of these syphilis comes first.

Unusual physical exertion is the most frequent exciting cause.

Men are most often affected. It occurs most often between thirty and fifty.

Morbid Anatomy and Pathology: The size of an aneurism varies much. It may be fusiform, saccular or cylindrical in shape. If the intima is ruptured and blood divides the layers we get a dissecting aneurism.

The inner layers of the arteries are sometimes destroyed. The stretched aorta becomes larger, hypertrophied and forms the sac. The cavity is usually filled with masses of thrombi.

Aneurism usually attacks the arch of the aorta. Aneurism of the descending portion is comparatively rare.

Symptoms: The subjective symptoms of aneurism are caused by pressure. There may be none till late in the disease. The pressure symptoms vary according to the location of the aneurismal tumor.

ANEURISM OF THORACIC AORTA.

Symptoms: An aneurism of this part may attain great size and present no symptoms. My experience has been that cases discovered before the tumor is visible are usually cases that have presented more or less definite symptoms of heart disease, and physical examination has revealed the tumor.

Pain, when it occurs, is apt to be more or less paroxysmal. It may be lancinating or of a dull aching character. Occasionally the pains simulate those of angina. When the growing tumor is eroding the vertebræ or ribs or sternum, as it sometimes does, the pain may be very distressing.

Tachycardia paroxysms are marked.

Dyspnea is another symptom that may result from direct pressure or from bronchitis. If the recurrent laryngeal nerve is compressed there may be cough, hoarseness and even aphonia.

Dysphagia sometimes occurs.

There may be localized edema and cyanosis of one arm and shoulder.

Inequality of the pulse in symmetrical arteries is an important sign.

The pupils may be uneven due to pressure on the sympathetic.

Tracheal tugging is a valuable symptom if it can be elicited. Oliver says: "Place the patient in the erect position, and direct him to close his mouth and elevate his chin to almost the full extent; then grasp the cricoid cartilage between the finger and thumb, and use steady and gentle upward pressure on it, when, if dilatation or aneurism exists, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand."

Physical Signs: *Inspection.* If the aneurism is deep-seated it may cause no physical signs. In other cases there is seen an abnormal area of pulsation. In front this is usually above the third intercostal space, and to the right. In rare cases it may be seen at the back between the vertebræ and the left scapula.

Later, in extreme cases, the tumor finally erodes the bones and becomes quite pronounced. I have seen two very large ones at the Metropolitan Hospital. One was in a woman, the other in a man. In both cases the tumor occupied the place of the upper part of the sternum and finally ruptured through the skin. The man was in the hospital several years.

In doubtful cases the X-ray may show a shadow.

Palpation will outline the area of pulsation.

Percussion dulness will mark out the area of the tumor. Commonly it is to the right, exceptionally to the left. Aneurism of the aorta, of course, gives the area of dulness above the base of the heart. It is unusual for the heart itself to be much enlarged in aneurism.

Auscultation does not help much. Osler says an important sign, if heard over a dull region, is a ringing accentuated second sound. Aortic regurgitation may co-exist.

Complications and Sequelæ: Aneurisms usually end fatally in perforation. They may rupture into neighboring parts—pericardium, œsophagus, trachea, bronchi or pleura. Sometimes externally in cases that have eroded the sternum.

Diagnosis: The diagnosis in some cases is very easy. In others it may be impossible to be positive.

Mediastinal tumor may be mistaken for aneurism or vice versa. The pulsation of the aneurism is a help. The cachexia of tumor is another.

Prognosis: The prognosis is always bad. The patient may live for several years after the aneurism is discovered, but sooner or later succumbs.

Treatment: In most cases the best that can be done is to have the patient live a quiet life, avoiding physical and mental exertion.

If possible, Tupnell's method may be tried. That is, absolute rest in bed and a very restricted amount of liquids. Tupnell allowed 10 ounces of solid food and 8 ounces of liquids a day. The theory is to lessen the rapidity and force of the circulation and increase the fibrin in the blood. This latter often fails.

Pressure, continuous, may be used where the aneurism is accessible. A few cases have been reported cured in this way.

Acupuncture has been used.

Iodide of potassium and iodide of sodium, 10 to 20 grains, three times a day, are recommended.

Baryta carbonica is underscored as a remedy for aneurism.

Drugs may be useful in conditions that lead up to aneurism. After the formation of the aneurism drugs may be useful for some of the symptoms. The aneurism itself must be gotten at by mechanical or surgical means.

ABDOMINAL ANEURISM.

Symptoms: About 5 per cent. of aortic aneurisms are abdominal. Most of them occur near the celiac axis.

The symptoms are pain in the back radiating along the spinal nerves. There is delay in the femoral pulse. There may be vomiting.

The aneurism may be felt as a pulsating tumor.

Complications and Sequelæ: There may be paraplegia from pressure. Rupture, as a rule, finally takes place.

Diagnosis: In thin subjects the abdominal aorta can be felt. To diagnose aneurism there must be a distinct pulsating tumor.

Other abdominal tumors may sometimes be eliminated in case

of doubt by placing the patient in the knee-chest position. Other tumors will fall away from the aorta, aneurism cannot.

Prognosis and Treatment: The prognosis and treatment are the same as for aneurism of the arch.

Aneurism may occur in any artery. In the peripheral arteries their treatment is surgical.

INFLAMMATION OF THE VEINS.

Synonym: Phlebitis.

Etiology: Phlebitis may be caused by traumatism. It may be caused by infective organisms of various kinds. It may follow infectious diseases or childbirth. It may occur in gout, syphilis, or other general diseases.

Morbid Anatomy and Pathology: Inflammation of a vein often results in occlusion by formation of a thrombus.

Symptoms: Phlebitis usually causes some pain of the part affected. There may be slight fever. After a day or two swelling of the part often appears if the affected vein is deep-seated. If the affected vein is superficial the distal part of it becomes cord-like and may be tender to touch.

Complications and Sequelæ: If the thrombus breaks down we may have suppuration and abscess formation.

If the thrombus breaks loose and goes off in the blood current we have an embolus that may stop at some point and cause a new lot of symptoms. It may cause sudden death by lodging in the heart or brain. It may cause acute pain in some organ of the body and formation of infarct. It may stop in a limb or other part and cause local edema due to damming of the blood current behind it. Varicose veins may follow.

Diagnosis: Phlebitis with formation of thrombus or embolus is an occasional sequel to childbirth, to surgical operations, and to infectious diseases. If sudden edema of an extremity occurs in a case like this, the vein is probably at fault.

I have an old lady under my care now who has decided edema of one leg from the knee down, which she dates from a fall some months ago. She has a valvular murmur. There is very little or no pain in the leg, it is simply swollen and a little stiff and

heavy. It tires quicker than the well one. I believe the condition due to a phlebitis with thrombus formation.

Prognosis: Simple phlebitis may subside in a few days.

If thrombus has formed the swollen part may remain enlarged until the end of life. If embolus takes place the prognosis depends entirely on where it stops. Many sudden deaths occur from this accident. I have known two women friends, not my patients fortunately, to die that way during the lying-in period.

Cases of chronic suppuration of the middle ear sometimes die from embolus lodging in the lateral sinus of the brain.

Treatment: Rest is indicated in phlebitis. If thrombus takes place, as evidenced by swelling and edema of a distal part, rest must be enjoined until we are sure a firm clot has formed, that we may prevent embolus as far as possible.

Drugs may be prescribed symptomatically.

SECTION VIII.

Diseases of the Respiratory Tract.

ACUTE CORYZA.

(Greek, κορυς, the head.)

Synonyms: Acute Rhinitis. Acute Nasal Catarrh. Cold in the Head.

Definition: Acute coryza is a catarrh of the nasal passages.

Historical Note: Acute coryza is a disease as old as the world. It is said not to exist in very cold countries.

Etiology: Sudden changes in the weather start up an acute coryza. Irritant vapors will produce it, or foreign bodies in the nose. And, finally, it is infectious and may be communicated from one person to another.

Morbid Anatomy and Pathology: There is congestion of the nasal mucous membrane, with swelling of the lymphatics as well as of the blood vessels.

Symptoms: A few hours after exposure the nose runs, the eyes water, the patient is feverish. The head feels full and uncomfortable. Sometimes he sneezes. The discharge is at first watery, occasionally excoriating. As the cold develops the discharge becomes thicker. There may be slight headache. The symptoms are like influenza but without the aching. Sometimes the nose is so stopped up that the patient has to breathe through the mouth. Or only one nostril may be stopped up at a time.

Complications and Sequelæ: Sometimes the cold may extend to the middle ear. More rarely some of the sinuses of the bones become involved. This is always distressing and sometimes serious.

Diagnosis: The diagnosis of nasal catarrh is easy. But there are several things to be remembered. (1) In infants of a few days or weeks this catarrh may be a manifestation of hereditary

syphilis. (2) In children big enough to play about it may mean that a foreign substance has been inserted in the nose. (3) The nasal condition may be the initial stage of measles. (4) It may be the initial stage of influenza. (5) It may be the beginning of hay fever in those subject to that condition.

Prognosis: The prognosis of an ordinary cold in the head is good. Some persons become so susceptible that they have frequent recurrences that eventually make the condition more or less chronic.

Treatment: Much can be done to prevent acute coryza by keeping warmly clad in cold or changeable weather, by keeping the feet dry, by avoiding exposure.

Accustoming one's self to cold baths in the morning is a good hygienic measure. It makes one less susceptible to colds.

I cannot recommend woolen underwear as is done in some text books. One patient wore woolen underwear for ten years or more, until he finally found himself in New Mexico as a "lunger." As he did not see how he could be any worse off he discarded the more or less uncomfortable wool for something better. He wears the thinnest linen or balbriggan now all the year round. It is best to have varying weights of overcoats, and protect one's self in that way. It is vastly more comfortable and healthier.

Neither can I recommend the Turkish baths as Osler does. The discomfort of a Turkish bath when one has a bad cold is intense.

Local treatment may be used for temporary relief, either as a spray or as a douche. My favorites are pankotine or abolene, diluted with water, one part to four or five.

If the douche is used the nose should be blown first. Then use the solution, at body heat, by throwing the head back and allowing the solution to run gently into the nostril. Hold for a moment without snuffing or breathing in. Then let it run out. Do not blow the nose for at least ten minutes afterwards. Snuffing or blowing the nose too soon may force some of the liquid into the Eustachian tubes and cause trouble with the ears.

Of homœopathic remedies very many may be indicated. A few are very often called for, others less often.

Camphor is said to ward off a head cold if given at the time of exposure.

Aconite may be given at the beginning of the congestive stage if the coryza is due to exposure to a cold wind.

Gelsemium may be given if the patient is feverish and hot and cold by turns. The slightest motion makes him feel chilly.

Euphrasia is indicated if the eyes as well as the nose run.

Iodide of arsenic is frequently of service if the nose has a profuse watery discharge.

Sambucus may be given if the patient has snuffles, especially a child.

Later in the disease *belladonna* may be indicated if the head feels heavy, if there is considerable fever, if light hurts the eyes.

Pulsatilla is indicated if the discharge becomes thick, is worse indoors and the sense of smell impaired.

Kali bichromicum is indicated if the discharge becomes thick and stringy.

CHRONIC CORYZA.

Synonyms: Chronic Rhinitis. Chronic Nasal Catarrh.

Definition: Chronic nasal catarrh is due to more or less permanent changes in the nasal mucous membrane.

Etiology: Chronic nasal catarrh may be the result of frequent acute attacks. It may be the result of certain other diseases as tuberculosis and syphilis of the nose.

Morbid Anatomy and Pathology: (1) Great thickening of the nasal mucous membrane may take place, hypertrophic rhinitis.

(2) This may be followed by atrophy.

Symptoms: In the hypertrophic form of rhinitis we have a condition similar to that of acute rhinitis except that the symptoms persist. The nose is occluded more or less all of the time. The patient is obliged to breathe through the mouth. The throat becomes dry, the breath disagreeable.

In the atrophic form crusts form and decompose. The breath becomes very offensive,—ozena.

Complications and Sequelæ: A continued chronic rhinitis may, by impaired breathing, cause malnutrition.

Diagnosis: The diagnosis can be made by examination of the nose.

Prognosis: The prognosis in the hypertrophic form is better than in the atrophic.

Treatment: Hygienic treatment is the same as for the acute condition.

Local treatment is necessary for cleansing purposes if for nothing else. Operative interference may be necessary. That should be left to the specialist.

Dr. J. Ivimey Dowling recommends the employment of tampons of cotton saturated with argyrol, forty grains to the ounce. These should be placed as high up in the nose as possible, in the region of the ethmoid cells, and left in situ for an hour or so. After withdrawal the nares should be thoroughly cleansed. Treatment may be repeated every other day till relieved.

Pulsatilla is perhaps the most frequently indicated remedy for chronic catarrh. The discharge is thick or may be greenish, and the trouble seems largely post-nasal.

Kali bichromicum is indicated if the discharge is tenacious.

Mercurius solubilis has crusts and bleeding surfaces.

Nux vomica is indicated in chronic congestion.

Aurum metallicum is indicated in congenital syphilis with necrosis of bone.

Cadmium sulphuricum is indicated in similar cases without caries.

HAY FEVER.

Synonyms: Autumnal Catarrh. Rose Fever, etc.

Definition: An acute catarrhal condition of the nasal mucous membrane due to irritation in susceptible individuals of some particular pollen. Usually the conjunctivæ are affected. Sometimes there are asthmatic attacks.

Historical Note: According to Osler, Bostock in 1819 was the first to describe the condition.

Etiology: It is pretty well established (1) that some form of pollen starts the condition each year. Many plants have been shown to be causative factors. One person is susceptible to one plant, another to another. According to Luebbert, of the Statz Hygienic Institute of Hamburg, working under Dunbar, "the hay

fever of Europe and the June cold of North America is principally produced by the pollen of grasses, the autumnal cold of North America by the pollen of ragweed (ambrosia) and of golden rod (solidago)." (2) In many patients there appears to be some diseased condition of the nasal mucous membrane which makes the individual susceptible. If that can be corrected the attacks may cease. (3) There is apparently a neurotic element because many persons are affected on a certain date regardless of other conditions so far as known. For instance, one of my patients begins sneezing promptly on the morning of May 30th each year, and keeps it up until July. She is in dread anticipation for a month before and invariably begins her attack on that particular date. Another patient of mine always began on a certain date in August. Still another, a man, has no particular date, but begins his seance after exposure to the dust of travel, either in July or August. If his business keeps him in New York during those months he escapes. Once started his attacks last until the first frost. These cases are typical of the vast majority. Heredity is also said to be a factor.

Morbid Anatomy and Pathology: The most common condition is swelling of the mucous membrane of the nose.

Symptoms: The group of conditions known as hay fever usually begin on a certain date, or after exposure to the particular obnoxious pollen, by paroxysmal sneezing, by a watery discharge from the nose, and frequently associated with them a somewhat congested conjunctiva with lachrymation. In short, the attack is like a beginning acute coryza. Many cases will have attacks of asthma, especially at night, so that they cannot lie down to sleep. These patients suffer acutely.

The condition usually lasts four to six weeks or longer, and before it is ended the patients sometimes are quite used up.

Diagnosis of the condition is easy. Diagnosis of the particular cause in each individual case is not always so simple.

Prognosis: The disease is self-limited, that is, cases run four to six weeks. Those that begin early in the spring or summer come to an end in a few weeks. Those that begin in the late summer or fall last until frost. The prognosis as regards suc-

cessive attacks each year is not good unless the environment can be completely changed. Occasionally cases seem to be permanently cured.

Treatment: The attack may sometimes be prevented if the patient can leave home at the approach of the susceptible time. Some of my patients have escaped altogether in the years they have been able to go to the White Mountains—a region that seems to be free of the offending cause. One or two others have escaped by going to the seashore. One patient, whose home was in the country on a big apple farm, was able to escape attacks by leaving home and living in New York City during the apple season.

The hygienic conditions must be of the best. The patient should sleep with windows closed. The patient's diet must be simple and nourishing.

Any disease of the nose should be corrected.

Dunbar's pollantin, a serum and powder, has been advocated by some. I have seen it fail.

Much can be done with the indicated homœopathic remedy. My experience leads me to give *arsenicum album* first place. I use the 6x. I cannot report cures with this remedy, but I have a number of patients who get along comfortably while taking the arsenic, but who are in distress without it.

Many other remedies may be indicated, *ipecac, iodide of arsenic, gelsemium,* etc.

Dr. Harriet W. Hale, of Brooklyn, uses *sabadilla* in the same way that I use *arsenic.*

In a discussion of hay fever at the New York State Homœopathic Medical Society meeting in 1909, Dr R. F. Rabe suggested giving the indicated remedy at intervals throughout the year as a cure and preventive. I have never tried that.

Antimonium tartaricum has helped the loose cough following hay fever.

Dr. G. B. Rice, of Boston, in the *New England Medical Gazette,* mentions *iodide of potassium, mercurius iodide, arsenic, quinine, sulphur, arsenate of strychnine, naphthaline, sabadilla, grindelia robusta, allium cepa, arum triphyllum,* as remedies that may be indicated.

EPISTAXIS.

(Greek, *επιστάζειν*, to distil.)

Synonym: Nose-bleed.

Definition: Hemorrhage from the nose.

Etiology: Epistaxis may be the result of traumatism. It may be the precursor of certain infectious diseases, as typhoid. It may be due to weakness of the blood vessels of the nose. In elderly people it may be due to changes in the walls of the blood vessels. It is frequent in cirrhosis of the kidney and liver. It may be due to ulceration from disease or from picking the nose. It may be due to diseases like anemia and chlorosis. In women it sometimes replaces the menstrual flow.

Morbid Anatomy and Pathology: In frequently recurring epistaxis the nose must be examined to see if any structural change has taken place.

Symptoms: The symptomatology is simple. It consists in the bleeding. Most cases bleed from the septum side of the anterior nares. Occasionally it is far back. In that case, or if the bleeding occurs at night, the blood may get into the naso-pharynx and be coughed out or swallowed and vomited.

Complications and Sequelæ: Occasionally hemorrhage is so profuse that it causes anemia.

Diagnosis: The diagnosis is self-evident.

Prognosis: The prognosis is good except in hemophilia. I have seen one death in a man a hemophiliac. An expert nose and throat specialist was called in consultation.

Treatment: Local disease must be cared for. In the majority of cases of simple bleeding cold water applied to the nose will stop it, or cold at the back of the neck—the traditional "key down the back"—or holding the hands above the head, etc.

Pressing the nostril on the affected side will stop it usually. The pressure should be kept up till a clot forms, and care must be exercised not to blow the nose till the clot is formed.

Tannic acid diluted with water will stop it. Adrenalin can sometimes be used.

Homœopathic remedies: *Aconite, hamamelis, nux vomica, pulsatilla,* etc., may be indicated.

Ipecac 200 cured a girl of 22 with repeated attacks covering several days.

ACUTE LARYNGITIS.

Definition: An acute inflammation of the larynx.

Etiology: "Catching cold" is the most frequent cause of acute laryngitis. It may be caused by exposure to dust or noxious gases, or to overuse of the voice. Frequently it is associated with a catarrhal condition of the nose and pharynx.

Morbid Anatomy and Pathology: There is congestion and slight edema of the mucous membrane of the larynx.

Symptoms: Laryngitis is usually associated with nasal and pharyngeal catarrh. There is nearly always some fever associated with it.

The laryngitis shows itself by a fulness in the throat at first. This is followed by hoarseness, which may go on to aphonia. There may be actual pain in the larynx, especially on trying to use the voice. It feels raw.

Cough is a common symptom; it has a dry, hard sound.

There is more or less expectoration, at first colorless and viscid, later more phlegm-like.

Dyspnea rarely occurs in adults.

A special form of acute laryngitis occurs in children—*laryngismus stridulus,* or *false croup.* The symptoms of laryngitis develop suddenly at night, usually waking the child after he has been asleep. As the mucous membrane of the larynx in children is loose in texture, it becomes much more swollen in this condition, and dyspnea is therefore a prominent symptom. The child has a croupy cough. There is evidence of catarrh during the day, but the distressing symptoms are worse at night.

The labored breathing causes the accessory muscles of respiration to be brought into play. The child is anxious and restless.

These symptoms may recur several nights in succession.

A notable feature of this condition is that it seems to be peculiar to certain families.

Examination in acute laryngitis shows the parts to be much reddened and sometimes swollen.

Complications and Sequelæ: Frequent acute attacks may lead to

chronic laryngitis. As noted above, a catarrhal condition of the nose and pharynx may be associated with laryngitis.

Diagnosis: The diagnosis of acute laryngitis is easy.

Prognosis: The prognosis is good.

Treatment: Prophylaxis consists in avoiding all those things that tend to cause laryngitis.

When the disease exists the patient should not use the voice. If the case is at all severe he should remain in bed. At all events he should remain in doors and in a warm room.

Coakley advises the sipping of a glass of very hot milk when the patient awakens in the morning to loosen the accumulated mucus.

He also recommends cold applications or counter irritation with turpentine or turpentine and sweet oil half and half.

Local treatment is counter indicated.

Codein, a half grain every four hours, is used for the cough by the old school.

Homœopathic medication usually obviates the need of codein.

Aconite heads the list of remedies useful in this disease. The cough is dry and hard.

Belladonna is indicated with a dry, hard cough, if the face is flushed, the eyes congested, the pulse hard and full.

Spongia has a hard cough, croupy in character. It is especially useful in laryngismus stridulus. There is almost complete loss of voice.

Phosphorus is the remedy of most use in laryngitis with aphonia. Pain may or may not be associated with it. Expectoration is late in appearing.

Hepar has hoarseness and a loose cough. The patient is very sensitive to cold.

Causticum has a cough, soreness of the chest, hoarseness, sometimes aphonia.

I have verified each of the above many times.

CHRONIC LARYNGITIS.

Definition: Chronic inflammation of the larynx.

Etiology: Repeated attacks of acute laryngitis may cause the chronic condition. Overuse of the voice may cause it. Irritat-

ing fumes may cause it. Excessive smoking is said to cause it. It is associated with other chronic respiratory conditions where coughing is frequent. Tumors so located as to cause passive hyperemia of the larynx cause it.

Morbid Anatomy and Pathology: There is congestion and infiltration of the mucous membrane. The secretion is increased and thickened. A form occurs where small nodules appear on the vocal cords.

Symptoms: The various forms of chronic laryngitis are evidenced by change in the voice. There may be any degree of hoarseness to aphonia. Use of the voice makes the symptoms worse.

There is also cough. The patient is constantly clearing the throat.

The expectoration is not characteristic.

In rare cases stenosis of the larynx develops causing labored respiration.

Diagnosis: Examination of the larynx is all important. This will show the actual condition of the larynx and vocal cords. In chronic laryngitis there is congestion of the mucous membrane and infiltration.

The symptoms may be caused by paralysis or by new growths on the vocal cords. The laryngoscopic examination will differentiate.

After examination of the larynx the other parts of the body must be examined as various other conditions, as noted in the etiology, may cause laryngeal symptoms. Certain general diseases, as syphilis, tuberculosis and Bright's disease, may be at the bottom of the laryngeal condition.

Prognosis: Simple chronic laryngitis is a tedious disease to cure. In cases caused by severe underlying systemic diseases the ultimate result depends on what can be done for them.

Treatment: The first requisite is to remove the cause. The patient should use the voice as little as possible. Smoking and the use of alcohol must be forbidden.

The use of warm drinks, especially hot milk, or hot milk and seltzer water should be encouraged.

The indicated remedy must be given. This is most important. Besides the remedies mentioned under acute laryngitis, one may consider:

Alumen in chronic laryngitis of old people.

Argentum metallicum in the laryngitis of singers.

Arnica is of service in cases caused by overuse of the voice.

Drosera for rough, scraping sensation in the throat. Voice deep and unnatural. Every effort to speak hurts.

Selenium, hoarseness—in old men.

EDEMA OF THE LARYNX AND GLOTTIS.

Definition: Edema of the entrance of the larynx, especially of the ary-epiglottic ligaments.

Etiology: It may be due to perichondritis of the larynx, to laryngitis, to certain of the acute infectious diseases, to chronic Bright's disease, to chronic heart disease, to foreign bodies in the larynx.

Morbid Anatomy and Pathology: The edema is most marked in the ary-epiglottic folds, the ventricular bands, and occasionally the tissue beneath the vocal cords.

Symptoms: The symptoms usually begin suddenly. Dyspnea is marked and may develop very rapidly. The voice becomes harsh and is then lost. Cyanosis comes on, due to the difficulty in breathing. Swallowing is difficult.

Examination shows the swelling. It may even be so marked that it can be felt by the finger.

Complications and Sequelæ: It is itself a complication. Death is often the result.

Diagnosis: The rapidity of its onset is usually sufficient, together with the swelling, to make the diagnosis.

Prognosis: The prognosis is frequently bad. If prompt treatment is instituted the patient may be saved.

Treatment: Ice internally and externally may give relief if the swelling is mild.

Scarification under cocaine may be attempted. Tracheotomy may have to be resorted to.

Apis may give relief.

TUBERCULOSIS OF THE LARYNX.

Synonym: Laryngeal Phthisis.

Definition: Tubercular infiltration or ulceration of the laryngeal tissue.

Etiology: Tuberculosis of the larynx may occasionally be primary. More often, usually in fact, it is secondary to pulmonary tuberculosis. The same underlying causes are at the basis of it as in pulmonary tuberculosis.

Bacteriology: The tubercle bacillus may be demonstrated.

Morbid Anatomy and Pathology: Tuberculosis of the larynx begins with an infiltration and then formation of small nodules. These form cheesy masses, then break down and ulcerate. These ulcers may coalesce. They are usually covered with a grayish exudate and the mucous membrane about them is swollen. The disease may destroy the vocal cords. It may extend to the surrounding structures, to the pharynx, etc.

Symptoms: The most noticeable clinical symptom of laryngeal tuberculosis is a change in the voice. This, at first, may be only a hoarseness that becomes better when the patient clears the throat. Later the hoarseness is permanent, then frequently progresses till there is complete aphonia.

If the tubercles break down and ulceration ensues there is increased expectoration. Then difficulty in swallowing ensues. At times this dysphagia is very marked and is a most distressing symptom.

Tubercular laryngitis is rarely primary, nearly always it is secondary to pulmonary tuberculosis. The systemic symptoms of pulmonary tuberculosis go with the laryngeal disease—fever, emaciation, and so on.

Examination of the larynx shows the mucous membrane to be very pale. There is apt to be seen an edematous swelling in the ary-epiglottic folds. The tubercles show as grayish elevations the size of a pin head. Coakley says "the appearance of any thickening of the interarytenoid region of the larynx in a patient who has pulmonary tuberculosis is almost certain evidence that this portion of the larynx is involved by tubercle."

Ulcers will be covered with a thin gray or yellow exudate containing the tubercle bacilli.

Complications and Sequelæ: Tubercular laryngitis is itself usually a complication of pulmonary tuberculosis. Owing to the difficulty in swallowing in advanced cases the patient suffers from lack of nourishment. The voice is frequently completely lost.

Diagnosis: The diagnosis is usually easy because of the associated pulmonary condition. Presence of the tubercle bacilli will clinch the diagnosis.

Prognosis: The prognosis of laryngeal tuberculosis, in my experience, is not good. A case of pulmonary tuberculosis that develops laryngeal involvement is nearly always hopeless. I know that cures are reported, but I have worked in conjunction with the laryngologist many times and the end has always been the same.

Treatment: The general rules for the treatment of laryngeal tuberculosis are the same as those for the treatment of pulmonary tuberculosis—rest, fresh air, nourishing food. In addition to these three factors local treatment is indicated. This must be carried out by the specialist.

Local applications of *lactic acid* are recommended.

The patients must be well fed. In case of dysphagia liquid food may be the only kind the patient can swallow. Cold drinks may be more grateful that hot.

The remedies mentioned under pulmonary tuberculosis and under chronic laryngitis may be looked up.

SYPHILIS OF THE LARYNX.

Definition: Syphilis of the larynx.

Etiology: The same as syphilis elsewhere in the body.

Morbid Anatomy and Pathology: The initial sore is very rare on the larynx. Secondary symptoms are prone to attack it. Likewise tertiary.

Secondary Stage. Shows itself as mucous patches, usually. There may be a general hyperemia of the larynx.

Tertiary Stage. Gummata, sub-mucous, may be found. They grow rapidly.

Ulcer. When gummata break down they cause destruction and deformity of the part. The epiglottis is sometimes destroyed.

Symptoms: When symptoms of the secondary stage of syphilis attack the larynx we get signs of laryngitis. The patient becomes hoarse.

Examination will show the lesion.

In tertiary syphilis there may be hoarseness, even aphonia. Sometimes there is dyspnea, sometimes dysphagia. The symptoms are largely due to pressure from the gummata and depend entirely on their location.

If ulceration takes place there is local pain. There may be a disagreeable odor to the breath.

The syphilitic ulcer is deep, excavated, and has indurated edges.

Respiration and deglutition may be interfered with, according to the location.

Complications and Sequelæ: There may be destruction of the parts.

Diagnosis: *Secondary.* Look for secondary symptoms elsewhere if the examination shows a mottled appearance of the larynx.

Tertiary. Gummata are like new growths, but gummata grow faster than other tumors. Inquire for a history of syphilis. Iodide of potassium will cause gummata to disappear.

The syphilitic ulcer is distinguished from the tubercular ulcer because it is deeper, has indurated edges, and responds to iodide of potassium.

Prognosis: If vigorous treatment is instituted the prognosis is good, except where the ulceration has destroyed too much tissue. Sudden edema is always a possibility where ulceration has taken place.

Treatment: The treatment is the same as in other forms of syphilis, mercury for the second stage, iodide of potassium for the third stage, plus the indicated remedy.

Salvarsan or neo-salvarsan may be used.

Hereditary syphilis of the larynx occurs in young children. This is a grave condition.

PARALYSIS OF THE LARYNX.

The *superior laryngeal nerve* supplies sensory fibres for the upper portion of the larynx to the glottis, and motor fibres for some of its muscles, especially the crico-thyroid muscles, and perhaps the depressors of the epiglottis.

Paralysis of the nerve is seen after diphtheria sometimes. When associated with anesthesia choking is possible. The paralysis of the crico-thyroid muscles makes the voice rough.

The *inferior laryngeal nerve, the recurrent* supplies sensory fibres to the larynx below the glottis, and motor fibres to (1) the openers of the glottis, (2) closers of the glottis, (3) tensors of the vocal cords.

Strümpell says most of the paralyses of the recurrent nerve are of peripheral origin. That is, they are caused by pressure of aneurism of the aorta, of tumor of the mediastinum, of the thyroid, or of the esophagus.

Complete paralysis causes complete aphonia, the patient is not even able to cough.

When the (1) openers of the glottis are paralyzed inspiratory dyspnea is caused. This may be serious enough to necessitate tracheotomy.

When the (2) closers alone of the glottis are paralyzed (rarely) we have hoarseness or aphonia.

When the (3) tensors of the vocal cords are paralyzed the voice is changed, it is hoarse and phonation is difficult. This may occur in hysteria, it may occur in acute or chronic laryngitis, or it may be due to overuse of the voice.

Treatment consists in removal of the underlying causes.

OTHER LARYNGEAL DISEASES.

Besides the special laryngeal diseases enumerated above there may be:

Laryngeal perichronditis, secondary to them, especially tuberculosis and syphilis; or secondary to other of the acute infectious diseases, as typhoid, small-pox, diphtheria, etc., nearly always this goes on to the formation of abscess.

There may also develop *new growths* in the larynx.
Abscesses and new growths require surgical interference.

ACUTE BRONCHITIS.

(Greek, βρογχος, windpipe.)

Synonym: Bronchial Catarrh.

Definition: Acute bronchitis is an acute inflammation of the larger bronchi causing a mucous discharge.

Etiology: Bronchitis is frequently caused by catching cold. It is often the extension downward of an ordinary coryza. Sometimes it develops without a cold in the head. Irritative vapors or inhalation of dust may cause it. It is a start of certain acute infections like measles, grip and often of typhoid fever. It may accompany a pneumonia. Certain micro-organisms sometimes cause it, as the streptococci, staphylococci and pneumococci.

Morbid Anatomy and Pathology: The trachea and bronchi are affected. The mucous membrane is red and swollen and covered with the characteristic discharge.

Symptoms: The patient has chilliness. There may be a slight rise of temperature to 100°-102° F. or more. Associated with this is a feeling of oppression of the chest. The patient may complain of rawness or a feeling of discomfort behind the sternum. Cough develops, which is at first dry and later becomes looser as the discharge becomes more profuse.

In the young and middle aged the disease lasts for a week or two, and then clears up. The patient is not very sick, although he may feel uncomfortable, especially in the beginning.

In very young children and in the aged the affection is prone to extend to the smaller bronchioles when it may be very serious.

Physical Signs: Respirations are but slightly if at all increased in frequency. Palpation and percussion give no definite signs. Auscultation reveals sibilant and sonorous rales scattered over the lungs, coughing changes them. They vary from time to time.

Complications and Sequelæ: Bronchitis may be part of a general cold, or part of another disease, as noted in the etiology. Very rarely it runs into the chronic form. In the very young or the very old it may develop into broncho-pneumonia.

Diagnosis: In influenza there is much more prostration.

In broncho-pneumonia we have the finer tubes affected with areas of consolidation.

Prognosis: In uncomplicated cases the prognosis is good. In the very young and the very old extension to the finer tubes not infrequently causes death.

Treatment: Persons who take cold easily should avoid exposure. Those working in places or trades that produce bronchitis should protect themselves from inhaling the noxious dust or vapor as far as possible. Cold sponging of the chest is a good measure of prophylaxis in those predisposed.

In ordinary cases the diet need not be restricted.

Homœopathic remedies are of great service.

Kali bichromicum is indicated for an irritating cough either dry or with tenacious mucus.

Pulsatilla if there is an associated coryza with thick discharge and impairment or loss of the sense of smell.

Bryonia for a hard, dry cough.

Antimonium tartaricum, coarse rales and loose cough.

Ipecac, fine rales and nausea on coughing.

Belladonna, dry, tickling in throat, cough with soreness in chest; hard cough with flushed face, congested eyes.

Phosphorus has cough with raw feeling behind sternum, or hoarseness, or both.

Sanguinaria, chest sore, cough very frequent, tickle in throat very annoying.

Hepar, loose cough, worse lying down.

Aconite is useful in the very beginning after exposure to cold, dry winds.

Ferrum phosphoricum is useful in the beginning.

CHRONIC BRONCHITIS.

(**Greek**, βρονχος, windpipe.)

Definition: Chronic inflammation of the bronchial tubes.

Etiology: Occasionally the acute form runs into the chronic. The same external causes that produce acute bronchitis, may, by prolonged action, produce the chronic form.

Often chronic bronchitis is a symptom of some other disease of the lungs—emphysema, tuberculosis, pleurisy. And very frequently it is a symptom in chronic heart lesions and in chronic nephritis.

Morbid Anatomy and Pathology: The bronchial tubes present a venous hyperemia of the mucous membrane with increased secretion. Later there may be an atrophy of the mucous membrane, and frequently there is a bronchiectasis.

Symptoms: The principal symptoms of chronic bronchitis are dyspnea—especially on exertion, cough and expectoration. Changes in the weather, to dampness and cold induce attacks. Osler says: "It is the winter cough of the old man, which recurs with regularity as the weather gets cold and changeable."

Cough and sputa vary.

We may have, (1) A dry form, *catarrhe sec* of Laennec, where the cough comes in paroxysms with very little viscid mucus.

(2) A *bronchial blenorrhea* or bronchorrhea, with a profuse thin expectoration, which commonly separates in a vessel after standing.

(3) A *fetid or putrid* bronchitis. In certain cases of chronic bronchitis the expectoration becomes putrid or fetid. It has an extremely disagreeable odor, is usually abundant, and when allowed to stand divides into layers. The upper layer is frothy. The next is muco-serous. The bottom is pus, containing dirty yellow masses—the so-called "Dittrich's plugs." There are crystals of fatty acids.

The sputa is not fetid all the time, but is that way at intervals.

The unpleasant character of the expectoration may be caused by abscesses, by gangrene, or by the accumulation and decomposition in bronchiectasis.

The physical signs may be localized. Occasionally lobular pneumonia is caused by the secretions.

Physical Signs: Inspection, palpation and percussion show little. There may be slight increase in frequency of respirations. On auscultation we usually hear coarser rales of all varieties, sibilant, moist, humming, etc., especially over the lower lobes.

Complications and Sequelæ: Chronic bronchitis may exist, *per se*. More often it indicates some other disease of the lungs or circulation or kidneys.

Search must be made for these complications.

Diagnosis: The diagnosis is comparatively easy. Associated conditions must be noted.

Prognosis: Chronic bronchitis is a stubborn disease. It may last for years and be marked by constantly recurring exacerbations.

When chronic bronchitis is a symptom of some other underlying lung, circulatory or kidney condition, the prognosis depends on the primary disease.

Treatment: The patient should, if possible, be removed from surroundings that act as exciting causes. Change of climate is valuable in many cases. If the bronchitis is only a symptom the underlying condition must be treated.

Care should be exercised in avoiding exposure to inclement weather. Woolen underwear is recommended.

Iodide of potassium, three to five grains, three times a day, is said by old school authorities to loosen tough secretions. Strümpell also advises a plentiful supply of warm drinks.

Inhalations of oil of turpentine may be used in cases with profuse secretion.

Homœopathic medication is of great service. Remedies like *bryonia, hepar, ipecac, kali bichromicum*, etc., on their various indications, as mentioned in the chapter on acute bronchitis may be used. Also,

Arsenic is useful in cases with exhaustion. The cough is dry, but more or less persistent. It is especially valuable in cases associated with nephritis.

Senega is useful in old people who have much mucus which is difficult to raise.

Sulphur is useful in cases with some dyspnea, loose cough, worse at rest. Better lying on right side.

FIBRINOUS BRONCHITIS.

Synonym: Croupous Bronchitis.

This is a rare form of bronchitis that may be acute or chronic —most often the latter—and is usually associated with some other respiratory condition, as tuberculosis or pleurisy.

Symptoms: The usual mucous or muco-purulent discharge is present, and in addition casts of the bronchial tubes are expectorated. These casts are whitish in color, of laminated structure, and have a free lumen inside. A specimen shown at a meeting of the New York Academy of Pathological Science by Dr. Horace G. Keith, of Yonkers, a few years ago, was several inches long and not unlike a miniature tree with its branches in general appearance.

The physical signs are not characteristic. There may be severe dyspnea which is relieved by expectorating the cast during a paroxysm of coughing.

Treatment: Inhalations of lime water are said to be of value.

BRONCHIECTASIS.

(Greek, εκτασις, dilatation.)

Synonym: Bronchial Dilatation.

Etiology: It is caused by various affections of the lungs and bronchi, as bronchitis, emphysema, whooping cough, fibroid phthisis, etc., where long continued paroxysms of coughing produce dilatation of the weakened bronchial walls. Traction due to an adherent pleura is also supposed to help.

There are two forms, the (1) cylindrical, uniform dilatation of a bronchial tube, and (2) saccular, dilatations confined to portions of tubes.

Morbid Anatomy and Pathology: Bronchiectasis is usually unilateral. The dilatations are frequently near an adherent pleura. The walls of the sacs are usually much atrophied. The blood vessels running through the walls of the cavities are often dilated and weakened. Hemorrhage from them occurs in consequence. Ulceration sometimes occurs. The expectoration is apt to be fetid.

Symptoms: The expectoration is abundant and comes in quantities during paroxysms of coughing. On standing the sputum separates into three layers. It has a disagreeable, sometimes a fetid odor.

Hemorrhage sometimes occurs.

Clubbed fingers are often found in those suffering from bronchiectasis.

Physical Signs: *Inspection* shows a contracted chest.

Percussion gives dulness over the affected area.

Auscultation reveals a loss of vesicular breathing, and an abundance of coarse, moist rales.

Complications and Sequelæ: Fetid bronchitis may result.

Diagnosis: Diagnosis is sometimes very difficult, sometimes very easy. Paroxysms of coughing, especially in the morning, or on change of position, raising large quantities of disagreeable sputa, is more or less characteristic. Also the distorted form of the chest and the clubbed fingers.

There is not the cachectic look of the tuberculosis patient and no tubercle bacilli are found.

Prognosis: Usually the disease runs for years.

Treatment is unsatisfactory. The patient must be put in the best possible general condition and in hygienic surroundings.

BRONCHIAL ASTHMA.

(Greek, *αασθμα*, panting.)

Synonyms: Spasmodic Asthma. Exudative Bronchiolitis.

Definition: Asthma may be defined as a special form of dyspnea due to spasmodic contraction of the finer bronchioles. It occurs in paroxysms of varying length of time lasting from a few minutes to hours.

Etiology: The view most generally accepted is that the dyspnea is due to spasmodic contraction of the smaller bronchioles.

The spasm may be caused by the odor from some particular plant or animal on the affected patient. Others may be affected by inhalation of dust or noxious gases. One of my patients, a jeweler, had attacks only after using sawdust and muriatic acid in cleaning jewelry. Others are affected by certain food. Some

again are susceptible only in the atmosphere of certain places. I know a physician who removed from Chicago to Texas because his wife had asthma in Chicago but is free from it in Texas. Emotional disturbance will cause asthmatic attacks in some. In others, reflex irritation from other parts of the body, especially of the nose or genitals, will cause attacks. I know a physician who always has an attack of asthma when under sexual excitement. Asthma is not infrequent in chronic heart and kidney disease. Heredity is said to play a part in its causation.

Morbid Anatomy and Pathology: The morbid anatomy of asthma is not distinctive. Old asthmatics develop emphysema, and then the anatomical changes are the changes found in that disease.

Symptoms: As a rule, the onset of an asthmatic attack is sudden. The patient is distressed for want of air—dyspnea. The voluntary respiratory muscles are brought into full play to assist in breathing. The patient is obliged to sit up, he cannot lie down. He may seek an open window for air. After a longer or shorter time the patient expectorates a thick sticky mucus. This expectoration contains plugs of mucus which, on close examination, are found to be casts of the bronchioles, "Cuschmann's spirals." There are also crystals, "Leyden's crystals," and eosinophile cells.

The breathing is hard and wheezing in character. The frequency of respiration is not necessarily changed—it may be normal, diminished or increased.

Physical Examination: During an attack ***inspection*** shows the accessory muscles of respiration of the neck and chest to be brought into play during inspiration, and the abdominal muscles on expiration.

Palpation will frequently convey a peculiar fremitus due to the coarse rales.

Percussion may be normal, or there may be hyper-resonance.

Auscultation reveals all sorts of noisy, fine and coarse, sibilant, sonorous, wheezing rales;—what one author calls "a veritable pandemonium of adventitious sounds."

Complications and Sequelæ: The tendency of asthma is to become a chronic disease. After the condition has lasted a long time the lungs become emphysematous and the chest barrel-shaped. Frequently there is a chronic bronchitis.

Diagnosis: The diagnosis of bronchial asthma must be made from the symptoms and physical signs. Asthma as a symptom of some underlying respiratory, heart or kidney condition must be differentiated from true asthma.

In asthma as a disease entity there may be no symptoms, either subjective or objective, between the paroxysms. In other cases there may be a general bronchitis, and in cases of long duration there may be emphysema.

In symptomatic asthma the underlying cause must be sought for and found.

Paroxysms of asthma are characteristic, and yet, even if the patient is seen during an attack, mistakes are sometimes made. I have known pneumonia and bronchitis to be diagnosed by the physical signs during an attack.

There is but one condition that I have ever encountered that will exactly simulate the physical signs of an asthmatic attack, and that is whooping cough during or just following a paroxysm of coughing.

Prognosis: The prognosis in idiopathic asthma as regards life is good. The disease is not often cured, but usually becomes chronic and lasts for years.

Treatment: Patients should avoid any known cause. The nose and throat should be kept in good condition.

Patients do best in a good, clean atmosphere, free from dust and noxious odors. Some patients will fare best at the seashore, others will fare best inland. Many chronic asthmatics from the northern and eastern parts of the United States will fare well in Texas.

Good, wholesome, nourishing food is necessary. It has been claimed that fats and sweets should be avoided. The heaviest meal is best taken at midday.

The skin must be kept in good condition by frequent bathing.

In severe cases inhalation of chloroform may be tried during an attack.

Injection of morphine will give relief, but there is always danger of forming the morphine habit.

Smoking of "asthma cigarettes" helps some patients. The cigarettes are made of stramonium or belladonna leaves.

Inhalation of nitrite of amyl perles will stop an attack.

The old school recommends iodide of potassium, ten to twenty grains three times a day.

I have seen very prompt results from the indicated homœopathic remedies.

Calcarea carbonica 200 gave prompt relief at different times in an old man of 77. It is worth trying.

Ipecac has served me well, especially in children. The attacks are frequent, the noisy breathing and prolonged expiration quite characteristic. There is a slight tenacious mucus. Vomiting sometimes occurs during or following the attack.

Phosphorus coughs on laughing. The patient chokes up, coughs, and then it seems as though he never would get his breath back.

Chlorine. This is used in the form of chlorine water and must be freshly prepared. The late Dr. Pierce once prescribed it in a case of mine in consultation. The leading characteristic is difficult expiration. Inspiration is easy. I have since verified it a number of times.

Natrum sulphuricum is said to be useful in asthma of children. Patient must hold chest when coughing.

Asclepias tuberosa is sometimes of service. I have used it often in cases with dry cough.

BRONCHO-PNEUMONIA.

Synonyms: Capillary Bronchitis. Catarrhal Pneumonia. Lobular Pneumonia.

Definition: An inflammation of groups of the smallest bronchioles and their terminal air vesicles.

Etiology: This form of pneumonia is most common in the very young and the very old. It may occur as a separate disease entity in those previously healthy from exposure or some other cause. It may be a complication or an end result of measles,

influenza, whooping cough or other of the infectious diseases. We may have it in tuberculosis. It may be the so-called *aspiration pneumonia,* due to passage of minute particles of food, drink or other substance, or sometimes the blood of hemoptysis into the minute bronchioles. Rickets and diarrhea are said to be predisposing factors. It is most prevalent in the winter and spring.

Bacteriology: There is no specific bacillus for this disease. Streptococci are frequently found. The specific bacillus of whatever disease precedes it is, of course, present.

Morbid Anatomy and Pathology: The essential lesion is the filling of one or more bronchioles and their air cells with a mucopus. Around this is apt to be an area of consolidation.

These areas of infiltration and consolidation are usually interspersed with areas of atelectasis.

The whole process may be in isolated spots, or a number of areas may be grouped together.

Symptoms: In the acute infections the onset of catarrhal pneumonia may be insidious. Cough and slightly increased respirations are the most frequent signs. In such cases the lungs should be carefully investigated at each visit.

As a rule, broncho-pneumonia begins with a sensation of chilliness; there is a rise of temperature. The temperature ranges from 102° to 104° F. Respirations are increased slightly in most cases. In others, particularly very young children, respiration may be very rapid. The pulse is usually increased in proportion to the temperature. Cough may not be much at first, later it may be considerable. The expectoration has nothing characteristic. Children under five years do not expectorate, sometimes they are two or three years older before they learn how.

Physical Signs: *Inspection* may show nothing abnormal; as a rule, however, the frequency of respirations is increased. *Palpation* shows the same. *Percussion* shows nothing early in the case, but as the disease progresses areas of consolidation can be mapped out in various parts of the lung. *Auscultation* tells us the most. Rales can usually be detected over the consolidated

areas. Occasionally even rales are hard to find. There may also be areas of broncho-vesicular breathing.

Complications and Sequelæ: It was formerly supposed that broncho-pneumonia frequently developed into pulmonary tuberculosis. It is now thought that such cases are tubercular from the start and that the broncho-pneumonia is merely an intercurrent disease.

Diagnosis: The most frequent mistake made perhaps is in confounding broncho-pneumonia with lobar pneumonia. This is especially true if the broncho-pneumonia involves a large amount of adjacent tissue. The onset of lobar pneumonia is more abrupt; the physical signs are more marked over an entire lobe; the pulse-respiration ratio tends to approach two to one or even less. Broncho-pneumonia shows usually affected areas in different parts of both lungs. The pulse respiration ratio tends to stay at four to one, both going up together. Lobar pneumonia is a self-limited disease from the initial chill, high continued fever, and frequently ending in crisis. Catarrhal pneumonia runs an indefinite course, fever not so high and ending in lysis. Frequently, especially in children, tuberculosis is mistaken for broncho-pneumonia.

Prognosis: In the very young and the very old the prognosis is not good. Neither is it good in those not otherwise in good condition. In other patients the prognosis is better. Broncho-pneumonia always calls for the closest attention on the part of the medical attendant.

Treatment: Attention to the nose and throat in infectious diseases will do much to prevent the onset of broncho-pneumonia. Avoidance of exposure to cold will also help to keep it away.

During the height of the disease the diet must be light, but nourishing. Water should be given in abundance.

Baths will be of service if the fever is high.

The old school depend on hygiene, stimulants if the heart becomes weak, some form of opium if the patient has much pain, or if the cough becomes excessive.

Fortunately homœopathy has many remedies of service in this condition.

Among the most important are *aconite* in the beginning.

Later *ipecac* is one of the most useful remedies. It has rapid respirations, and the stethoscope reveals very fine crepitation. I have verified it frequently.

Antimonium tartaricum is somewhat similar to *ipecac*, except that it has coarse rales.

Phosphorus may be used if the patient is hoarse and if there is a rawness under the sternum.

Tuberculinum in the higher potencies has been reported to be of value.

Kali bichromicum is useful in a more or less constant cough with a tough, stringy mucus.

Arsenic, bryonia and other remedies may be indicated.

PULMONARY EMPHYSEMA.

(Greek, ἐμφυσαν, to inflate.)

Synonym: Alveolar Ectasis.

Definition: An unnatural distension of the air vesicles of the lungs with rupture of their walls. It may be caused by excessive respiratory effort, as in whooping cough, playing of wind instruments, etc. It is frequently associated with other conditions of the lungs.

Historical Note: First described by Laennec. Our knowledge of the disease has been added to by Rokitansky and, in 1857, by Sir William Jenner.

Etiology: Emphysema may occur at any age. In children it sometimes follows whooping cough, measles, or other diseases having bronchitis as part of the condition.

Musicians playing wind instruments, glass blowers or others using forced expiration are subject to it. Those working at dusty trades or occupations causing chronic bronchitis are subject to it. Those engaged in continued severe muscular effort; we have many such cases at the Metropolitan Hospital, broken down laboring men who have worked at hard physical labor for years. More rarely the onset of emphysema may be sudden due to excessive physical exertion. Some persons removing to high altitudes will develop emphysema after a time. Some patients are supposed to have an hereditary predisposition to it.

Morbid Anatomy and Pathology: (1) In the *hypertrophic* form the chest is barrel shaped. When the chest cavity is opened the lungs do not collapse, but may bulge outward. The anterior margins may be in contact all the way beneath the sternum. The diaphragm is depressed. The lungs are light in color, soft, and pit on pressure. (2) In the *atrophic* form, senile emphysema. The chest is narrow and contracted, the lungs are dark and collapse when the chest is opened. The walls of the vesicles are wasted away and the air cells run together. This form occurs only as a part of a general atrophy of all the organs and tissues of the body.

Symptoms: The onset of emphysema may not cause any special symptoms at first. When the disease is well advanced, dyspnea, on exertion, becomes a marked feature. When at rest the patient is not troubled with it. Later on there are attacks of asthma. Cyanosis is another marked feature. This may be present even when the patient is not in distress from dyspnea.

Cough is more or less constant, due, probably, to the associated bronchitis. With the bronchitis there is also a certain amount of expectoration.

Sometimes hemoptysis occurs.

The blood pressure is low. Polycythemia may be present.

Arteriosclerosis is often present in late stages of the disease.

Patients with emphysema are usually worse in the winter when the weather is cold and changeable. In warm weather they do very well.

There is a more or less characteristic facial expression. The patient is somewhat cyanotic, looks depressed, and the lines of the face are marked. The fingers are often clubbed.

There is a general venous obstruction, due to the damming back of the blood in the lungs. In time the heart gives way and we have failing compensation with all its evils.

There is passive congestion of the liver and kidneys. Sometimes the liver is enlarged. The urine is scant and high colored.

Physical Signs: *Inspection* reveals the so-called "barrel-shaped" chest. The antero-posterior and lateral diameters of the chest are increased because of the increase in the dorsal curve of the spine, and the more nearly horizontal position of the ribs. The patient's back is rounded. The clavicles are prominent.

The accessory muscles of respiration are called into play. The respiratory movements are lessened in extent. Expiration is prolonged.

A line of dilated veins frequently show over the line of attachment of the diaphragm. There is an epigastric impulse with the heart beat.

Palpation. Vocal fremitus is diminished. The heart impulse over the cardiac area is feeble, but there frequently is an epigastric impulse. Pressure at this point increases the pain markedly. The tenderness is said to be due to the morbid condition of the right side of the heart.

Percussion. There is hyperresonance on percussion. The area of heart dulness is lessened or lost. The liver is at a lower level than normal—sometimes the spleen also.

Auscultation. The breathing sounds are weaker than normal, as a rule. The expiratory sound is much prolonged. Coarse rales are frequently heard.

The heart sounds, owing to the heart being covered by the lungs, are usually feeble. A tricuspid murmur may appear late. The second sound over the pulmonary valve is accentuated.

X-Ray Examination. According to Fowler (Allbutt's System of Medicine) a skiagraph may be of service. The lungs are clearer and larger than normal. The diaphragm is low down in the chest. The heart is more vertical.

Although emphysema is often of slow development, it may be brought on suddenly by excessive physical exertion. When once developed it is practically incurable. In 1913 I had a case of sudden onset at the Flower Hospital. A man, aged 34, had rowed twelve miles, and then returned by motor boat without a coat. A day or two later he became troubled with shortness of breath. On entering the hospital he was in great distress from dyspnea. The chest was distended and the voluntary muscles were called into play in breathing. Resonance was increased. Auscultation revealed whistling and musical sounds in the lungs and a much prolonged expiratory murmur. He improved slightly with rest. He was given *bryonia.*

Complications and Sequelæ: Bronchitis is usually a part of emphysema. An intercurrent pneumonia may prove fatal. Tuberculosis may be a complication.

Chronic heart disease is not at all infrequent. Failing compensation and dropsy sometimes supervene.

Arteriosclerosis and chronic interstitial nephritis are sometimes complications.

Diagnosis: The diagnosis is usually easy and can be made on the characteristic appearance and physical signs.

Prognosis: The tendency of the disease is to progress to a fatal termination. It is very chronic and may last for years. Failure of the heart with dropsy is the most frequent cause of death. An intercurrent pneumonia may close the picture. Rarely pulmonary hemorrhage is the end. Complications affect the prognosis.

Treatment: A suitable climate where the patient can be free from bronchitis, and the ability to live free from worry and work will prolong life.

Fowler recommends compressed air.

Strümpell recommends compressing the chest on expiration for five or ten minutes every day. It must be done by another person exerting pressure on the lower sides of the chest.

The medicinal treatment must be purely symptomatic.

Arsenic—dyspnea with great anxiety and restlessness, face cyanotic.

Chlorine—inspiration easy, expiration difficult.

Terebinth—distention of air vesicles, apex beat in pit of stomach, least exertion causes dyspnea.

Ammonium carbonicum may be of service in those very sensitive to cold air. Patient feels oppressed for air on exertion. Old people.

Antimonium arsenicum is said to be useful with excessive dyspnea.

Lobelia is also mentioned by Boericke.

PULMONARY ATELECTASIS.

(Greek, ἀτελής, imperfect; ἔκτασις, expansion.)

Synonyms: Compression of the Lungs. Collapse of the Lungs. Apneumatosis (without inflation).

Definition: Atelectasis in the newborn is failure of the air cells

to dilate. Later on it is more correctly termed apneumatosis, and is collapse of the lung due to blocking of the smaller bronchial tubes.

Etiology: In weak and puny newborn babes atelectasis may be congenital.

The acquired form is more often found in children than in adults, and is always due to something that interferes with the free access of air to the air cells. This may be plugging of the smaller bronchioles with mucus, when the imprisoned air is finally absorbed and the cut-off portion collapses; or it may be due to pressure of effusion or tumor which prevents the lung expanding; or it may be due to rickets, associated as it is with a softening of the bony structure and narrowing of the chest.

Severe bronchitis, whether a disease by itself or a complication or sequel of measles, whooping cough or other condition, is the most frequent cause of bronchial plugging. Compression may be due to effusions, tumors of the chest or abdomen, to spinal deformity; in short, to any cause bringing pressure to bear on the lungs.

Morbid Anatomy and Pathology: In congenital atelectasis large areas of the lung, sometimes whole lobes, are affected. The extreme lower portions and the apices are especially prone to collapse.

In the acquired form isolated areas scattered throughout the lung tissue are affected more often, except where pressure of an effusion or a growth may compress a large area. In recent cases the lung may be reinflated with a blow-pipe after removal from the body. In cases of long standing the lung tissue where affected has become carnified.

On examination the collapsed portions are found to be depressed below the normal lung tissue, and to have a soft leathery feel. They are of a dark blue color, airless and sink in water.

Very frequently there will be found compensatory emphysema in other portions of the lung if the collapsed portions are considerable. There may also be venous congestion and even edema.

Symptoms: Atelectasis as usually encountered is a symptom or

complication of some other condition, and unless a large amount of lung tissue is involved may be suspected rather than definitely demonstrated.

In marked cases the breathing is quite characteristic. It is more rapid than normal, is shallow, and if the collapse is in the lower part of the lung the breathing is carried on by the upper and anterior portions. In the lower portions of the chest there is inspiratory retraction. In cases that develop rapidly death may occur abruptly.

Physical Signs: Unless a considerable portion of the lung is at fault physical signs may be uncertain. The signs on *inspection* and *palpation,* as noted above, are rapid respiration with the breathing noticeably in the upper part of the chest. *Percussion* may detect limited areas of dulness. *Auscultation* gives us bronchial breathing and rales, which may be fine or coarse. Or if the affected area is considerable, breathing sounds may be faint or absent. Strümpell speaks of a frequent form of mild atelectasis in those long sick and who have remained in one position in bed where, on first sitting up, crepitant rales may be heard, which disappear after a few deep inspirations.

Complications and Sequelæ: Atelectasis is itself a complication. It may cause emphysema in other parts of the lung. When extensive it may produce enlargement of the right side of the heart, venous stasis and pulmonary edema.

Diagnosis: The diagnosis must be made partly on the antecedent disease and partly on the symptoms and physical signs. In cases of bronchitis a progressive distress in breathing and lividity, without an added rise in temperature or special physical signs, should suggest the onset of atelectasis.

The various forms of pneumonia must be differentiated. In pneumonia fever is an important factor. In atelectasis the temperature is not disturbed or may fall with the increase in other symptoms.

Prognosis: In the congenital form, if the collapsed portion of lung is not great, the prognosis may be favorable. In the acquired form it is always doubtful.

Treatment: In catarrhal pneumonia, or in the various forms of

bronchitis, the patient must not remain in one position too long. Care in this may prevent the onset of atelectasis.

The primary disease is the main thing to be treated.

PULMONARY EDEMA.

(Greek, οἰδέω, to swell.)

Synonym: Edema of the Lungs.

Definition: Pulmonary edema is an exudation of fluid into the interstitial tissue and into the alveoli of the lungs.

Etiology: Pulmonary edema is practically the end result of some other condition, notably of Bright's disease, of heart disease, of profound anemia, of cerebral conditions, and frequently occurs in the death agony, no matter what the cause of death.

Morbid Anatomy and Pathology: In some cases the edema seems to be the result of stasis. In others it is due to paralysis of the left ventricle of the heart. In others again it is due to unknown changes in the walls of the blood vessels.

Symptoms: In pulmonary edema occurring during the course of other diseases we have an aggravation of all the symptoms. The pulmonary condition causes marked dyspnea. The respirations are rapid and labored. The accessory muscles of respiration are brought into play. Cyanosis develops and increases.

Percussion is not much changed from normal. *Auscultation* reveals fine and coarse moist rales generally distributed. There may be a large amount of frothy, bloody expectoration.

Complications and Sequelæ: In most cases the end is death. In some cases of renal and cardiac disease the condition may be relieved to return at some future time.

Diagnosis is usually easy, as it is an end result.

Prognosis is usually bad. At the best it is very doubtful.

Treatment: Prophylactic treatment consists in watching the heart in those conditions where edema is a possibility.

Camphor or ether every half hour may be used hypodermatically in extreme cases. Earlier, *strophanthus* in ten drop doses of the tincture may be used. Black coffee may also help.

Adrenalin will give relief in some cases. Adrenalin causes death in rabbits, experimentally, by producing edema of the

lungs. It is best used hypodermatically in a ten per cent. solution of the commercial preparation. I reported two cases in which adrenalin was used successfully in this way in the *Hahnemannian Monthly* for February, 1911.

Apis is the most reliable homœopathic remedy. I have seen it cure patients who appeared to be water-logged. It takes a day or two for results to show.

Antimonium tartaricum will relieve in cases with loose, coarse rales, the patient is almost in collapse.

PULMONARY CONGESTION.

Synonym: Congestion of the Lungs.

Etiology: Congestion of the lungs may be (1) active or (2) passive.

(1) Active congestion is the first stage of inflammation. This is well recognized as the first stage of pneumonia. Osler states that some French authors have designated active congestion as a distinct syndrome, but that their descriptions are "of an affection which is difficult to recognize from anomalous or larval forms of pneumonia."

The conditions which cause pneumonia,—chilling, exposure, etc., would cause active congestion of the lungs first.

(2) Passive congestion can more properly be described as a disease entity. It may occur from mechanical causes, as pressure from tumors, or obstruction due to disease of the heart (brown induration). Or it may be what is known as "hypostatic congestion," found in patients who have been confined to the bed for a long time. This form is supposed to be due partly to gravity, and partly to a weak heart.

Morbid Anatomy and Pathology: (1) Active congestion is an excess of blood in the lungs—an acute condition.

(2) Passive congestion produces an engorged lung which is large and heavy, and brownish in color. The capillaries are distended, the connective tissue is increased, the cells are filled with degenerated blood pigment.

Symptoms: (1) In active congestion we have a chill followed by sudden rise in temperature, full pulse, pain sometimes in the chest, increased respirations, possibly cough.

Physical signs reveal little, possibly a change in resonance and a few fine rales.

(2) In passive congestion the physical signs are very similar. The temperature and pulse will be that of the associated disease.

Diagnosis: A diagnosis can be made only by the careful weighing of all the symptoms.

Prognosis: The prognosis must depend on the underlying cause. The active form most often progresses to pneumonia. The passive form is dependent on relief of the condition producing it.

Treatment: Avoidance of exposure is the best prophylaxis of the active form. Attention to the heart in long illnesses will assist in preventing the passive form. Tumors will have to be removed.

Aconite and *ferrum phosphoricum* are both useful in active congestion and may promptly relieve it. Either one must be given very early in the condition.

The *aconite* patient is frightened, the *ferrum phosphoricum* patient is not; I have verified both.

PULMONARY HEMORRHAGE.

Synonyms: Hemoptysis. Hemorrhage from the Lungs.

Etiology: The most frequent cause of hemorrhage from the lungs is undoubtedly pulmonary tuberculosis. Occasionally pneumonia will begin with quite a profuse hemorrhage. Gangrene and abscess of the lung may cause pulmonary hemorrhage. Heart lesions may cause it. Rarely hemorrhage from the lung is due to vicarious menstruation. Finally infarct may cause it.

Morbid Anatomy and Pathology: Pulmonary hemorrhage—hemoptysis—is due to erosion of a blood vessel, whence the blood reaches the bronchial tubes and is expectorated.

Symptoms: Hemorrhage from the lungs always comes on suddenly and is usually unexpected. It may be scanty, showing itself simply as blood-streaked sputa, or it may be profuse—pure blood in smaller or larger quantity.

When the bleeding is continued for any length of time, even if the quantity is not great, the irritation produces cough. In some cases the blood will rise to the throat and cause the patient to cough it up. This may continue for a few minutes, the loss of blood not being very great. It may continue more or less interruptedly for days. I have seen a hemorrhage of this kind go on for several weeks, the patient eventually bleeding to death. There may be repeated small hemorrhages at intervals of hours or days.

In other cases there will be a sudden flux of blood to the mouth almost choking the patient to death. The blood may come so fast that it gushes from the nostrils and mouth. Hemorrhages of this character, if they do not stop spontaneously, will cause death in a short time.

Sometimes, in pulmonary tuberculosis, a sudden profuse hemorrhage will end the scene. I have seen a number of cases die in this way in a few minutes with their first hemorrhage.

If the amount of blood is considerable, whether a small flow lasting a long time or a large flow all at once, it may literally drown the patient. Occasionally the irritation will induce a pneumonic process and the patient die of that.

As a rule, pulmonary hemorrhage alarms the patient very much, especially if it is a first hemorrhage. I know from personal experience. The patient will be highly nervous and apprehensive. The heart will beat tumultuously, and the pulse be much accelerated. The temperature will go up. The respirations will be increased.

Examination of the chest with the stethoscope will usually locate the seat of trouble. Fine crackling sounds will be heard, mostly over a very limited area in the ordinary case.

The blood in hemoptysis is bright red and frothy. It may or may not be mixed with sputa.

Complications and Sequelæ: In early cases without much bleeding there may be no special complications. In others more severe a pneumonia-like process may result. In others again considerable quantities of blood may be swallowed, only to be vomited up later. In late stages of tuberculosis, or in case of ruptured aneurism, death may ensue promptly.

Diagnosis: As a rule, hemoptysis is easily diagnosed. The blood is bright red, frothy and is coughed up.

Bleeding from any part of the nose, mouth or throat must be eliminated. I have known of cases where blood from the posterior nares or from the gums has run into the throat, caused a cough, and on expectoration given needless alarm to the patient.

The blood in hematemesis—from the stomach—is dark colored and is vomited, not coughed, up.

Prognosis: In the vast majority of cases hemoptysis ceases spontaneously. The immediate prognosis is good. If the hemorrhage continues for hours or days the prognosis becomes rapidly worse. In advanced cases of tuberculosis the prognosis is always grave, and the case may be immediately fatal. In any case prognosis should be guarded at first. As time goes on without a recurrence the physician may have more confidence in the favorable outcome of the case.

Treatment: Absolute physical rest is the first requisite. Next the patient must be reassured as far as possible so as to induce mental rest.

Most patients feel better in a reclining position with the head somewhat high. Osler says turn the patient on the affected side if known.

Morphine is recommended by most authors to quiet the patient. It has no effect otherwise on the bleeding. It may even do harm by stopping the cough necessary to get rid of the blood.

Ergot I cannot recommend.

Aconite is a most excellent remedy, and is used by those of largest experience in the old school, as well as by the homœopath.

Ferrum phosphoricum is my first choice. I have a standing order to use that in my wards at the Tuberculosis Infirmary of the Metropolitan Hospital. Its effectiveness in ordinary cases of hemoptysis has been clinically verified many times. I use the 6x potency.

Ipecac is one of the most useful remedies in frequently recurring small hemorrhages.

Hamamelis is sometimes of service in less active cases.

Millefolium seems to act sometimes when the sputa is simply blood streaked.

HEMORRHAGIC INFARCT OF THE LUNGS.

(Latin, *infarcio,* to fill in.)

Synonym: Pulmonary Apoplexy.

Definition: Hemorrhagic infarct of the lungs is a plugging of a part of a pulmonary vessel by thrombus or embolus, causing infiltration and hemorrhage into the air cells.

Historical Note: We are indebted to Virchow and Cohnheim for our knowledge of the pathology of this condition.

Etiology: Hemorrhagic infarct of the lungs is usually the result of thrombi from heart disease, less often it comes from thrombi formed in the veins. A large majority of the cases that I have seen have been in men.

Morbid Anatomy and Pathology: The infarction may be single or multiple. It may involve a small or it may involve a considerable area of lung tissue—nearly a whole lobe sometimes. It is dark in color, firm in consistency, and wedge or cone shaped. The base is towards the periphery of the lung and may be seen and felt through the pleura. Fibrinous pleurisy occurs at this place. Red cells and leucocytes are found in the occluded air cells. Occasionally an infarct is reabsorbed, leaving scar tissue and pigmentation of the affected area behind.

Symptoms: Small infarcts may cause no symptoms. Occlusion of a large pulmonary vessel may cause instant death. We may assume that a pulmonary infarct has formed when a patient with an acute or infectious endocarditis suddenly develops pain in one side of the chest, dyspnea more or less marked, and a more or less bloody and frothy expectoration. If the infarct is a large one an area of dulness may be mapped out over the affected area. Occasionally the condition is mistaken for pneumonia.

A patient in my service at the Metropolitan Hospital in 1903 came in with infective endocarditis. Careful inquiry into his history led to a diagnosis of gonorrheal endocarditis. A few days later he suddenly began to expectorate frothy blood-streaked mucus, he had pain in the chest, and suffered intensely from dyspnea. These symptoms led to a diagnosis of pulmonary infarct. Both conditions—the gonorrheal endocarditis, and the

pulmonary infarct—were confirmed at the autopsy. Gonococci were found in the heart muscle.

Sometimes the pulmonary condition is preceded by an embolus elsewhere. A patient in my service at the Flower Hospital in 1911, suffering from acute endocarditis with failing compensation, suddenly developed marked edema of one arm. It was diagnosed as due to embolus, and the prognosis made that death would probably occur instantly and might happen at any moment. The prognosis was fulfilled two or three days later.

Complications and Sequelæ: Infarct is usually an end result. Occasionally infarcts are reabsorbed. Sometimes an infarct breaks down into an abscess, or it may cause gangrene.

Diagnosis: The diagnosis of infarct may be made when a patient with endocarditis suddenly develops pain in the chest, dyspnea, and bloody expectoration. A large infarct may be mistaken for pneumonia on account of similar physical signs—rapid respirations, increased fremitus, dulness on percussion and so on. But the temperature curve of pneumonia is somewhat typical, whereas infarct may occur without fever.

Prognosis: The prognosis is always bad, recovery being the exception. As a rule, the patients die either at once, or in a very few days, from the lung condition or from embolus elsewhere.

Treatment: The accident of infarct may be guarded against to a certain extent by keeping the patient with a bad heart absolutely at rest.

The underlying heart condition requires the utmost care. After the accident has happened I know of nothing that will act directly on the condition. The only thing to do is to prescribe absolute rest and meet symptoms as well as possible.

ABSCESS OF THE LUNGS.

Synonyms: Suppurative Pneumonitis. Pulmonary Abscess.

Definition: Abscess of the lung is where the lung tissue itself breaks down and forms pus.

Etiology: The most frequent cause of abscess of the lungs is: (1) Deglutition or aspiration pneumonia. It may follow other pneumonias, either catarrhal or lobar. (2) Extension of pus

from neighboring structures by perforation. (3) Metastatic abscess in pyemia. (4) In association with pulmonary tuberculosis.

Bacteriology: The streptococci, pneumococci and staphylococci have all been found.

Morbid Anatomy and Pathology: Abscess cavity or cavities of varying size are found in the lung.

Symptoms: Abscess of the lung may be suspected when a case of pneumonia does not clear up properly, and the patient instead of getting better runs into a septic condition. An irregular type of fever develops. The expectoration becomes purulent and offensive. On examination with the microscope it is found to contain shreds of elastic lung tissue. Physical signs of cavity may be found.

Complications and Sequelæ: Abscess of the lung may be a part of a general pyemic condition.

Diagnosis: The physical signs of cavity, together with purulent sputa containing elastic lung tissue, make the diagnosis certain.

Empyema will present the same general symptoms, but the expectoration will not contain elastic lung tissue.

Prognosis: The prognosis is usually bad, although occasionally cases recover.

Treatment: The treatment is not satisfactory and must be symptomatic.

Sometimes such remedies as *arsenic, hepar, silicea* and other homœopathic drugs indicated in pus conditions will be of assistance.

A few cases of large single abscesses have recovered after surgical interference.

GANGRENE OF THE LUNGS.

(Greek, γαγγραίνα, a sore.)

Definition: Death of lung tissue.

Historical Note: The disease was first described by Laennec.

Etiology: Gangrene of the lung is always due to the entrance of the bacteria of putrefaction. The lung tissue must be in impaired condition to develop them. Disease of the lung itself is the most frequent predisposing cause, as pneumonia, pulmon-

ary tuberculosis, etc. It most often follows the inhalation form of pneumonia, or inhalation of particles from the upper air passages. It may follow infarction. It sometimes occurs in diabetes.

Bacteriology: The staphylococcus may be found in the expectoration.

Morbid Anatomy and Pathology: There are two forms. (1) The diffuse, which takes in a large area of lung tissue. The diseased tissue is greenish, mushy, soft and without definite line of demarcation. It may follow the circumscribed form. (2) The circumscribed form is in small areas, either single or multiple. At first dry, then later it becomes soft and breaks down. In either case a bronchus may be entered and a foul expectoration coughed up.

Symptoms: Cough, with an exceedingly foul and fetid expectoration, is the leading symptom. There is no odor comparable to it, fortunately. If put in a conical glass the sputa separates in three layers, as is the case in the expectoration of fetid bronchitis. The fever is irregular. The patient greatly prostrated.

Physical signs of cavity may develop. Fatal hemorrhage sometimes occurs.

There is always an associated bronchitis.

Complications and Sequelæ: Fatal hemorrhage may occur.

Diagnosis: Is difficult between fetid bronchitis and abscess sometimes.

Prognosis: Is always grave.

Treatment: Is unsatisfactory. A carbolic spray may be used. Osler says *guaiacol* has been used hypodermatically.

Raue recommends *arsenic, carbolic acid, carbo vegetabilis, kreosote* and *silica.*

PNEUMOKONIOSIS.

(Greek, πνευμων, lung, κονις, dust.)

Definition: Disease of the lungs due to the inhalation of dust.

Historical Note: Zenker was the first to describe the effect of continued inhalation of dust on the lungs.

Etiology: Pneumokoniosis is caused by the continued inhalation of dust. It is essentially an occupation disease. Thus, we

have *anthrocosis,* coal miner's disease; *chalicosis,* stone cutter's disease; *siderosis,* metal worker's or grinder's disease; *kaolinosis,* clay worker's disease; and so on. Persons working in dyes, in grain mills, in tobacco factories are also subject to pneumokoniosis, due to the dust of their respective callings.

Morbid Anatomy and Pathology: All of us breathe a certain amount of dust, and dwellers in cities breathe enough to cause pigmentation of the lungs. In cases that become pathological there is a bronchopneumonia followed by the formation of new connective tissue. The lungs contain nodules which are hard and gritty. The dust is taken up by the leucocytes and is carried to the lymphatics, and deposited in the bronchial lymphatic glands. The nodules are most often found in the apices and on the surfaces of the lungs, although there may be large areas affected, formed by the coalescence of smaller nodules. There is bronchitis, and often emphysema.

Symptoms: The patient has chronic bronchitis. There is cough. The expectoration shows discoloration, due to the particular kind of dust inhaled. As the condition grows more marked emphysema develops. Later the patient may have all the symptoms of pulmonary tuberculosis.

Complications and Sequelæ: The cases frequently end in pulmonary tuberculosis.

Diagnosis: The history of the case is the principal clue to the diagnosis.

Prognosis: This depends on the stage of the disease. Also on whether or not the patient can give up his occupation.

Treatment: First prophylactic. The workshop should be well ventilated. The workmen should use respirators. The patient should live under as good hygienic conditions as possible.

Symptoms must be treated as they arise, although it is obvious that no headway can be made if the cause of the disease still persists unchanged.

CIRRHOSIS OF THE LUNGS.

(Greek, κιρρος, yellow.)

Synonyms: Chronic Interstitial Pneumonia. Fibroid Phthisis. Contraction of the Lung. Sclerosis of the Lung.

Definition: An increase of connective tissue in the lungs tending to replace normal lung tissue.

Historical Note: Cirrhosis of the lungs was first described and named by Corrigan, of Dublin.

Etiology: Increase in the connective tissue of the lungs is caused by all inflammatory conditions of those organs. Localized areas are caused by the presence of tumors, abscesses, syphilis, infarcts, and so on.

Pneumonia, tuberculosis and inflammatory pleuritic diseases may cause the diffuse type.

Morbid Anatomy and Pathology: In an advanced case of the massive form of the disease the chest wall is sunken in. The whole lung is often affected. The lung, shrunken and airless, is crowded into a small space. The heart is drawn to the affected side. The lung itself is hard and firm. The new formed tissue has a grayish appearance. There may be evidence of tuberculosis. There is always evidence of catarrhal inflammation.

In the disseminated form the areas are small, are scattered, and are apt to be bilaterally distributed. The condition is more frequent in the lower lobes.

Symptoms: The disease is essentially chronic. The only symptoms may be cough and a slight shortness of breath. Hemoptysis sometimes occurs.

Advanced cases may be somewhat cyanosed as well. The patient is usually well nourished and without fever.

Physical Signs: *Inspection.* When the case is marked the affected side of the chest is smaller than the other, the chest walls are more or less collapsed, the shoulder on that side is lower than the other. On respiration the affected side is restricted in motion. The heart is displaced to the affected side.

Palpation may give increased vocal fremitus.

Percussion is either dull or flat over the affected side, and may be hyperresonant over the well side. The heart becomes hypertrophied. The liver and intestines may be displaced upward.

Auscultation gives exaggerated voice sounds. The breathing sounds may be weak or absent.

The unaffected lung may be emphysematous.

As noted above, the heart is displaced toward the affected lung. The right side may finally give out from the extra work thrown on it and dropsy may appear

Complications and Sequelæ: Tuberculosis may develop. Heart failure may appear. Hemorrhage sometimes occurs.

Diagnosis: In marked cases the diagnosis ought not to be difficult. Pleurisy with contraction never has hemorrhage. The history of the case will help to distinguish contraction due to new growths.

Prognosis: The prognosis is good, as a rule, because the disease tends to run a chronic course. If tuberculosis supervenes, or if the heart begins to fail, the prognosis is not good.

Treatment: The treatment is mostly hygienic. The patient if possible should live in a warm climate. Out of door living with good nourishing food are the essentials. Any symptoms that arise must be met.

NEW GROWTHS IN THE LUNGS.

New growths in the lungs include benign tumors, carcinoma, sarcoma, echinococcus, and syphilitic changes.

Morbid Anatomy and Pathology: This will vary with the lesion so far as the growth itself is concerned. The tumor causes compression and atelectasis of the surrounding lung tissue. If the growth is large compensatory emphysema of the sound parts of the lung may take place. Or there may be congestion, edema, and even hemorrhage, due to pressure.

Pleuritic effusion may complicate malignant tumor.

Symptoms: The most general symptom is a progressive dyspnea. There is apt to be a feeling of pressure and distress in the chest as the tumor grows. There is cough which is more or less spasmodic and labored, according to the location of the tumor. Strümpell says the expectoration of cancer sometimes assumes a characteristic consistency, containing blood and being "currant jelly-like" in appearance. It may also contain elements of the tumor, which may be found with the microscope. Hemoptysis may occur, especially with cancer.

Physical Signs may enable one to map out the tumor. If the

tumor is large and near the surface there may be a visible swelling and slight edema of the skin over the affected area.

Complications and Sequelæ: In malignant disease we may find swelling of the lymph glands of the neck and axilla. This is of great value in diagnosis. Tumors in the lung may cause pneumonia, pleurisy, gangrene or atelectasis. All conditions caused by the presence of a foreign element.

Diagnosis: The diagnosis of new growth in the lung may be impossible or difficult. Cases without cardiac or pulmonary lesion presenting dyspnea, distress in the chest, expectoration tinged with blood, may be suspected of lung tumor. In cancerous conditions, swelling of the glands and final development of cachexia will be of great help.

The aspirating needle and the x-ray may be resorted to in doubtful cases.

If echinococcus cysts exist the hooklets may be coughed up.

Syphilis of the lungs may occur, but is rarely marked.

Prognosis: Malignant disease of the lungs is fatal. In echinococcus the sac may rupture into a bronchus and the offenders be coughed up and the patient recover.

Treatment: Is very unsatisfactory. The symptoms must be met as they arise. Echinococcus may be treated surgically. Syphilis of the lung requires anti-syphilitic treatment. Cancer of the lung has been operated on successfully a few times.

PLEURISY; DRY; WITH EFFUSION; EMPYEMA.

Synonym: Pleuritis.

Definition: Inflammation of the pleura.

Forms: There are many forms of pleurisy. It may be acute, subacute or chronic. It may be dry (without effusion) or with effusion. The effusion may be sero-fibrinous or purulent. The pleurisy may be primary or it may be secondary to some other condition. Finally, Strümpell mentions a peripleuritis. These conditions may be found in varying combinations. Clinically, it is perhaps simplest to distinguish between: (I.) *Dry pleurisy;* (II.) *pleurisy with effusion,* although the first sometimes runs into the second, and (III.) *empyema,* a condition where the effusion has become purulent.

Etiology: I. *Dry pleurisy, acute plastic* or *fibrinous pleurisy.*

This form of pleurisy may be. (1) Primary, due to exposure to cold; it may be of traumatic origin; it may be rheumatic in character. It is more common in cold weather. It is said to be rare in perfectly healthy persons.

(2) It may be secondary to some other disease of the lungs. It is nearly always present in pneumonia, and is frequently present in pulmonary tuberculosis. It may also occur in other lung conditions, as in infarct, abscess, cancer or gangrene. It may be associated with chronic Bright's disease, alcoholism or with inflammation of other serous membranes.

II. *Sero-fibrinous pleurisy, pleurisy with effusion.*

This form has the same etiological factors as the preceding.

III. *Empyema.*

The effusion of sero-fibrinous pleurisy may become purulent, or the effusion may be purulent from the start.

Bacteriology: According to Anders all forms of pleurisy are caused by bacteria or their products. The bacteria vary with the underlying condition; one may find the pneumococcus, the tubercle bacillus, the typhoid bacillus; frequently the streptococcus, the staphylococcus or some other form of bacteria.

Morbid Anatomy and Pathology: (I.) An exudate is thrown out on the adjacent layers of the pleura, the latter appear congested and dull. There are numerous embryonic cells and these may result in the formation of new tissue and adhesions. The layers of the pleura may become much thickened.

(II.) In pleurisy with effusion we have an abundant serous or sero-fibrinous exudate thrown out between the layers of the pleura. Sometimes adhesive bands run through this. More often the fluid accumulates in the lower part of the chest. It varies in amount, as much as four quarts has been found. If the quantity is very great it may crowd the lung back against the spinal column, and squeeze all the air out of it—atelectasis. The heart may be displaced by an excessive amount of fluid.

This last may become purulent, when we have (III.) empyema. The effusion may be purulent from the start.

Symptoms: (I.) Pleurisy usually begins with a chilliness or

feverishness after exposure. There is a certain amount of pain in the affected side, a stitch-like pain, on breathing. The patient has a slight rise in temperature. The pulse is slightly accelerated. Occasionally the onset is abrupt and the symptoms very severe.

If effusion takes place the pain on breathing disappears. Usually there is cough that causes pain.

(II.) Very often in effusion the onset will be so mild that the patient does not know he has pleurisy until the amount of fluid is enough to cause discomfort. I have seen cases where pleurisy was not suspected until an x-ray picture showed the effusion.

In cases with a large amount of fluid there is marked dyspnea. The pulse may be rapid and weak on account of pressure on the heart. There may be some rise of temperature.

(III.) If the effusion is purulent we have empyema. This may come about through a change from a sero-fibrinous to a purulent fluid, or the effusion may be purulent from the start. I have seen a number of cases during the past few years where empyema followed lobar pneumonia. The cases ran a typical pneumonia course until the expected crisis when an irregular septic fever supervened, with sweats, chills, rapid and weak pulse, etc., and the patient had every appearance of being extremely ill. Physical signs pointed to fluid, and the aspirating needle revealed pus.

Physical Signs: On *inspection* we see restricted motion on the affected side. In the dry form, with considerable pain. The patient is apt to lie on the affected side, if in bed, so as to restrict the motion as much as possible. If he has a cough he will try to suppress it on account of the pain. If there is a large amount of fluid present it may give the affected side an appearance of fulness not seen on the opposite side. Dyspnea is evident.

Palpation will reveal the lessened motion on the affected side. If the pleura is much thickened and there is no fluid there may be increased vocal fremitus. More often there is fluid, then vocal fremitus is dulled or lost.

Percussion, over a dry pleurisy, is not changed. Over an effusion dulness, or if very great flatness is found. In this last case there may be an increased resonance on the well side.

Auscultation reveals friction sounds over a dry pleurisy. Over an effusion the breathing sounds seem far away—they are lessened in intensity, and finally lost.

As the effusion disappears the physical signs will gradually return to normal. Friction sounds over the base of the lung may, however, persist for months afterwards.

If there is a large effusion the heart will be pushed away from the affected side so that its apex will be displaced to the right or left as the case may be. The heart's action may be irregular because it is crowded. Often the sounds will be more or less masked.

Pleuritic effusion is impervious to the x-ray, and a radiograph will show a dark place instead of the normal condition.

Complications and Sequelæ: Tuberculosis follows pleurisy after a longer or shorter period in a large number of cases.

Repeated attacks will lead to a thickened pleura.

Pleurisy itself may be a complication of various conditions, as noted in the etiology.

Diagnosis: Dry pleurisy, with its pain on respiration, may be mistaken for intercostal neuralgia, or vice versa. In the latter there are superficial tender spots, and there are no friction sounds.

Pleurisy with effusion and pneumonia are hard to differentiate at times. As a rule, pneumonia is of more sudden onset, the general symptoms are more severe, the fever higher. Pleuritic pain is a frequent accompaniment of pneumonia, but pleuritic effusion is not. Inspection will show restricted motion in both conditions. Lobar pneumonia in the stage of consolidation gives increased vocal fremitus on palpation, pleuritic effusion gives diminished fremitus.

Lung consolidation does not displace the heart or liver—a large effusion does.

Auscultation may be much the same in both conditions.

Pneumonia runs a definite limited course. If it does not clear up the aspirating needle should be used to look for fluid.

Empyema runs a septic temperature.

Prognosis: In dry pleurisy and in sero-fibrinous pleurisy the immediate prognosis is good. The possibility of tuberculosis must never be lost sight of. Even empyema, properly treated, has a favorable prognosis.

Treatment: Persons who have had one attack of pleurisy must guard themselves against exposure as they are particularly susceptible to subsequent attacks.

Local applications may be serviceable for the pleuritic pain. Simple strapping with adhesive plaster will often relieve it. Antiphlogistine is satisfactory in some cases. Others will be benefitted by warm applications.

In effusion the fluid should be withdrawn with an aspirating syringe, care being used not to allow air to enter the chest. The point of election is the sixth intercostal space in the mid- or posterior-axillary line. The fluid must be withdrawn slowly. If any untoward symptoms appear, stop. As much as four pints of fluid are sometimes removed when the effusion is large.

Tapping may have to be repeated a number of times, as the fluid frequently reaccumulates in a few days or weeks.

Empyema requires resection of one or more ribs so that free drainage may be established. This is a distinctly surgical procedure and should be done by one with surgical experience.

When it comes to remedies the homœopath has a distinct advantage.

Bryonia is the most important remedy, it acts directly on serous membranes. It relieves the sticking pains of pleurisy.

Aconite is of service in the beginning of pleurisy as it is in the beginning of any inflammatory condition.

Squilla maritima has dyspnea with stitches in chest.

Kali carbonicum is useful in cases worse at 3 a. m.

Cantharis is useful after effusion has taken place. There is still pain.

Apis is useful when the fluid encroaches on the air space.

Sulphur will often clear up old effusions after other remedies have failed.

Iodium has caused effusion to disappear when the other symptoms, cough, hoarseness and so forth, called for *iodium*.

PERIPLEURITIS.

Peripleuritis is the formation of an abscess between the costal pleura and the ribs. Its treatment is purely surgical.

PNEUMOTHORAX; HYDRO- OR PYO-PNEUMOTHORAX.

Definition: *Pneumothorax* is air in the pleural cavity. It is usually associated with a serous fluid—*hydro-pneumothorax,* or pus—*pyo-pneumothorax.*

Etiology: The most frequent cause is pulmonary tuberculosis. A tubercular cavity opens into the pleural cavity.

Rarely physical exertion will cause a break in a perfectly healthy lung.

External wounds of the chest may cause it.

Breaking through the diaphragm in malignant diseases of the esophagus, stomach or colon may cause it.

It is more common in adults than in children, in men than in women.

Bacteriology: Rarely the gas bacillus in pleural exudates may cause it.

Morbid Anatomy and Pathology: Air is found in the chest cavity under pressure. The lung of the affected side is compressed, the heart is drawn to the normal side. Usually there is more or less fluid in the cavity—serous or sero-fibrinous in hydro-pneumothorax,—purulent in pyo-pneumothorax.

Symptoms: Pneumothorax is found sometimes at autopsy when it was unsuspected during life.

As a rule, the onset is sudden. The patient complains of severe pain in the side; there is dyspnea, with cough, the patient is obliged to sit up; the pulse rate goes up to 140-160; the patient is cyanotic; the patient is often in collapse.

Physical Examination: *Inspection.* The affected side is distended—the intercostal spaces stand out. The side is more or less immobile. The apex beat of the heart is displaced.

Palpation gives diminished vocal fremitus.

Percussion. Over the affected area the note is almost tym-

panitic. This area may extend much lower than is normal. The heart is displaced to the opposite side. The liver or spleen, as the case may be, is pushed downward.

Auscultation. The breathing sounds are usually lost. Sometimes a metallic tingling—amphoric breathing—sound may be heard. Placing a metal pleximeter over the affected area and tapping it with another piece of metal will give a clear metallic sound through the stethoscope. When fluid is present a splashing sound is sometimes heard on shaking the patient.

Change of position of the patient will sometimes give change of position of the percussion and auscultation findings.

Complications and Sequelæ: Pneumothorax is itself a complication. Most often this condition hastens the end of the patient.

Diagnosis: The sudden onset of pain, dyspnea, increased pulse rate, etc., in a case of tuberculosis points to pneumothorax.

The physical signs are somewhat like those of a large cavity. Cavity, however, is usually at the apex, the walls are sunken in, the heart is not displaced. Pneumothorax is at the base of the lungs, the chest is distorted, the heart is displaced.

Prognosis: The prognosis is bad. In tuberculosis the patient usually dies in a few days or weeks at the most. Occasionally cases get well.

Treatment: The patient must be made as comfortable as possible. Some cases will get along without special treatment. Others will require tapping. If pus is present resection of the rib and drainage is indicated.

Homœopathic remedies will be of service, according to the indications. Raue places *arsenic* first on the list.

HYDROTHORAX.

Synonyms: Dropsy of the Pleura. Thoracic Dropsy.

Definition: This is an effusion of serous, non-inflammatory fluid into the pleural cavity. It is not accompanied by inflammation of the pleura.

Etiology: This condition is secondary to diseases that cause dropsy, particularly heart and kidney lesions. It may also be caused by pernicious anemia, leukemia and chronic intestinal

troubles. Or it may be caused occasionally by chronic diarrhea, cancer or syphilis. Sometimes it is due to the local pressure of tumors.

Morbid Anatomy and Pathology: The fluid of hydrothorax is usually a clear serous fluid of low specific gravity—1012 or thereabouts, and has few cells in it. It is usually bilateral. A case in my service at the Flower Hospital in March, 1911, due to heart disease, will illustrate. From the left side 10 oz. of fluid was withdrawn on the 16th, 16 oz. on the 22d, 25 oz. on the 27th. From the right side 16 oz. on the 17th, 48 oz. on the 21st. Patient died suddenly on the 30th from embolus.

The pleural surfaces are smooth and pale.

Symptoms: The patient usually has dropsical symptoms elsewhere. The fluid in the chest causes dyspnea on account of its crowding of the lungs. There is cyanosis and cough.

Physical examination will reveal the fluid. As the lung tissue may be normal and there are no adhesions we may hear bronchial breathing.

Complications and Sequelæ: Hydrothorax is in itself a complication.

Diagnosis: Hydrothorax is always secondary to something else. It is usually bilateral. Pleuritic effusion is usually unilateral. Moreover pleurisy is an inflammatory condition and hydrothorax is not. The case referred to above ran a subnormal temperature.

Prognosis: This depends on the underlying condition. Repeated tapping will give relief to the breathing, but the great majority of patients who reach this stage are near the end.

Treatment must be primarily to relieve whatever has caused the hydrothorax. If the original disease can be cured the hydrothorax will disappear.

Apis is the most valuable remedy.

HEMOTHORAX.

Definition: Blood in the thorax.

Etiology: Usually traumatism. Less often rupture of a blood vessel.

Symptoms, diagnosis, prognosis and **treatment** all depend on the cause.

MEDIASTINAL TUMORS.

Definition: The mediastinum is the space left in the median portion of the chest by the non-approximation of the two pleuræ. It extends from the sternum in front to the spine behind, and contains all the viscera in the thorax excepting the lungs (Gray). A mediastinal tumor is a tumor in this space.

Etiology is unknown.

Morbid Anatomy and Pathology: There may be abscess, aneurysm, carcinoma, sarcoma, lymphadinitis, syphilitic gummata.

Symptoms: The symptoms are somewhat vague. There is usually pain, which may be more or less indefinite in character, or it may be extremely severe. It is more pronounced in the upper sternal region—although it may extend through to the back. If the tumor causes pressure on the brachial plexus the pain extends down the arms. If the pressure is on the trachea, on a bronchus, or on the recurrent laryngeal nerve there is dyspnea. There is usually cough and there may be aphonia. If the tumor presses on the sympathetic there may be irregularity of the pupils.

Physical Signs: *Inspection.* If the tumor is large enough it may be visible. *Palpation* may reveal pulsation and absence of fremitus. *Percussion* will give an area of dullness. *Auscultation* may reveal total absence of breath sounds over the tumor.

It must not be forgotten that pleuritic effusion may be associated with tumor, and the signs of tumor may be masked by the liquid.

Diagnosis: The diagnosis is difficult. The X-ray may be of assistance.

Prognosis is bad except in a very few cases that may be given operative relief.

Treatment consists in palliation of symptoms. Narcotics may be necessary. Surgical interference may be possible in some cases.

SECTION IX.

Diseases of the Digestive Tract.

FOUL BREATH.

Synonym: Fetor Oris.

Etiology: Catarrhal conditions of the nose and throat. The various forms of stomatitis. Unclean or diseased teeth and gums. The use of certain foods, as onions or garlic; or drinking alcoholic liquors. Smoking. Indigestion.

Treatment: Consists in allaying the cause. Catarrhal conditions, stomatitis and indigestion need to be corrected. The use of food or drink that is offensive, or smoking, should not be indulged in if one knows he is to go where his breath may offend. This is particularly true of the physician who, of necessity, comes into such intimate contact with his patients.

The utmost care should be used in keeping the mouth and teeth clean and in good condition.

GLOSSITIS.

Definition: Inflammation of the tongue.

Etiology: There may be direct injury to the tongue by trauma or by irritant poisons, or septic germs may gain access in some unknown way.

Symptoms: There is swelling and pain. Salivation. Eating and talking are almost impossible. The cervical glands are swollen.

Treatment: Ice applied locally. Sometimes scarification is necessary.

For remedies, see next chapter.

LEUKOPLAKIA.

Synonyms: Ichthyosis Lingualis. Lingual Psoriasis. Eczema of the Tongue.

Etiology: May occur in heavy smokers. In the majority of cases the cause is unknown. Many of the patients have had syphilis.

Symptoms: Except for the odd appearance of fissures and patches there may be no symptoms. In other cases the tongue is red and dry and itches. In others there is a whitish coating. Sometimes the tongue will become irritated and sore, and interfere with deglutition and speech.

One patient, a syphilitic woman, from time to time has acute exacerbations. When she wakes in the morning the tongue is dry and movement of the tongue is very painful.

Complications and Sequelæ: It may be the starting point of cancer.

Prognosis: The prognosis, usually, is good, although the case may last a long time.

Treatment: Avoid irritating substances and smoking. Anti-syphilitic treatment seems to have no effect.

Applications of *nitrate of silver* to the spots may give great relief.

In the case referred to above applications of pure *carbolic acid,* followed by *alcohol,* were used with good results. Relief from pain was almost immediate. This treatment has been used at irregular intervals for three years.

Remedies are often of service.

Lachesis has a dry, red, cracked tongue. The gums are swollen.

Muriatic acid has a dry tongue, sordes, on teeth. Aphthæ.

Apis, the tongue is swollen, there may be vesicles on it.

STOMATITIS.

(Greek, στομα, mouth.)

Definition: Inflammation of the mouth. There are several forms of stomatitis: 1. *Catarrhal,* or *simple stomatitis;* 2. *Ulcerative* or *fetid stomatitis;* 3. *Gangrenous stomatitis, cancrum oris,* or *noma.*

Etiology: All of these forms are probably directly due to germ infection of some sort. Many things may act as excit-

ing causes. Any irritant, chemical or mechanical, may cause stomatitis. The excessive use of tobacco, mercury, some of the dust producing trades. The irritating effects of these are facilitated by carious teeth. Stomatitis also occurs with some of the infectious diseases. It may also occur by extension from neighboring parts.

Morbid Anatomy and Pathology: Varies with the severity of the case. It may be a simple catarrhal inflammation, the trouble may be severe enough to form ulceration; in rare cases there may be actual gangrene. (1) The *catarrhal* form may invade the gums, buccal mucous membrane and even the tongue. (2) The *ulcerative* form usually starts at the margin of the gums. Sometimes it is severe enough to loosen the teeth. The ulcers are covered with an adherent grayish membrane. (3) *Gangrene* is rare and usually occurs in poorly nourished children, although it sometimes occurs in adults. The only case I have seen was in a man who had gangrene elsewhere.

Symptoms: (1) The mildest *catarrhal* cases have a redness and dryness followed promptly by increased secretion of the mucous membrane of the cheeks and gums. The tongue is coated. The secretion may become muco-purulent, have a dirty appearance, and be very profuse. Vesicles may appear, break down, and leave superficial ulcers.

There is pain in masticating the food. There may be a bad taste in the mouth and foul breath.

(2) The *ulcerative* form usually starts on the gums, which are inflamed and bleed easily. Ulcers form on the lips and cheeks where in contact with the teeth, and the gums may ulcerate to such an extent as to loosen the teeth. There is salivation, and the discharge is purulent. The breath is very foul. The glands of the throat may be swollen. This form may be epidemic.

As it is difficult to take nourishment the patient becomes weak. There is apt to be some fever.

(3) *Noma,* the *gangrenous* form, occurs most often in poorly nourished children. It usually starts as a small spot, followed by edema of the surrounding parts. Then necrosis sets in and the disease spreads. The decayed tissue sloughs away and is very offensive.

As the disease goes on constitutional symptoms become marked.

Complications and Sequelæ are slight except in noma. Here there may be infective pneumonia or diarrhea; or general sepsis.

Diagnosis: Stomatitis must be differentiated from aphthæ and thrush.

Prognosis: The prognosis is good in the catarrhal and ulcerative forms. It is almost invariably fatal in the gangrenous form.

Treatment: Mouth washes are indicated. This may be a two per cent. *carbolic acid* solution; or a two per cent. *chlorate of potash* solution. Or better, a saturated solution of *boric acid,* or *peroxide of hydrogen* one part to four of water.

Liquid diet is necessary. Cold or lukewarm drinks are preferable; sometimes cracked ice is agreeable. Acids, sweets and condiments should be omitted.

The best remedy is *mercurius solubilis.* The tongue is coated, and bears indentations of the teeth. The gums are affected; spongy and bleed easily, the breath is foul.

Apis has vesicles on the tongue.

Borax has an aphthous condition of the mouth. Fear of downward motion.

Argentum nitricum, excessive secretion in mouth and sensation of splinter.

Kali muriaticum: Grayish white coating of tongue. White spots and ulcers.

APHTHOUS STOMATITIS.

(Greek, απτω, to set on fire.)

Synonyms: Aphthæ. Fibrinous Stomatitis.

Definition: Aphthæ is characterized by small grayish white raised spots with a red areola.

Etiology: Usually occurs in children at time of first dentition. Sometimes it occurs in adults.

Morbid Anatomy and Pathology: There is a thickening of the epithelium and a slight fibrinous exudation.

Symptoms: The disease first appears as little vesicles along the edges of the tongue, on the frenum, and frequently on the inner side of the lips and cheeks. The vesicles soon become cloudy, forming grayish spots with a red areola. The exudate cannot be removed without causing bleeding. Superficial ulcers may form.

The mouth is sore and the patient has difficulty in masticating —in severe cases even in talking. The saliva is increased. Lymph glands may be slightly enlarged. There may be slight constitutional disturbance, fever, etc.

Complications and Sequelæ: Usually occurs with indigestion or some infectious disease.

Diagnosis: Must not be confounded with thrush—aphthæ has no micro-organism.

Prognosis: The prognosis is good.

Treatment: The treatment is the same as for stomatitis.

THRUSH.

Synonyms: Soor. Muguet. Parasitic Stomatitis.

Definition: A stomatitis caused by a fungus, the saccharomyces albicans.

Etiology: Occurs most often in poorly nourished children. Lack of cleanliness in care of bottles and feeding utensils. May occur in adults suffering from tuberculosis, cancer, etc.

Morbid Anatomy and Pathology: Due to a fungus. The microscope shows a mass of tangled filaments and spores.

Symptoms: The mucous membrane is usually dry, red and swollen. Whitish spots appear on the tongue, which gradually coalesce. Thence the trouble extends to the lips, cheeks, and pharynx, covering the entire mucous membrane of the buccal cavity. The membrane can usually be removed leaving the mucous membrane intact. The microscope shows the fungus.

There is a stomatitis. Sometimes one condition appears first, sometimes the other. There may be slight systemic disturbance. The mouth is acid.

Complications and Sequelæ: The fungus may extend down the esophagus to the stomach. Occasionally the fungus penetrates a bood vessel causing thrombosis.

Diagnosis: The microscope shows the fungus.

Prognosis: The prognosis is good.

Treatment: The treatment is the same as for stomatitis.

SECONDARY SUPPURATIVE PAROTITIS.

Definition: Swelling and inflammation of the parotid due to infection. It is a complication of some other condition, not a primary affection.

Etiology: It may occur in any of the acute infectious diseases and is probably due to infection by way of the duct, possibly through the blood.

Injury or disease of the abdominal or pelvic organs may cause it.

Bacteriology: Most often the streptococcus is found introduced from lack of cleanliness of the mouth. Sometimes the germ of the original disease is found.

Morbid Anatomy and Pathology: The gland is swollen. Suppuration is apt to take place. Cross section reveals numerous small abscesses.

Symptoms: The gland rapidly becomes excessively swollen. The patient is unable to open the mouth, so that the taking of nourishment is difficult. There is pain, heat and redness. Suppuration may take place.

Gangrene is possible.

Complications and Sequelæ: If the suppuration is excessive, or if gangrene takes place, there may be permanent injury to the facial nerve with resulting paralysis; or there may be deafness due to extension to the middle ear.

Diagnosis: The diagnosis is easy.

Prognosis: The prognosis is usually good.

Treatment: Prophylactic—cleanliness of the mouth in severe illness is essential.

Applications of cold or heat may be of service.

Open the abscess if suppuration occurs.

Belladonna is almost a specific.

Hepar is necessary if suppuration occurs.

SORE THROAT.

Synonyms: Angina Simplex. Pharyngitis.

Definition: An acute inflammation of the mucous membrane of the pharynx, and usually also of the soft palate and tonsils. It may become chronic.

Etiology: Exposure to cold or wet. Excessive use of the voice. The use of tobacco. Irritating vapors. Constitutional diseases, as gout. Digestive disturbances. Infections. Sometimes extension from neighboring parts. It occurs most often in early life.

Morbid Anatomy and Pathology: There is an inflammation of the mucous membrane affected.

Symptoms: There is frequently slight chilliness and fever. The throat feels uncomfortable. There may be considerable pain, especially on swallowing, sometimes on talking due to extension to the larynx. The patient may be hoarse. There are sticking pains, or the throat simply feels raw and sensitive and tender. It may be worse on one side. Sometimes there is salivation, and patients often feel the need of empty swallowing.

There may be extension to the Eustachian tubes causing slight deafness. Inspection shows the parts to be red and congested.

Complications and Sequelæ: Frequent attacks may leave a chronic sore throat.

Diagnosis: The diagnosis is made from the appearance of the throat.

Prognosis: The prognosis is good.

Treatment: Avoid irritating the throat in any way.

Aconite may be used in the beginning.

Belladonna is useful if the congestion is very great, if there is fever, and if the face is flushed.

Phytolacca is indicated if the tonsils are involved.

Mercurius vivus is also an excellent remedy.

Hydrastis is indicated in cases with a thick, yellow, tenacious mucus.

Sanguinaria is useful in cases with cough, the throat is dry and sometimes ulcerated.

RETROPHARYNGEAL ABSCESS.

Definition: Abscess formation between the posterior wall of the pharynx and the spinal column.

Etiology: Occurs most often in childhood. It may be a complication in the acute infections.

Symptoms: There is restlessness and fever. There is pain on swallowing, and an excessive flow of saliva. Regurgitation of food may occur. The lymph glands are swollen. After a time dyspnea appears due to pressure. A tumor can sometimes be seen, oftener it is only felt by examining with the finger.

Complications and Sequelæ: There is danger of choking or suffocation from spontaneous rupture.

Diagnosis: Diagnosis is made by sight and touch.

Prognosis: The prognosis is good if the condition is promptly discovered and the abscess opened.

Treatment: Surgical. The abscess must be opened with the head erect;—then bend forward to avoid swallowing.

LUDWIG'S ANGINA.

Synonym: Cellulitis of the Neck.

Definition: A rare phlegmonous inflammation of the floor of the mouth.

Etiology: Usually a secondary condition following the infections, as scarlet fever, diphtheria, etc.

Symptoms: The condition starts with swelling of the submaxillary gland and progresses rapidly until it involves the whole floor of the mouth and the neck. Talking and swallowing are painful or impossible.

Fever is a marked symptom.

Complications and Sequelæ: May form an abscess with sloughing and systemic infection.

Diagnosis: The diagnosis is made by sight.

Prognosis: Doubtful.

Treatment: Surgical.

Apis may help the swelling.
Rhus toxicodendron if the glands are swollen.
Arsenic for a general adynamic condition.

ESOPHAGITIS.

Definition: Esophagitis is inflammation of the esophagus.

It may be acute or chronic; it may be catarrhal, follicular, ulcerative, phlegmonous or necrotic.

Etiology: Esophagitis may be part of an acute infection. It may be caused by chemical or mechanical agents, or the drinking of too hot liquids; or it may be due to extension of inflammation from adjacent tissues.

Morbid Anatomy and Pathology: The mucous membrane may become swollen and then desquamate. There may be a croupous inflammation with grayish exudate, or there may be phlegmonous inflammation attacking the submucosa; occasionally there is gangrene.

Symptoms: Mild cases present no special symptoms. More severe ones have pain on swallowing, sometimes with regurgitation of food. This is associated with pain behind the sternum.

In the necrotic form, which may follow swallowing of corrosive poisons, death usually occurs very promptly. If it does not, necrosis takes place, followed by stenosis.

Hemorrhage may occur if the part ulcerates.

Complications and Sequelæ: Perforation may take place. Stenosis is apt to occur.

Diagnosis: The particular form of esophagitis cannot be diagnosed without the history of the case.

Ulceration may sometimes be located by having the patient swallow two feet of silk thread, and leaving it in place two or three hours. The blood on the thread will locate the site of the ulcer.

Prognosis: The prognosis depends on the severity of the case.

Treatment: This is unsatisfactory. External applications of heat or cold may be tried.

In poisoning cases use an antidote.

Olive oil and bismuth may act as a soothing mixture.

Cracked ice will sometimes give relief. Give enemata; no food should be given by mouth.

SPASM OF THE ESOPHAGUS.

Synonyms: Spastic Stenosis. Esophagismus.

Etiology: Esophagismus occurs in hysteria, hypochondriasis, hydrophobia and other nervous diseases.

Symptoms: The patient thinks he cannot swallow. The act of swallowing causes a feeling of constriction about the throat. The passing of a bougie becomes easy after a time, when the spasm subsides.

Treatment: Search out the underlying condition.

Cajuputum is said to be useful.

STENOSIS OR STRICTURE OF THE ESOPHAGUS.

Etiology: Stenosis or stricture of the esophagus may be caused by corrosive poisons. It may be due to cancer, to syphilis, to tumors, to healed ulcers. Tumors outside the esophagus may cause stenosis by pressure.

Morbid Anatomy and Pathology: A stricture may cause a dilatation of the esophagus above the constricted point.

Symptoms: The leading symptom is dysphagia, a difficulty in swallowing. The trouble occurs first on swallowing solids, later the same difficulty occurs on swallowing liquids. The food takes too long to travel from the pharynx to the stomach. This can be detected by auscultation. The stethoscope should be placed to the left of the sixth dorsal vertebra. Normally, in six or seven seconds there is an esophageal bruit, followed from five to twelve seconds later by a splash. In stenosis the sound is obscure or absent.

After a time of difficult swallowing regurgitation of food occurs. Finally, from lack of sufficient food, inanition takes place.

Complications and Sequelæ: If stricture is permanent a dilatation of the esophagus takes place above it.

Diagnosis: An esophageal sound must be passed to make sure of the diagnosis. Kemp says a flexible stomach tube is the best form of sound to use.

H. S. Plummer has devised a method to differentiate between a stenosis and a diverticulum. He has the patient swallow three yards of silk thread at night with aid of water. Next morning the patient swallows three yards more. If there is an opening the thread will be washed through the stomach and bowel far enough to resist being readily withdrawn. A whalebone probe provided with an olive tip, having an opening, is threaded with the free end of the silk and passed into the esophagus until an obstruction is reached. If this is due to stenosis, the tip will not change its level when the thread is tightened. If the tip is in a diverticulum, tightening the thread will elevate the tip. Normally the distance from the teeth to the esophagus is six inches, the esophagus is ten inches long.

Prognosis: The prognosis is doubtful.

Treatment: Except in case of cancer dilatation may be tried with bougies.

It may be necessary to feed the patient through a stomach tube, or it may even be necessary to resort to rectal feeding.

The rectum should be thoroughly cleansed before injecting a nutrient enema.

Enemata may be composed of peptonized food, milk, eggs or wine.

Leube's pancreatic meat emulsion may be given, rectally. This is made up of 5 oz. of meat cut very thin and then minced finely, and 2 oz. of minced pancreas (from the calf), free from fat; these to be stirred with 3 oz. of luke-warm water until the mixture has the consistency of gruel. One such enema may be given daily.

Baptisia may be given as a remedy.

Condurango may be tried.

DILATATION AND DIVERTICULUM OF THE ESOPHAGUS.

Definition: An enlargement of the calibre of the esophagus with thickening of its walls.

Etiology: Dilatation of the esophagus is due to a stenosis below the point of dilatation.

Morbid Anatomy and Pathology: There is a thickening of the walls of the esophagus to overcome the obstruction; then a relaxation and dilatation.

A pressure diverticulum is like a hernia.

Symptoms: There is difficulty in swallowing. The food does not reach the stomach. It lodges in the enlarged part of the esophagus and decomposes. There may be a visible swelling, after taking food, which can be emptied by pressure from without. The patient suffers from lack of nourishment, and may even starve to death.

Complications and Sequelæ: The bottom of a diverticulum may ulcerate. The pouch may rupture into a bronchus, causing choking. Pressure may cause dyspnea.

Diagnosis: A bismuth meal may be given and the patient X-rayed.

The method devised by Plummer may be tried. See chapter on Stenosis.

Prognosis: This is always doubtful.

Treatment: Surgical.

Rectal feeding may be resorted to.

CANCER OF THE ESOPHAGUS.

Etiology: Occurs more often in elderly men, and nearly always in those with cancer elsewhere.

Morbid Anatomy and Pathology: The lower part of the esophagus is affected. The cancer may be of any variety.

Symptoms: The symptoms are those of stenosis; namely, dysphagia, regurgitation of food, emaciation.

The vomitus is mixed with blood. The lymph nodes are enlarged.

Complications and Sequelæ: Broncho pneumonia may occur.

Diagnosis: The x-ray may show the tumor. Passing of a bougie may bring up adherent particles that can be examined under the miscroscope.

Prognosis: This is bad.

Treatment: The same as for stenosis.

SYPHILIS, MALFORMATIONS, RUPTURE OF THE ESOPHAGUS.

There may be *syphilitic gumma* which causes symptoms of stenosis.

There may be *malformations.*

A stenosis or dilatation may cause *rupture,* with sudden nausea, vomiting, and collapse.

ACUTE GASTRITIS.

Synonyms: Acute Gastric Catarrh. Acute Dyspepsia.

Definition: Acute inflammation of the mucous membrane of the stomach with disturbance of digestion.

Etiology: Acute gastritis is very common, and occurs at all ages. It may be due to improper food or to overeating; to the ingestion of decomposing food or of alcohol, or of too hot or too cold food or drink. It may occur with the acute infections. I have seen it come from swallowing too much salt water while bathing. Emotional disturbance may cause it.

Morbid Anatomy and Pathology: There is an acute inflammation of the lining of the stomach. Sometimes there are minute hemorrhages. I have seen them after a debauch. Micro-organisms may be present.

The pyloric end of the stomach is most often affected.

Symptoms: There is loss of appetite; this may be gradual or abrupt. The tongue is coated and food don't taste good. Certain things may still be tasted a long time after eating. The breath is foul.

There is often nausea with or without vomiting.

The vomitus is usually of undigested food at first, and may come on immediately after eating, or may be delayed for several hours after. Blood may be mixed with it, especially if the patient has been drinking. There is pain and tenderness in the epigastrium.

In milder cases there are sour or bitter eructations.

There may be constipation or more often diarrhea.

The above symptoms may be ushered in with a chill or chills.

Often there is a certain amount of fever, thirst and headache. The patient's abdomen is bloated and he belches some. He is restless and does not sleep well.

A mild acute case may come on after overeating or drinking, or after the use of some noxious food. The patient has cramps in the stomach, there is vomiting and purging, after which the patient feels better. The nausea and pain come on in waves; emptying the stomach and bowels gives relief for a few minutes or an hour or two, when the cycle is repeated. This may happen several times, gradually growing less severe till after a few days the patient feels all right again.

Examination of the stomach contents shows diminished hydrochloric acid.

The urine is apt to be scanty and dark.

Complications and Sequelæ: Acute gastritis may run into chronic gastritis.

Sometimes jaundice occurs.

Diagnosis: Care must be taken in making a diagnosis, because many cases of acute infectious disease begin with nausea and vomiting.

Appendicitis usually begins that way. Careful palpation will show a rigidity of the right rectus muscle. In gastritis the tenderness is in the epigastrium.

Typhoid fever may rarely begin with nausea and vomiting. The other symptoms of typhoid will unfold as the case progresses.

Prognosis: Except in severe poisoning cases of acute gastritis usually get well.

Treatment: Rest is of great benefit. Stop all food for twenty-four hours. In severe cases the stomach may be washed out. If a stomach tube is not at hand, give an emetic.

Applications of heat to the epigastrium frequently give relief. Sipping hot water may help.

In the milder cases bicarbonate of soda may be given, 5 to 10 grains at a dose, in water. This is especially useful if there is much gas.

Dilute hydrochloric acid, 10 to 15 drops, may be given after meals in more protracted mild cases.

The indicated homœopathic remedy is always of great assistance.

Aconite has helped in cases that started with a chill, were feverish, nervous, had nausea, and where the stomach was tender to touch.

Arsenic, no appetite, stomach sensitive; nausea with or without vomit, everything liquid or solid causes it. *Arsenic* in trituration, dry on the tongue, will sometimes give prompt relief when *arsenic* given in water is immediately vomited.

Ipecac is especially useful where there is vomiting and diarrhea both.

Pulsatilla follows *ipecac* well after vomiting has subsided but diarrhea remains. *Pulsatilla* is also useful where there is not much appetite, the sense of taste is lessened or lost; or food is tasted a long time after ingestion. Also in cases caused by emotion.

Carbo vegetabilis and *nux vomica* combined are useful in cases with frequent eructations.

Nux vomica is useful in thin, irritable patients; after drinking; pain in the stomach some time after eating. Constipation is an additional symptom.

Sanguinaria cured a case caused by eating strawberries.

Hydrastis will relieve gastritis in patients who have frequent attacks of catarrh of the stomach. It is a very useful remedy.

CHRONIC GASTRITIS.

Synonyms: Chronic Gastric Catarrh. Chronic Dypspesia.

Definition: Chronic inflammation of the mucous membrane of the stomach, with changes in the gastric juice and disturbance of digestion.

Etiology: Chronic gastritis may be caused by recurrent attacks of acute gastritis; by poor teeth resulting in swallowing of improperly masticated food; by excessive use of coffee, tea, alcohol, tobacco or of cold drinks; by condiments; by overloading the stomach; by any of the things that cause acute gastritis. Chronic gastritis may be secondary to heart or liver conditions,

to tuberculosis, Bright's disease, gout, etc., to cancer or ulcer of the stomach.

Morbid Anatomy and Pathology: The mucous membrane is grayish or slate colored, thickened and covered with thick tenacious mucus.

The veins are enlarged and there may be patches of ecchymosis.

The microscope shows the glands to be dilated and elongated with degeneration of the epithelium and infiltration of the cellular tissue; or there may be thinning of the stomach walls and destruction of glandular elements.

Symptoms: The appetite is capricious. The patient likes highly seasoned food. The tongue is coated, the breath is foul. The patient wakes up with a bad taste in his mouth in the morning. He belches much gas, and has eructations that may be sour or bitter. There may be heartburn. Nausea and sometimes vomiting occurs. If in the morning the mucus is thick.

There is pain in the stomach, often a slight tenderness on pressure. The patient cannot stand tight clothing. The abdomen may be bloated from gas. There is a heavy feeling like a lump in the stomach, especially after eating; in other cases when the stomach is empty. The pain may not come on until an hour or two after eating.

There may be headache or vertigo. The patient is irritable, he may be melancholic.

Palpitation is sometimes caused by the pressure of gas. The patient is often constipated. Loss of weight occurs in long-standing cases.

Physical Examination: Shows on *inspection* the somewhat bloated abdomen. *Palpation* shows slight tenderness. *Percussion* shows tympanites from gas.

Examination of the stomach contents shows lessened hydrochloric acid.

Complications and Sequelæ: Chronic gastritis may end in atrophic gastritis, or in achylia gastrica.

Diagnosis: The diagnosis is best made by examining a test breakfast. This should consist of:

Bread, 60 grams.

Water, 300 c.c.

An hour after eating the test breakfast is withdrawn by the stomach tube.

Examination will show the total acids diminished, and hydrochloric acid diminished. According to Lockwood lactic acid is always absent. The bread will be in coarse pieces mixed with mucus.

Ulcer of the stomach has pain after eating. Vomiting of blood occurs.

Cancer usually gives a history of long-continued discomfort with sometimes a history of ulcer a long time before.

Nervous dyspepsia has erratic and irregular symptoms.

Prognosis: Chronic gastritis runs a long course. If secondary to some other disease the prognosis depends on the primary condition.

Treatment: In chronic gastritis the integrity of the gastric circulation is affected and a change takes place in the secretions of the stomach. The most important factor in this disease seems to be the lack of hydrochloric acid in the gastric juice, thereby allowing the food to undergo abnormal fermentation; this sets up an irritation of the mucous membrane, and thus we have a disease which perpetuates itself. The mucous secretion is increased, and as this is alkaline in its reaction, it tends to further impair digestion by neutralization of the normally acid contents of the stomach.

The contractions are also lessened by impairment of the muscular coat. In prescribing a diet for gastritis, either acute or chronic, we must first find out what does and what does not agree with our patient. All articles which irritate the mucous membrane must be prohibited.

Hydrocarbons and carbohydrates, on account of their tendency to excessive fermentation, must be excluded. Alcohol should be vigorously excluded because, even in very small quantities, it tends to retard digestion. Tobacco should be tabooed. A strictly milk diet, or milk with lime water or vichy if necessary, should first be given for several days or weeks. Four to six ounces at a feeding every two or three hours. Koumyss, bacillac or buttermilk may be used. After a few days add boiled eggs, broths

and some of the numerous artificial preparations of pepsin. Bovinine and malted milk are both preparations which I have found to be serviceable.

It is best to feed patients with chronic gastritis small meals every two or three hours.

Sipping a cup of hot water, as hot as can be borne, an hour before meals is an efficient aid to treatment.

It goes without saying that the cause should be removed if possible. The habits should be regulated.

Sometimes bicarbonate of soda, five grains, given one hour before meals in hot water will help.

Remedies useful in acute gastritis may be useful in the chronic form.

Lycopodium, the food tastes sour, a little makes the patient feel full. There is gas, the patient is uncomfortable and has no ambition. The urine deposits a red sediment.

Plumbum, the patient is very constipated with his chronic indigestion.

Phosphoric acid, the tongue is furred, the patient has a bad taste in the mouth. There is pain in the stomach a half hour to an hour after eating. He is homesick.

TOXIC GASTRITIS.

Definition: Toxic gastritis is destruction of the lining of the stomach caused by the irritant action of violent poisons.

Etiology: The mineral acids, strong alkalies and other irritant poisons like phosphorus, bichloride of mercury, etc., cause toxic gastritis. They are never taken except by accident or with suicidal intent.

Morbid Anatomy and Pathology: There is violent inflammation causing destruction of the lining of the stomach followed by sloughing.

Symptoms: Symptoms progress rapidly. The mouth, pharynx and stomach are all affected. There is intense pain throughout. Swallowing becomes difficult or impossible. There is tenderness over the stomach. The patient vomits blood frequently.

The face is pale, pulse weak, and the patient in collapse.

Complications and Sequelæ: These vary with the poison. For instance, phosphorus causes jaundice.

Diagnosis: The general appearance of the patient will indicate grave trouble. The lips and pharynx must be inspected for evidences of poison. The breath may give a clue, as in carbolic acid poisoning.

Prognosis: Is always doubtful. Many cases are fatal.

Treatment: Fill the stomach with water or other bland fluid, if possible, and then empty it.

Pure alcohol is an antidote to carbolic acid.

In other cases water with two drams of mustard to the pint is useful.

The whites of raw eggs, milk or olive oil, may be of service, and plenty of them.

In phosphorus poisoning oil must not be used.

PHLEGMONOUS GASTRITIS.

Synonyms: Suppurative Inflammation of the Stomach. Abscess of the Stomach.

Etiology: This condition is probably due to micro-organisms.

Morbid Anatomy and Pathology: The stomach will be found to contain one or more abscesses.

Symptoms: Usually there is a history of indigestion. When abscess develops it runs a rapid course. There is severe epigastric pain. The patient develops a septic condition, with chills, irregular temperature, rapid pulse. There is vomiting.

Diagnosis: This can be positively made only by a laparotomy.

Prognosis: The prognosis is bad.

Treatment: This is entirely surgical.

ACHYLIA GASTRICA.

Synonym: Atrophy of the Stomach.

Definition: Functional perversion of stomach action, characterized by absence of gastric secretion.

Historical Note: Achylia was first noted in pernicious anemia

by Fenwick in 1877. Others have noted it since. It occurs late, and is probably secondary to other stomach conditions.

Etiology: Achylia is due to chronic disease of the stomach. It may occur with diabetes or pernicious anemia. A temporary functional achylia occurs in neurasthenia, in gastroptosis, etc.

Symptoms: The symptoms are somewhat indefinite and may be absent. There may be fulness with pain, belching and vomiting. The intestinal symptoms are more marked than the gastric. There are attacks of intestinal gas and diarrhea.

Complications and Sequelæ: Diseases of the gall-bladder are often found with achylia.

Diagnosis: This is made largely from examination of the stomach contents. The total acidity is low. Hydrochloric acid is absent.

Prognosis: The prognosis is tedious.

Treatment: The diet must be nutritious and easily taken care of by the saliva and intestinal secretions. Carbohydrates are permissible, fats should be curtailed some, and the proteids given sparingly.

Dilute hydrochloric acid may be given ten to fifteen drops, well diluted in water, after meals.

Of remedies, *nux vomica* probably is the most useful.

The homœopathist will find indications for various other remedies as well.

ACHLORHYDRIA HEMORRHAGICA GASTRICA.

Definition: Absence of hydrochloric acid, with occult gastric blood and micro-organisms. Is secondary to some other disease, as a rule.

Historical Note: Has been described by Pilcher, of the Mayo hospital.

Etiology: The condition is said to be secondary to some other disease as appendicitis, gall bladder disease, pancreatic disease, and so on.

Bacteriology: Streptococci, the colon bacillus, and other bacteria are present in large numbers.

Morbid Anatomy and Pathology: There seem to be erosions in various parts of the mucosa.

Symptoms: The symptoms are indefinite. There may be pain usually after, sometimes before meals. Alkalies relieve the pain. There is frequently nausea, belching and vomiting. Sometimes blood is mixed with the vomitus. Constipation and diarrhea alternate. There is loss of weight. The patient tires easily. He often has periods of depression.

Complications and Sequelæ: Ulcer or cancer may develop.

Diagnosis: The stomach contents, after a test meal, show the bread broken up with mucus, of a yellowish tinge, sometimes with a slight odor. Hydrochloric acid is absent. Occult blood is always present.

Ulcer has a different history, though Kemp says achlorhydria hemorrhagica gastrica may run into ulcer.

Cancer occurs in older subjects, as a rule, and finally develops cachexia.

Prognosis: As this disease is usually secondary to some other condition, relief by operation or otherwise of the primary disease results in cure in more than half the cases.

Treatment: Search out the cause and relieve that if possible. Other than that the treatment is the same as for chronic gastritis.

HYPERCHLORHYDRIA.

Synonym: Hyperacidity.

Definition: An increase in the secretion of hydrochloric acid.

Etiology: No age and neither sex is exempt. It occurs in neurotic patients, from worry or emotional disturbance. It occurs in chlorotic subjects, in gastroptosis, with gastric ulcer. Those who use tobacco, strong coffee, etc., to excess may have it.

Symptoms: Pain comes on in one to three hours after eating. There is an uncomfortable fulness with slight tenderness. There are eructations with heartburn and pain. Acids relieve the pain.

Albuminous foods relieve it, starchy food makes it worse. The appetite is good and the patient is often hungry. There is also thirst. Constipation is the rule.

The patient suffers from headache. Sometimes he vomits.

These symptoms may occur after ingestion only of certain particular things. The patient is worse at one time than at another.

Complications and Sequelæ: Atonic dilatation may result from spasmodic closure of the pylorus.

Diagnosis: The test breakfast, after one hour, shows increased hydrochloric acid.

Ulcer has immediate pain and vomiting of blood after eating.

Gastralgia occurs with no relation to the intake of food.

Prognosis: Prognosis is good.

Treatment: Give the patient extra feedings. The diet should consist mostly of albuminoid foods, with a moderate amount of vegetables; carbohydrates should be cut out.

Tobacco, alcohol and strong condiments should be forbidden.

The patient should exercise.

Bicarbonate of soda, 5 grains, or milk of magnesia, a dram or two in water, will sometimes help.

The remedies under gastritis may be looked up.

GASTROSUCCORRHEA.

Synonyms: Hypersecretion. Excessive Flow of Gastric Juice. Reichmann's Disease.

Definition: The excessive formation of gastric juice, even when the stomach is empty.

Historical Note: Reichmann first called attention to this condition.

Etiology: The factors that cause hyperchlorhydria may produce this condition. Frequently it is a neurotic condition.

Symptoms: The symptoms usually begin at night. The patient awakes with pain, nausea and vomiting. He is faint, almost in collapse. The vomitus is profuse, watery and acid. Vomiting gives temporary relief. After a little time the symptoms repeat.

In the more chronic form where the symptoms return each night, sometimes in the daytime when the stomach is empty, the taking of albuminous food, as eggs or milk, will give relief.

Diagnosis: The gastric crises of locomotor ataxia may be distinguished by finding the other signs of that disease.

Hypersecretion may be a symptom of organic disease of the stomach. This must be excluded.

Examination of the stomach contents when fasting show a large amount of pure gastric juice.

Prognosis: The prognosis is favorable.

Treatment: The treatment is the same as that for hyperchlorhydria.

Lavage may be of service.

Belladonna may be given as a remedy.

ULCER OF THE STOMACH AND DUODENUM.

Synonyms: Gastric Ulcer. Peptic Ulcer. Duodenal Ulcer.

Definition: An ulcer of the lining membrane of the stomach or duodenum.

Etiology: Ulcer occurs most frequently in early adult life. Mechanical injury through the abdominal wall as in tight lacing, wrong articles of food or drink may start the trouble. Single ulcers tend to heal spontaneously. But if the patient is run down, and the digestive powers weak, the tendency is not to heal. Anemia and chlorosis seem to be predisposing factors.

Morbid Anatomy and Pathology: The ulcers are usually round, sometimes oval, they are funnel shaped. I have seen ulcers as big around as a quarter dollar. They may perforate the muscular walls of the stomach as well as the mucous membrane. In one case at autopsy the stomach was full of blood. Most often they are found on the posterior wall of the stomach at the pyloric end.

Gastric and duodenal ulcer occur in about equal proportion.

Symptoms: Ulcer of the cardiac end of the stomach may exist without symptoms. In most cases of either gastric or duodenal ulcers that produce symptoms the onset is insidious. A history of chronic indigestion should always cause the physician to think of peptic ulcer. Especially is this true if there is much belching and sour eructations with relief from eating. After a time the symptoms progress till there is vomiting after eating. There is more or less constant pain, extending to the back. There is a tender spot at the epigastrium, and Boas' sign, tenderness to the left of the ninth, tenth and eleventh vertebræ, at the back.

Vomiting occurs frequently shortly after eating. Hyperchlorhydria is present. If the vomiting contains blood, or the stools contain blood, ulcer is almost certain.

Profound anemia occurs in some cases.

The X-ray shows a break in the peristaltic wave.

Complications and Sequelæ: Recent investigations show that ulcer is frequently the precursor of cancer. Lockwood has found the first symptom of malignancy to be the appearance of lactic acid in the stomach contents.

Perforation of the ulcer may occur.

Anemia may become a very serious complication. It was so in one of my cases.

Diagnosis: Ulcer is probable if there is vomiting with blood some time after eating, one to five hours. If there is epigastric pain relieved by (a) vomiting, (b) the taking of food or drink, (c) the ingestion of alkaline liquids, (d) lavage. A test meal shows excess of hydrochloric acid and absence of lactic acid.

Einhorn has devised a method of diagnosis. He has the patient swallow a small bucket attached to a thread at night. At 8 in the morning this is pulled out. The moist surface of the ulcer makes a stain on the thread at a definite point.

Bismuth meals may be given and the X-ray used.

Cancer has the cancerous cachexia. The tumor may be palpable. Hydrochloric acid is diminished, lactic acid is present.

Appendicitis does not cause vomiting of blood. There is rigidity of the rectus muscle.

Perforation may occur. Dr. G. M. Cushing, of Chicago, in the *Journal of the American Institute of Homœopathy* says: "If there is intense pain, with the most unyielding stiffness of the abdominal muscles, a perforation has probably occurred. The patient cannot make even a pretense to breathe deeply, and his replies to one's questions are jerked out with an effort, the end of which is cut short with a spasm of pain." There is an anxious, apprehensive look.

Prognosis: Fairly good, the majority of cases get well under proper medical treatment. Cases with repeated hemorrhage or with perforation should be operated.

Treatment: When diagnosis of ulcer of the stomach is made the patient should be put to bed. Food should be withheld for a time; this may be done for several days. Thirst may be allayed with cracked ice.

After two or three days to a week without food my habit is to begin with a small quantity of milk and water, half and half. This is to be repeated every three hours. After a day or two, without discomfort following the milk and water, the quantity is increased. Then the milk is given with less water until undiluted milk is given. After a few days soft boiled eggs may be added. Broths, gruels, somatose, shavings of raw beef, milk toast, may be added from time to time until, at the end of a month or six weeks, the patient is back on a full diet. Hemorrhage or severe pain will necessitate abstinence from food or the use of dilute milk for a day or two, and then gradually increasing the diet as before. I have seen many cases apparently recover on this regime.

In very severe cases with frequently repeated vomitings, rectal feedings may be necessary.

Arsenicum heads the list of remedies for ulcer of the stomach. It has all the gastric symptoms in its pathogenesis.

All other remedies are of lesser importance, although some of them may be indicated at times.

CANCER OF THE STOMACH.

Synonym: Carcinoma Ventriculi.

Etiology: Cancer of the stomach is slightly more common in men. It occurs mostly from forty to sixty years of age.

Mayo found 75 per cent. of cancer cases to follow ulcer of the stomach.

Traumatism possibly is a causative factor.

Cancer of the stomach may rarely be secondary, it is usually primary.

Morbid Anatomy and Pathology: Any form of cancer may be found. In order of frequency they are: 1. Adenocarcinoma or epithelioma; 2. medullary carcinoma; 3. less often scirrhus, and 4. least often colloid carcinoma; or there may be a combina-

tion of one or more forms. In a majority of the cases the pylorus is affected, although no part is exempt.

Ulceration of the growth may occur. Perforation of the stomach wall may take place. Secondary growths frequently occur elsewhere in the body.

If the pyloric end of the stomach is much contracted the stomach may be dilated. If the cardia is affected the stomach is contracted and the esophagus enlarged. The organ may be displaced by the tumor.

Symptoms: There may be no symptoms at the beginning. Usually there are indefinite symptoms of indigestion. According to Wegele, persons previously in good health who suddenly suffer from anorexia that does not yield to treatment should cause a suspicion of carcinoma of the stomach.

The symptoms of indigestion increase. The patient feels full and the discomfort gradually increases until there is more or less pain that is continuous. Then comes occasional vomiting, once or twice a day of food, sometimes hours after eating.

There is diminution of hydrochloric acid, increase of lactic acid, and the presence of the Boas-Leffler bacillus.

Hematemesis occurs late, as a rule, giving a coffee ground appearance to the vomitus.

Constipation is usual.

An important sign is blood in the stools when it occurs.

Emaciation, anemia and weakness begin early.

The blood shows reduction of hemoglobin out of proportion to the decrease of red cells. Poikilocytosis is marked.

Finally a tumor may be palpated.

The X-ray may be of service in diagnosis.

Cancer of Cardia: Dysphagia is common, and there is pain behind the ensiform cartilage. The cervical lymph glands are enlarged.

Cancer of Pylorus: Pain and frequent vomiting. Motor insufficiency.

Complications and Sequelæ: Metastases to neighboring organs is common. Ascites and edema sometimes occur.

Diagnosis: The onset of digestive disturbance with anemia

and emaciation, and with more or less epigastric pain that is continuous in a previously well person of forty to sixty, usually indicates cancer of the stomach. Vomiting without reference to the time of eating is another symptom. Hydrochloric acid is diminished or absent. Lactic acid is present.

Ulcer occurs earlier in life. There is pain after eating relieved by vomiting. There may be profound anemia without emaciation. Hydrochloric acid is normal or increased. Lactic acid is absent.

Gastralgia has paroxysmal pain relieved by pressure. Vomiting bears no relation to the time of eating.

Chronic gastritis has a long history, but there is no deep-seated pain.

Prognosis: A few cases have apparently been cured by early operation. Otherwise it is fatal after a year or two.

Treatment: The patient should be put on a liquid or soft diet with frequent feedings.

Remedies may be tried according to the indications.

The best treatment for cancer of the stomach is surgical.

HEMATEMESIS.

Synonym: Gastrorrhagia.

Definition: Hematemesis is hemorrhage from the stomach. It is a symptom, not a disease.

Etiology: The most frequent cause is ulcer of the stomach, the next most frequent cause, cancer. Acute gastritis may cause hemorrhage; *e. g.,* I have met with it after an alcoholic debauch, I have seen it in an inordinate tea drinker. In both cases the hemorrhage was undoubtedly due to an excess of the offending beverage; as the offence was never repeated the hemorrhage never recurred. Hematemesis may be secondary to the venous engorgement of heart or liver disease. It may occur in scurvy, purpura, aneurism, etc. Rarely it is due to traumatism.

Symptoms: There is vomiting of a smaller or larger quantity of blood. The vomited blood is usually dark and may be fluid or clotted. Blood may also be found in the stools. If the hemorrhage is severe, or is frequently repeated, anemia occurs. There is weakness, perhaps syncope or even convulsions.

Diagnosis: A patient who vomits blood must be carefully examined to eliminate any possibility that the blood was swallowed first.

Hemorrhage from the stomach is usually easily differentiated from hemorrhage from the lungs because the latter comes with a cough and is bright red.

Prognosis: The prognosis depends altogether on what caused the hemorrhage. Ulcer cases usually get well.

Treatment: For the hemorrhage itself rest is the first indication. The patient should be put to bed.

Food should be withheld for a day or two, with cracked ice held in the mouth, to allay thirst as needed.

The underlying cause must be found and treated.

CYCLIC VOMITING.

Definition: Periodic attacks of vomiting occurring in young children and without known cause.

Historical Note: This is an obscure condition that has been described only during the past ten or fifteen years.

Etiology: It occurs most frequently in children of families of a neurotic or gouty tendency.

Morbid Anatomy and Pathology: The only recorded autopsy that I know of was made by Russell, of England. He thought the vomiting due to recurrent spasm of the pylorus.

Symptoms: The little patient has some languor, and loss of appetite. The vomiting is violent and prolonged, first of food, then of bile, possibly some blood. The patient is prostrated and has slight fever. The throat may be red and sore from the violent acid vomiting.

A given attack may last for a day or two, the stomach rejecting all food. Then the symptoms disappear to return after a few weeks or months. The patient may have several attacks.

I have the record of one case, daughter of an old school physician, who had had a number of severe attacks extending over two or three years. She was always desperately ill. Some of the best known pediatrists in New York, after using morphine. apomorphia, bicarbonate of soda, lavage, rectal injections,

castor oil, and what not, at various times, had at last given up. The attacks always lasted several days. In desperation, and with apologies for calling a homœopath I was sent for. *Ipecac* was indicated and given, the third, 5 drops in half a glass of water, two teaspoonfuls every hour. The child vomited but twice in the next twenty-four hours and that was the end. She never had but one other attack, that came some six months later, and was controlled at once with *ipecac*. That was in 1902. She has never had another.

Diagnosis: A diagnosis of cyclic vomiting can be made only after all known possibilities have been eliminated.

Prognosis: This is good. Recovery is rapid.

Treatment: Stop all food. Put the patient to bed.

Ipecac is the remedy of choice.

Arsenic may be indicated if vomiting follows every mouthful.

Pulsatilla is useful after the vomiting stops.

ATONY AND DILATATION OF STOMACH.

Synonym: Gastrectasis.

Definition: Relaxation and weakening of the muscular walls of the stomach causing motor insufficiency and dilatation. It may be acute or chronic.

Etiology: *Acute.* Dilatation may follow operations under general anesthesia. It may follow infectious diseases. It may follow excessive eating or drinking. It may follow injuries to the head and spine.

Chronic. Chronic dilatation may be due to pyloric obstruction, to pressure from tumors. It may occur in the middle-aged or elderly.

Morbid Anatomy and Pathology: The stomach is enlarged. Its walls are thin and atrophic.

Symptoms: There is a fulness and discomfort after meals. There is some belching and vomiting long after eating. The food stays in the stomach a long time; the result is malnutrition. The patient gets worse and worse until the stomach cannot expel the food. The vomited matter becomes large in amount and very offensive. It separates into layers, at the bottom undigested food, above it liquid, topped by a layer of froth.

The patient is constipated. The quantity of urine diminished.

Physical Signs: The abdomen is prominent. Peristaltic waves can be felt and sometimes seen. A splashing sensation may be gotten by palpating with one hand and tapping with the other. Percussion reveals tympanites. If the patient drinks a glass of water there will be an area of dulness between the tympanitic note of the stomach and of the intestines when standing; this disappears when the patient lies down.

A bismuth meal will outline the stomach for the X-ray.

Complications and Sequelæ: Gastroptosis may be a sequel.

Diagnosis: The diagnosis is made from the physical signs, largely.

Prognosis: This depends on the cause.

Treatment: The stomach should be washed out at intervals.

The patient should be fed frequently small meals of concentrated foods. Meat and eggs are best. Fats and starches should be avoided. Little liquid should be given.

Surgery may be able to do something.

GASTROPTOSIS AND ENTEROPTOSIS.

Synonym: Glénard's Disease.

Definition: Prolapse of the stomach and colon, congenital or acquired. It may be of different degrees.

Historical Note: Kussmaul and Glénard first called attention to the condition.

Etiology: The condition is more common in women. Tight lacing and frequent pregnancies seem to be predisposing causes. Other organs may also drop, notably the right kidney.

Morbid Anatomy and Pathology: There is a general dislocation of the abdominal organs.

Symptoms: In some cases there may be no symptoms. Usually there is more or less disturbance of digestion. There is gas with fulness and distress. The patient is constipated. There is an irritable bladder.

Palpitation occurs. There is headache, sometimes hysteria.

Physical Signs: The abdominal walls are thin and flaccid. The epigastrium sinks in and the abdomen protrudes. There is marked pulsation of the abdominal aorta.

Complications and Sequelæ: There may be ptosis of organs other than the stomach and intestines.

Diagnosis: This is easy.

Prognosis: Doubtful.

Treatment: Patients should correct bad habits of tight lacing. A special band or belt made for these cases should be worn. Rest as much as possible.

The diet should be concentrated and nutritious. The amount of liquids should be limited.

Surgery may be of some assistance.

NERVOUS DYSPEPSIA.

Synonym: Neurasthenia Gastrica.

Definition: Indigestion due to nerve disturbance. The symptoms are out of proportion to any known pathological condition.

Etiology: The same causes that produce neurasthenia may produce nervous dyspepsia. Mental and physical overwork, shock, worry, emotional disturbance, sexual excesses.

Symptoms: The patient is usually of a nervous temperament. He worries about his condition and is afraid to eat. The tongue is clear, the appetite variable. The patient is irritable, he has vertigo and headache.

Diagnosis: Care must be used in taking the case that the symptoms are of nervous origin, and not of other pathological conditions.

Prognosis: This is good if the cause is removed.

Treatment: The patient should keep quiet, and be put under the best hygienic condition. The utmost regularity should be exercised as to the eating of meals.

GASTRALGIA.

Synonym: Neuralgia of the stomach.

Definition: Intense paroxysmal pain in the epigastrium, pain that comes and goes irrespective of meals.

Etiology: Worry, excessive use of tobacco. Gastralgia may be a reflex disturbance from some other part of the body.

Symptoms: There is sudden intense pain in the epigastrium. There may be collapse.

Diagnosis: Care should be used to eliminate other causes.

Prognosis: Good.

Treatment: Heat usually gives relief.

Belladonna might be used in cases of fever.

CONSTIPATION.

Synonym: Costiveness.

Definition: Infrequent or difficult evacuation of the bowels.

Etiology: Diseases of the stomach may cause constipation. Lack of intestinal secretion or motor insufficiency of the intestines may cause it. It may be only a symptom of some other disease. It may be due to overindulgence in cathartics. Constipation may be due to improper food. More often it is due to lack of attention to the desire to stool; the patient postpones the function until some more convenient time, and finally the bowel loses the habit of regularity. Finally, constipation may be due to obstruction by tumors or by a displaced uterus.

Symptoms: There is considerable difference among well persons as to the bowel movements. The average adult has one stool a day. Some have two a day, others one every other day or less often. One of the healthiest men of my acquaintance has one movement a week. That is his habit and has been for thirty years. In all that time he has never been under a physician's care for any purpose whatsoever.

When constipation exists and produces symptoms, there is a fulness and discomfort in the region of the rectum, there is loss of appetite, coated tongue, belching, headache, and so on. Sometimes the patient worries about the condition, and may even become hypochondriacal.

In other cases the patient may show symptoms of severe autointoxication with fever, irrational ideas and so on.

The taking of cathartics may cause severe intestinal colic, even diarrhea, without dislodging the accumulated fecal mass.

In severe cases the fecal matter may form a tumor-like mass that can be felt through the abdominal wall. It can readily be felt through the rectum.

In infants constipation may be found, due usually to faulty food.

Complications and Sequelæ: Constipation may cause hemorrhoids, ulcers or fissures in and about the anus.

Diagnosis: The diagnosis of constipation must consider not only the frequency of movements, but the quality and the effort required to expel them.

Prognosis: The prognosis is good, but chronic cases are hard to handle and usually very tedious.

Treatment: The first and most important thing to do in a case of constipation is to regulate the habits. The patient should be told to have a regular time for the daily evacuation.

The diet should include plenty of coarse food, graham or rye bread, oat meal, succulent vegetables, fruit. A glass of cold water, taken each morning, before breakfast, will often help.

In infants, of course, the milk is changed in some way. A feeding of malted milk may help in mild cases.

Massage or vibration may be of help. The patient should take regular exercise.

In cases with deficient secretion a spoonful or two of olive oil, by mouth, may act as a lubricant.

To relieve the bowel itself injections may be used. The ordinary soap suds enema may be sufficient. In other cases rectal injection of an ounce or two of oil may be necessary to soften the mass. In bad cases of impaction the rectum may actually have to be dug out with the finger or with a spoon.

Cathartics should be interdicted if possible; at any rate only an occasional dose at rare intervals should be permitted.

The homœopathic remedy will work wonders if the patient can be controlled. I have seen cases of many years' standing cured. The indicated remedy may have to be given at infrequent intervals for weeks or months.

Bryonia is one of the most frequently indicated remedies. The stools are hard and dry. The patient has headache often. This has cured many cases for me.

Graphites is indicated where the stools are large and difficult to expel. The skin is inclined to be oily or else very dry.

Patients of a scrofulous habit. Given twice a day for several months it cured one case of many years' standing.

Opium in potency may help where the bowel is inactive, there is no desire, and voluntary effort produces nothing.

Plumbum is indicated if the first part of the movement is like bullets. There is gas in the stomach.

Nux vomica is indicated in cases due to gastric disturbance.

Many other remedies may be indicated, but the above have served me best in the order given.

INTESTINAL STRICTURE AND OBSTRUCTION.

Synonym: Ileus.

Etiology: The obstruction may be due to ulcer, cancer, impacted feces, adhesions from trauma or from operation, compression by tumors. It may be due to volvulus or intussusception.

In rare cases of newborn infants it is congenital.

Morbid Anatomy and Pathology: The part above the obstruction becomes swollen and inflamed. Sloughing may occur with perforation. The intestine below the obstruction is poorly nourished and empty.

Symptoms: There may be a very considerable constriction without bowel symptoms. I saw one case of cancer at the ileocecal region where the lumen of the intestine was just the size of an ordinary lead pencil. The bowel movements had not been affected.

In cases with symptoms there is usually constipation. If the trouble is low down in the bowel the feces may be ribbon-shaped. With these symptoms there may be pain. Finally the obstruction may be absolute. Then there is colic, tympanites and excessive peristalsis with vomiting, which rapidly becomes stercoraceous. Finally, there is collapse, with pinched facial expression, sunken eyes, cold, clammy skin and rapid, weak pulse.

The urine shows excess of indican.

In cases of obstruction from tumor and so on the symptoms come on gradually until the obstruction is complete when the symptoms of collapse develop rapidly.

Acute obstruction with rapid onset of the above symptoms may

come from: (1) Volvulus, a twist in the bowel; (2) intussusception or invagination, where one part of the bowel slips into another; (3) strangulation, where a portion of the bowel gets caught as in strangulated hernia.

Complications and Sequelæ: Peritonitis may result if relief is not gotten quickly.

Diagnosis: The diagnosis is made on the symptoms. The abdomen should be inspected and palpated for peristalsis and for tumor.

Acute peritonitis as a disease, *per se,* has pain and tenderness over the abdomen. There is fever. There is no stercoraceous vomiting.

Prognosis: The prognosis is usually bad, though even the most desperate case sometimes recovers.

Treatment: In obstruction from impaction of feces injections of warm water or of oil may serve. Ox-gall, 2 drams to a pint of water, may be used in bad cases. Removal by the finger or scoop may be necessary.

In volvulus sometimes atropine will relieve.

In acute cases prompt operation may be necessary to save the patient's life.

Cathartics are contraindicated.

INTESTINAL CATARRH.

Synonyms: Catarrhal Enteritis. Diarrhea.

Definition: Too frequent stools of liquid consistency.

Etiology: Diarrhea may be caused by improper food—too rich food—food of poor quality. The excessive use of cathartics may cause it.

It may be due to infection.

It may be due to chilling or overheating of the surface of the body.

It may be due to profound emotional disturbance.

Morbid Anatomy and Pathology: There is swelling and redness of the intestinal mucosa with an increase in secretion—a true catarrhal condition. In bad cases the hypersecretion may become purulent. In some cases there is atrophy of the intestinal wall.

Symptoms: An attack of diarrhea is usually ushered in with colicky pains of a more or less severe character due to increased peristalsis. The increased intestinal secretion causes loose stools, which may be few or many during the twenty-four hours. The first one or two stools may not vary much from normal, but the desire to stool is urgent. Then in a short time follow others that are loose and watery. Borborygmus is frequent, and there may be considerable flatus with the discharge. The color of the movement is changed to yellowish, or brown, or greenish, sometimes almost colorless. In mild cases one or two loose movements may end the trouble.

In others the condition may go on for several days or a week or two. There may be headache, aching all over, chill. There may be a slight rise in temperature, 102° F. The patient may feel quite weak and exhausted. In other patients the sensation is one of emptiness, the patient feels cold and clammy, almost in a state of collapse. Although the abdomen may feel sore, heat or pressure will often relieve the colicky pains.

If the small intestine is the seat of the trouble there is apt to be a bad taste in the mouth, nausea, sometimes vomiting. The stools become entirely liquid with slight flocculent shreds floating about.

If the large intestine is the seat of the trouble there may be lumps of fecal matter and mucus. There is usually tenesmus.

If the stools are frequent enough and the condition long continued the anus is apt to become sore and excoriated.

Membraneous Enteritis or mucous colic is a condition the cause and pathology of which are little understood. The stools contain a large amount of mucus and pieces of mucus of considerable size may be voided. The membrane may even be diphtheritic. Frequently this condition alternates with constipation.

The condition is most often found in hysterical subjects.

Diagnosis: As a rule, the diagnosis is easy. It should not be forgotten, however, that diarrhea sometimes may occur during the onset of typhoid. In that case the other symptoms are constantly getting worse, whereas in acute diarrhea there is usually abdominal distress, then urgent desire for stool, with relief after the stool.

Appendicitis may have purging in the beginning, but localized pain and tenderness on the right side appears in a few hours. There is no relief after stool.

Chronic diarrhea is most often a symptom of some severe constitutional disease.

Prognosis: The prognosis is good for acute diarrhea. Chronic diarrhea is very tedious.

Treatment: The patient with a diarrhea should keep quiet and is best off in bed. Warm cloths or a hot water bag to the abdomen will often afford comfort.

Colonic flushings are recommended in severe cases.

The diet had best be restricted to milk, milk and vichy, milk toast, clam broth, soft boiled or poached eggs, dry toast, etc.

A dram of brandy in water may be given if there is much pain.

The homœopathic materia medica is rich in remedies for diarrhea. The following list is compiled from my case records of cases apparently cured:

The first remedy I think of for diarrhea is *pulsatilla*. The patient has pain and desire for stool which is relieved when the stool is passed. There is gas, the motion varies in color and consistency. The patient may taste food eaten some time before. *Pulsatilla* is particularly useful in the simple diarrheas of hot weather. I have verified it again and again in all sorts and conditions of people. A number of patients carry a vial of *pulsatilla* with them during the summer season. Usually two or three doses at one or two hour intervals will give prompt relief. I use the third dilution.

Ipecac is the remedy when there is diarrhea with vomiting. *Ipecac* follows or precedes *pulsatilla* remarkably well.

Aconite is useful if the patient is cold and chilly and cannot get warm. He may have nausea and vomiting and be nervous. I found it frequently indicated in patients treated during my sojourn in New Mexico, where the altitude was high—five thousand feet—and the air dry.

Gelsemium is somewhat like *aconite* but has headache with the other symptoms.

Arsenicum album has vomiting after eating or drinking. The stool is frequent and may be watery or bloody. The patient is exhausted and restless. He may perspire freely.

Calcarea carbonica is especially useful in children. The movements are loose and slimy. The child has goose flesh and appears cold.

Mercurius solubilis has loose, frequent stools, with pain relieved by doubling over. The patient has an empty feeling and cannot get enough to eat.

Mercurius corrosivus has frequent mucous stools with tenesmus, which is not relieved by passing the stool. There is some pain in the stomach. The patient is weak and exhausted.

Veratrum album has frequent stools that come with a gush. There is griping pain and a cold, clammy feeling.

CHOLERA INFANTUM.

Synonyms: Cholera Morbus. Cholera Nostras.

Definition: An acute gastro-intestinal affection resembling Asiatic Cholera. It is probably due to a specific germ.

Etiology: It occurs most often in hot weather, and in young children. Poverty, bad hygiene and improper food are the most frequent causes.

Morbid Anatomy and Pathology: There is an acute inflammation of the gastro-intestinal tract.

Symptoms: The disease sets in suddenly with violent vomiting or diarrhea or both. The child rapidly becomes exhausted and collapsed. The face is sunken, there is considerable fever and great thirst. The stools are large, frequent and soon become a sour, almost colorless liquid. The urine is much diminished.

The patient is restless. Stupor and convulsions are common. Adiposum sclerma—stiffening of the extremities—may occur.

The disease is very rapid, death may ensue within twenty-four or forty-eight hours.

Complications and Sequelæ: Stupor, coma, convulsions.

Diagnosis: The diagnosis is made from the rapid development of the symptoms.

Prognosis: The prognosis is bad, especially for young children. Recovery is slow in those that recover.

Treatment: Withhold all food for a few days. Give hot baths for the collapse; cracked ice or iced champagne may be used for thirst.

Hypodermoclysis with normal salt solution, or irrigation of the bowel, may help.

Barley water or albumin water in small quantities is the first food that can be permitted.

Camphor in frequent doses may be used.

Veratrum album covers many of the symptoms, vomiting and purging and collapse.

Cuprum may be tried if the colic is severe.

APPENDICITIS.

Synonyms: Perityphlitis. Typhlitis.

Definition: Inflammation of the appendix and sometimes of the surrounding tissues.

Historical Note: Formerly inflammation in the right iliac fossa was called typhlitis or perityphlitis. It is only since about 1880 that appendicitis has been considered a disease entity.

Etiology: Appendicitis is probably the result of infection, possibly by the coli communis. As exciting causes errors of diet, worms and fecal impactions seem at various times to originate the disease. It is most often found in young adults. Osler speaks of some unknown factor that seems to make it a family disease. I have two families in each of which a son was operated for appendicitis, and a year or two later the father was operated. I have another family where within six months a married daughter had appendicitis and recovered under treatment; the father, aged 68, died of it without operation (the oldest patient I have ever treated with it); two surgeons in consultation refused to operate; a son, aged 30, died after operation; the mother recovered from an interval operation. The two young people were married and had homes of their own.

Bacteriology: The coli communis, pus organisms, occasionally the typhoid bacillus, and more rarely the tubercle bacillus have all been found in cases of appendicitis.

Morbid Anatomy and Pathology: Various investigators have

reported cases of catarrhal appendicitis, cases with inflammation going on to abscess, necrosis, even to obliteration. Dr. F. A. Palmer, in 1913, stated that he believed appendicitis always began in the lymphoid structure of the appendix and was always inflammatory, never catarrhal.

Symptoms: An acute attack of appendicitis usually begins suddenly with pain, like intestinal colic, and frequently with vomiting. It often follows some real or fancied indiscretion in diet, and the patient thinks he has an ordinary stomachache. In one patient the trouble started after jumping from a street car.

After a few hours the pain is worse in the lower abdomen. At this time it is possible by careful palpation to detect a slight rigidity of the right rectus muscle. There is some tenderness of the abdomen which is localized, usually at McBurney's point; this is on a line between the navel and the right anterior superior spinous process of the ilium, and nearer the ilium. In some patients a tumor may be felt at this point.

Frequently there is a peculiar facial expression, the patient looks sicker than the symptoms seem to warrant. The features have a drawn pinched look.

In one patient the vomiting began suddenly at 10 p. m. When I saw him at 5:30 a. m. the pain was in the region of the bladder, the rigidity of the right rectus could be felt by 9. The temperature was 98° F., pulse 64. At 6 p. m. the temperature was 100 2/5° F., pulse 80. At 10 p. m. a gangrenous appendix was removed.

In another patient vomiting began about 2 a. m. At 4 p. m. on a second visit the pain was in the region of the bladder, and the patient had to be catheterized. Vomiting kept up at intervals all day, likewise diarrhea. At 10 p. m. the temperature was 104° F. At noon on the second day the temperature was 100° F., pulse 88. The symptoms were so indefinite that the surgeon, Dr. G. W. Roberts, thought operation unnecessary. At 9 p. m. the pulse had gone up a little. Operation was decided on, and pus was found.

These two cases were typical. Sudden onset with vomiting; in a few hours pain over the lower abdomen; rise in temperature; increase in pulse rate; rigidity of right rectus.

In doubtful cases the blood should be examined. There will be found in appendicitis a leucocytosis. If that increases operation is imperative.

The pulse is a more valuable sign than the temperature so far as indication for operation is concerned. If the pulse rate increases operate.

Sudden cessation of pain is bad. It usually means rupture and operation then may be of no avail.

Recurrent Appendicitis: Some patients will have recurrent attacks of vomiting, tenderness over the abdomen, perhaps diarrhea, followed by fever. The symptoms may not be definite enough to think them due to appendicitis. One patient, a boy, had such attacks at irregular intervals during several years. I put them down to indiscretions in diet, worms, and what not. Finally an attack came that was unmistakably appendicitis. It subsided in a day or two. Nine months later he had another acute attack and was operated. The boy was then twelve years old. My records showed thirteen such attacks during six years. Although I did not recognize eleven of them as appendicitis, I now think they were, because since then, six years, he has had no trouble of that kind.

Chronic Appendicitis: Occasionally a patient will have one or two mild attacks of appendicitis that subside under treatment. But he will continue to have a certain amount of discomfort in the region of the appendix. Walking upstairs will aggravate this. He will be nervous and tire easily.

Complications and Sequelæ: Acute cases may cause perforation or rupture of the appendix, with resulting peritonitis.

Diagnosis: A classical case of appendicitis is easy of diagnosis. Others are not. For example the case of the boy noted above whose repeated attacks I supposed due to overeating or other indiscretion. One symptom this boy had each time was that his facial expression was that of a very sick child, the face was drawn, the eyes sunken, he looked thin.

Another case that began with vomiting had a temperature of 104° F., pulse 100, respiration 30. I found rales over the right lower lobe and diagnosed pneumonia. Next day appendicitis was unmistakable. There was no pneumonia.

If the pain and the tenderness are high up in the abdomen, one may think it is a gall-stone colic. The unfolding of the symptoms will usually make it clear.

In women the diagnosis may be quite difficult because an inflamed ovary may simulate appendicitis. A vaginal examination will help to differentiate.

Prognosis: This is always doubtful without operation. If operation is done early the prognosis is usually good. Many mild cases recover under medical treatment. A few cases die after operation.

Treatment: In suspected appendicitis cathartics and opiates are tabooed.

Sometimes warm applications will relieve the pain. Cold applications I do not approve of.

I have seen cases of appendicitis recover under the medical treatment recommended by Hale; namely, *belladonna* and *mercurius solubilis* in alternation. I use the third decimal potency of each in water, a dose every half hour alternately. Many times the symptoms have subsided in a few hours. If they do not get better in twenty-four hours at most I call a surgeon. Even if the pain and temperature improve, if the pulse increases in frequency, or loses volume, I call the surgeon.

TUBERCULOSIS OF THE INTESTINES.

Etiology: Tuberculosis of the intestines is usually secondary to pulmonary tuberculosis. In rare cases it may be primary.

Bacteriology: The tubercle bacillus may be found in the feces. It should not be forgotten that swallowed bacilli may possibly show in the feces also, so that other symptoms are necessary to prove an actual intestinal tuberculosis.

Morbid Anatomy and Pathology: Tuberculosis may affect the large or small intestine. It starts in the lymph glands as a single focus, or a number of them. These fuse. There is infiltration, finally there may be tubercular ulcers.

Symptoms: The principal symptom is a stubborn chronic diarrhea. Hemorrhage from the bowel sometimes occurs. There may be a certain amount of pain and tenderness. Occasionally

the infiltration may be so great as to present a tumor-like mass. Added to the local symptoms are fever and emaciation.

Fistula in ano is a frequent tubercular condition.

Complications and Sequelæ: Fatal hemorrhage may occur. The ulcer may perforate into the peritoneal cavity and set up peritonitis.

Diagnosis: The diagnosis in a primary case is not easy. In a secondary case it may be assumed if chronic diarrhea develops in a patient with pulmonary tuberculosis.

Prognosis: The prognosis is bad. An occasional case may be benefited by operation—exploratory incision.

Treatment: The general hygienic measures used for a tuberculosis patient may be tried. The diet must be suited to the diarrhea. Further treatment is unsatisfactory.

Dr. M. J. Bliem, of San Antonio, Texas, a few years ago reported some cures with *chaparo armagossa,* a Mexican plant. My own experience with it has been less favorable.

Dr. W. D. Duckworth, when an interne on my service at the Tuberculosis Infirmary, had good results from *aloes, causticum, croton tiglium,* and *rhus toxicodendron,* according to the homœopathic indications.

SYPHILIS OF THE INTESTINES.

Etiology: The same as elsewhere in the body.

Morbid Anatomy and Pathology: The rectum is the part of the intestinal tract most often attacked by syphilis in adults for obvious reasons. There is induration, nodular thickening, tumor formation. The rectum feels like an inverted funnel. Ulceration may take place resulting in the formation of cicatrices. There is distension of the bowel above the lesion.

In infants, with congenital syphilis there is enlarged abdomen, the liver is increased in size. The lymphatic glands can be felt through the skin.

Symptoms: The bowels are apt to be irregular. There is catarrh of the rectum with discharge of mucus and pus, sometimes much, sometimes little.

In children there is progressive anemia and emaciation and fever.

Diagnosis: May be difficult. Usually it is not, because the patient presents the general systemic symptoms of syphilis.

Prognosis: The prognosis is bad, especially in children.

Treatment: This must be antisyphilitic.

CANCER OF THE INTESTINES.

Any type of malignant growth may attack the intestines.

Etiology: Cancer of the intestines occurs most often late in life, or, at least, rarely before middle age.

Trauma in some form is frequently the starting point.

Morbid Anatomy and Pathology: It is rare except at the rectum. It may occur at other points, especially the ileo-cecal region. The growth usually encircles the part affected, and gradually encroaches on the lumen of the intestine until constriction and finally occlusion occurs.. The part of the intestine above the growth may become dilated.

Symptoms: The symptoms vary according to the location of the tumor. In a case of mine, a man aged 46, operated by Dr. E. G. Tuttle in 1900, a colloid cancer together with fourteen inches of gut was removed from the ileo-cecal region. The mass was as large as the two fists, and the lumen through the intestine no larger than an ordinary lead pencil. The symptoms before operation were progressive loss of weight over several months, a feeling of discomfort and sometimes pain in the abdomen, a tendency not to hold himself erect when standing, a mild secondary anemia. The only bowel symptom was blood in the stools a few times shortly before operation. The mass was palpable, although a number of other physicians had overlooked it. The patient made a complete recovery from the operation, and now, fourteen years later, is the picture of health and an exceedingly active business man. But that is incidental, the point is the indefinite symptoms in a malignant condition.

Cancer of the rectum usually presents symptoms of pain and tenesmus on defecation. The pain shoots about. The stools are mucous and bloody. Diarrhea and constipation alternate. The feces may be narrow or ribbon-shaped. Paralysis of the sphincters may occur. There is emaciation.

Digital examination reveals an irregular hard growth.

In all cases of cancer of any part of the body a bad color of the skin and general emaciation occur late in the disease.

Complications and Sequelæ: Metastasis to the liver or other parts is liable to occur. The mass may ulcerate through causing hemorrhage or perforation of the bowel.

Diagnosis: Early in the disease diagnosis may be difficult. Later tumor may be found. The feces should be examined in suspicious cases.

A person of middle or advanced age, with progressive emaciation and with indefinite abdominal symptoms, should be carefully examined for possible cancer.

Prognosis: Usually bad.

Treatment: When cancer is suspected a good surgeon should be called in.

Meantime symptomatic treatment should be tried.

Phytolacca is a good remedy for cancer. The patient mentioned above took it for a year after his operation. I like to think it was of service in preventing a recurrence.

Mercurius corrosivus controlled the diarrhea in a case of cancer of rectum.

Nux vomica, on the gastric symptoms, relieved the pain and controlled nausea and vomiting.

HEMORRHOIDS.

Synonym: Piles.

Definition: Varicose dilatation of the hemorrhoidal veins at the lower end of the rectum.

Etiology: Pregnancy, constipation, portal congestion, are all causes of hemorrhoids. Aside from women during pregnancy, middle aged men are most often affected.

Morbid Anatomy and Pathology: The hemorrhoidal masses may be internal or external according to their relation to the sphincter. Large varices push out the mucous membrane, constriction takes place, causing swelling. There may be ulceration and bleeding. The surrounding tissue is inflamed.

Symptoms: There may be no symptoms in mild cases. In most

cases they are very distressing. There is burning pain, worse at stool. The tumors may bleed then. The pain is more or less constant. The voluntary muscles about the anus tend to contract, adding to the misery. There is pain in the sacral region. Micturition is difficult. When the hemorrhoids become constricted they are exquisitely painful and defecation is agony.

Diagnosis: The diagnosis is not difficult. The tumors can be felt, sometimes seen.

Prognosis: The prognosis is good under appropriate treatment.

Treatment: Rest is the first essential. Sitz baths help to allay inflammation. Gentle exercise may be indulged in after improvement sets in. The diet should be concentrated. The patient should not be allowed to become constipated.

Hamamelis, externally and internally, is easily first choice in the majority of cases.

Nux vomica is another remedy that often helps.

Calcarea phosphorica helped one case.

ACUTE PERITONITIS.

Definition: An acute inflammation of the peritoneum.

Etiology: Peritonitis may result from cold, trauma, surgical operations; it may be caused by extension of inflammation of the gastro-intestinal organs, or be due to perforation of ulcer of the stomach or intestines. It may be the end result of Bright's disease, gout or arterio-sclerosis. In infants it may be caused by an inflamed navel. In women it may be caused by disease of the pelvic organs.

Peritonitis may be localized or general.

Bacteriology: Various pus forming organisms may be found, or the coli communis, the gonococcus, or the pneumococcus.

Morbid Anatomy and Pathology: The peritoneum and intestines are reddish in color and glued together with a serofibrinous or purulent exudate. Recent adhesions are not very firm, old ones quite so. The mass may have an offensive odor. If the peritonitis follows perforation air may be present.

Symptoms: In secondary cases the symptoms may be masked by the original condition.

In typical cases there is abdominal pain and tenderness. The latter may be extreme. The patient lies with the knees up to relieve the pain. The abdomen is distended, the intestines likewise, and often there is tympanites. This latter depends somewhat on the abdominal wall, a flabby one will be more tympanitic, a firm one hard and board-like. In advanced cases with much exudate the abdomen feels doughy to the touch. The percussion note will vary according to circumstances.

The heart and liver may be pushed upward by the distension.

Vomiting, either spontaneous or following the ingestion of food, occurs. Eructations of gas take place. Constipation due to inactivity of the bowels is the rule, though diarrhea may occur.

There are often chills at first, then high fever. The respirations are rapid and shallow to limit the pain. The pulse is fast, weak, and may be irregular. As the disease progresses the patient may go into collapse with subnormal temperature.

Urination is frequent, the urine high colored and scant and contains indican.

Circumscribed peritonitis: The symptoms are milder, as a rule, and the inflammatory condition localized.

Diagnosis: The diagnosis is usually easy. Exploratory puncture may be made with an aspirating needle and some of the fluid withdrawn for examination.

Rupture or perforation of an ulcer may send a patient into collapse, with the rapid development of peritonitis later.

In intestinal obstruction there is no fever at first. Abdominal pain comes later and increases. There is visible peristalsis and later fecal vomiting. There is no exudation.

Prognosis: This is always doubtful.

Treatment: The old school recommend opiates. They should not be used.

Hot applications are of distinct benefit.

In collapse, stimulants may be necessary.

Surgical interference is necessary in cases due to perforation or to pelvic disease. It must be prompt.

Homœopathic remedies are of service.

Belladonna in acute cases with high fever and full pulse. Slightest touch hurts.

Bryonia in cases with exudate. Lips dry and parched. Irritable.

Apis where there is fluid with tender abdomen and aggravation from warmth.

Aconite at the very beginning with the characteristic mental anxiety, high fever, hot, dry skin.

Cantharis where the abdominal symptoms are associated with a tenesmus of the bladder.

CHRONIC OR TUBERCULAR PERITONITIS.

Etiology: Chronic peritonitis is usually due to tuberculosis of the peritoneum. Acute peritonitis may run into the chronic form.

Morbid Anatomy and Pathology: The peritoneum is thick and adherent. It is frequently covered with tubercles.

Symptoms: Peritonitis of this type is most apt to occur in those with tuberculosis elsewhere. There is moderate distention of the abdomen. On palpation adhesive bands can sometimes be felt, also the lymph glands. There is very often diarrhea. Sometimes the adhesive bands will cause obstruction of the bowels.

Diagnosis: This is often difficult. The symptoms must be carefully weighed. They are often masked by the original condition.

Prognosis: This is always doubtful.

Treatment: Rest is essential. The patient must be put under the best possible hygienic conditions. The underlying cause must be eliminated if possible.

Surgical interference occasionally gives brilliant results—opening of the abdomen.

NEW GROWTHS OF THE PERITONEUM.

Cancer is the usual form of new growth.

Etiology: Cancerous nodules of the peritoneum are usually secondary to cancer of the gastro-intestinal tract, pancreas or liver.

Morbid Anatomy and Pathology: The lymph glands are swollen. Usually there are many small nodules, sometimes a few large ones. I have met with both types.

Symptoms: The disease occurs most often in middle life or later. The patient has the cancerous cachexia—the limbs are thin, the face drawn, the belly usually big. There may be ascites.

Frequently nodules appear in the skin. One of my patients must have had a hundred. She was cancerous throughout.

Diagnosis: This must be made on the symptoms and physical examination.

Prognosis: This is bad.

Treatment: If detected early enough surgical interference may be of service. If the disease is general nothing can be done except to make the patient comfortable.

ASCITES.

Synonym: Hydroperitoneum.

Definition: A collection of serous fluid in the abdominal cavity due to venous stasis. Ascites is a symptom, not a disease.

Etiology: Ascites is due to obstruction of the portal circulation by disease of the liver, abdominal tumor, etc. Or it may be the result of chronic heart disease, Bright's disease and occasionally of other conditions.

Morbid Anatomy and Pathology: This varies with the cause.

Symptoms: If the amount of fluid is slight there may be no symptoms. Even when there is a large amount it may not show during life. I made an autopsy on one case at the Ward's Island Homœopathic Hospital while an interne where ascites was not suspected. The patient, a man, was much emaciated, the abdomen was flat. Yet on opening the body several quarts of fluid were removed. The abdominal organs were shrunken, the liver and heart and lungs forced upward by the fluid. This condition was unusual.

As a rule, much fluid causes distension of the abdomen. As the quantity increases the skin becomes tense and distended. The veins about the navel stand out—the caput medusa. With

change in position of the patient the fluid becomes pendant. There is fluctuation on palpation. Tapping one side of the abdomen will send a wave to the other. Percussion is dull over the pendant portion. Lightly place the fingers on the abdomen, then suddenly press them in, and the organs can be felt underneath.

A large amount of fluid in the abdomen pushes up the diaphragm and interferes with the heart's action and with respiration by displacing the heart and lungs.

The urine is scant.

The fluid may be withdrawn by tapping. In most cases it is pure serum. In malignant disease it may be bloody. With occlusion of the thoracic duct it may be chylous.

Diagnosis: An ovarian cyst may occupy the whole abdominal cavity, but a change of position does not alter its location.

An overdistended bladder presents a tumor low in the abdomen that may be emptied by catheterization.

Prognosis: This is doubtful. Frequent tappings may give relief and sometimes cure.

Treatment: The underlying condition must be sought for and treated.

Diuretics and cathartics are sometimes used to advantage.

If the pressure is very great relief can be gotten by tapping. A trocar and canula are used. They must be sterilized. The skin is sterilized. Puncture is made at a point half way between the umbilicus and symphysis.

Homœopathic remedies are of great service.

Apis mellifica is indicated if the ascites is the most prominent symptom. It takes a day or two to act. It should cause an increase in the quantity of urine.

Apocynum is indicated if there is edema elsewhere as well as ascites.

Arsenicum album is indicated if the patient is exhausted and restless.

Lycopodium is indicated if the patient is an alcoholic.

CATARRHAL JAUNDICE

Synonym: Icterus.

Definition: Discoloration of the skin and mucous membranes by absorption of bile pigment.

Etiology: Usually due to obstruction of the bile ducts, either by inflammation within, or foreign bodies, as gall stones within, or pressure of tumors within or without. It may be due to infection or to mental shock.

Morbid Anatomy and Pathology: This varies with the condition. Usually the lining of the bile ducts is swollen and covered with tenacious mucus. The flow of bile is interfered with, it is thrown into the thoracic duct and lymphatics and thence travels through the system.

Symptoms: There may be more or less gastro-intestinal disturbance for a few days, with nausea, vomiting, pain, etc. Loss of appetite and general languor.

Then the icterus appears, first in the conjunctiva, then in the skin and mucous membranes elsewhere. The discoloration may be slight—just enough to detect in the conjunctiva, or it may be marked, the entire body being a yellow color.

There may be itching, scratching may cause skin lesions. In rare cases we have xanthelasma, bright yellow spots on the eyelids or elsewhere.

The stools are clay colored, there may be constipation, less often diarrhea. The movements have a bad odor.

The pulse is slow, 60 or less, the temperature subnormal, the patient is tired. These symptoms are due to poison of the bile acids.

The urine is high colored, has a yellow froth on shaking and stains filter paper. Shaking the urine with chloroform causes the color to settle at the bottom as a precipitate. Albumin and bile stained tube casts may be found.

The liver may be enlarged and tender and the gall bladder palpable.

In severe cases there may be profound disturbance, delirium, coma, convulsions. Mild delirium is not uncommon.

Complications and Sequelæ: Nose bleed.

Diagnosis: Is usually easy. In old persons with jaundice search for malignant disease, gall stones, etc.

Prognosis: This is good. Patients recover in two to six weeks.

Treatment: Rest is an important aid. The patient should be given a nearly fat-free diet. Milk, broths, eggs, lean meat, bread, cereals. My own preference is for a milk diet for a few days first, then add some of the other articles.

Rubinat water may be given if the constipation is marked.

For the itching cold water baths are good. Rub the skin with slices of lemon.

Bryonia is the most frequently indicated remedy. Constipation, thirst.

China is indicated in cases with a suspicion of malaria.

Mercurius solubilis, skin yellow, feces almost white; there may be pruritus.

Nux vomica, worse in morning. Very irritable.

Podophyllum, jaundice with nausea or with diarrhea.

HEMATOGENOUS OR HEMOLYTIC JAUNDICE.

This form appears in the infections; there is fever, rapid pulse and symptoms of infection. The discoloration is due to deteriorated blood and is always of very serious import.

ICTERUS NEONATORUM.

Synonym: Jaundice of the Newborn.

Etiology: Unknown.

Symptoms: Icterus neonatorum usually appears at the second or third day. In my cases it appeared from the fifth to the ninth days. It lasts a couple of days to two weeks.

As a rule, remedies are entirely unnecessary, the discoloration disappears of itself.

Podophyllum, bryonia, mercurius and other remedies may be tried if thought desirable.

ACUTE YELLOW ATROPHY OF THE LIVER.

Synonyms: Malignant Jaundice. Icterus Gravis.

Definition: An acute widespread autolytic necrosis of the liver cells of unknown origin, characterized by jaundice, toxemia and a reduction in the volume of the liver. (Osler.)

Historical Note: It was first described by Ballonius.

Etiology: May be primary or secondary. It is very rare, more common in women, and in them during pregnancy. It sometimes follows the acute infections. Occasionally it is the sequel of cirrhosis of the liver. Chloroform or phosphorus poisoning may cause it.

Morbid Anatomy and Pathology: The liver is shrunken and the capsule wrinkled. It is yellowish or yellowish-green in color. It may cut firmly or may be very soft. The cut surface is mottled yellow and red—the red being the more advanced stage. The lobules are indistinct. The microscope shows a fatty degeneration. The blood vessels are engorged. There are crystals of leucin and tyrosin.

The heart, frequently the kidneys, also show fatty degeneration. The spleen is enlarged. Ecchymoses throughout the body are common.

Symptoms: Usually, first mild gastro-intestinal symptoms. There is loss of appetite, languor, vomiting, tenderness over the epigastrium, possibly slight fever and jaundice.

After a few days or a week or two severe nervous symptoms supervene. These include headache, sleeplessness to delirium. The muscles twitch and sometimes there are convulsions. The violent stage lasts only a day or two then the patient sinks into a stupor and coma.

The temperature may not change much though it is frequently elevated just before death. The pulse is rapid and weak.

Vomiting usually persists throughout. The jaundice deepens and hemorrhages occur in the skin and mucous membranes. The stools are clay colored and constipated.

Pregnant women abort.

The urine contains bile pigment. Urea is diminished and leucine and tyrosine are present.

Physical examination shows the liver to be much diminished in size. The spleen is enlarged.

Complications and Sequelæ: Abortion in pregnant women.

Diagnosis: Early it is like ordinary catarrhal jaundice. The course of the disease, later, is quite typical—jaundice with vomiting, diminution in size of liver, the severe nervous symptoms, ecchymoses, and leucine and tyrosine in the urine.

Phosphorus poisoning may resemble it, but gastric symptoms are more marked. The liver is large and very painful, leucine and tyrosine are not present.

Prognosis: Almost invariably fatal, usually in a few days after onset of grave symptoms.

Treatment: None known that is of any use. We must meet symptoms as they arise.

HYPEREMIA OF THE LIVER.

Synonym: Congestion of the Liver.

Etiology: It may be active or passive.

The active form is due to indiscretion in diet, to alcoholism, to infectious diseases. It may be due to suppression of hemorrhage elsewhere, as piles and menses.

The passive form is due to chronic diseases that interfere with the portal circulation, particularly heart disease, emphysema, etc.

Morbid Anatomy and Pathology: The liver is enlarged. The hepatic veins are engorged. The center of the lobules is dark, the periphery paler. Changes may go on to atrophy of the cells.

Symptoms: The symptoms in acute congestion are very indefinite. There is anorexia and digestive disturbances.

In chronic congestion the liver is enlarged. It may be felt to pulsate. Jaundice is frequent. There is a feeling of pressure and weight. There may be hematemesis, ascites.

Diagnosis: Easy, as a rule.

Prognosis: The prognosis depends on the cause.

Treatment: The treatment depends on the cause.

CIRRHOSIS OF THE LIVER.

Synonyms: Chronic Diffuse Interstitial Hepatitis. Lænnec's Cirrhosis. Alcoholic Cirrhosis. Multilobular Cirrhosis. Portal Cirrhosis. Hobnail Liver. Gin Drinker's Liver.

Definition: A chronic diffuse interstitial inflammation of the liver, with secondary atrophy of the parenchyma.

Etiology: It is caused by excessive use of strong drink, hence is found mostly in middle aged men. Whiskey, gin and brandy drinkers are more prone to it than beer or wine drinkers, though these latter are not exempt. There may be a bacterial origin, as in children, following the infectious diseases.

Morbid Anatomy and Pathology: There are two types of cirrhosis of the liver:

1. *Atrophic:* The liver is much reduced in size. It is granular and hard to cut.

2. *Fatty:* The liver is enlarged, smooth, yellowish-white in color; it cuts with resistance.

There is cellular infiltration and formation of new connective tissue. The liver has a mottled appearance.

Cirrhosis of the liver is usually associated with a catarrhal condition of the stomach and intestines. There is obstruction of the portal circulation and consequent dilatation of the veins of the cardiac end of the stomach, of the esophagus, and about the umbilicus.

Symptoms: The disease is of insidious onset; for a long time there may be no symptoms. Usually the first thing noticed is disturbance of the digestive organs. There is loss of appetite, nausea, sometimes vomiting. There are eructations. The bowels are constipated. As the disease progresses the gastric symptoms become worse. There may be hemorrhage from the stomach and blood in the stools. There may be slight jaundice.

Finally ascites develops, due to the obstructed circulation.

Examination shows enlarged veins on the abdomen. The liver is enlarged at first and may be to the end. It can be easily felt below the ribs; the border is smooth. Later, in some cases, it becomes small and nodular.

The spleen is also found to be enlarged.

The urine is diminished, is high colored, and may contain albumin. Urates are plentiful.

In advanced cases the body is emaciated except the belly.

At the end there may be delirium, stupor and coma.

There is little or no fever.

Complications and Sequelæ: The heart and kidneys are usually diseased also. Bright's disease and cirrhosis of the liver are frequent companions. Tubercular peritonitis may occur.

Diagnosis: This must be made from the history and symptoms. Syphilis of the liver will give a history of syphilis.

Prognosis: This is always unfavorable. Death may be due to exhaustion or to some intercurrent or associated disease.

Treatment: The habits must be corrected. Alcohol and spices must be forbidden. Meat must be limited. Milk, eggs, vegetables and cereals may be permitted. An exclusive milk diet may be used for a time.

Laxatives and diuretics are sometimes useful.

Apis may be given for the ascites.

Apocynum is useful in alcoholics particularly.

Arsenicum album is of service where there is nephritis with the cirrhosis.

Mercurius is useful in cases with moist skin, but perspiration gives no relief. The liver is large and tender.

Chelidonium, pain under right shoulder blade; liver tender.

China, enlarged, hard liver. Bitter taste.

Nux vomica, podophyllum, hydrastis, phosphorus, may be indicated.

HYPERTROPHIC CIRRHOSIS.

Synonyms: Biliary Cirrhosis. Hanot's Disease.

Definition: Enlargement of the liver and spleen, chronic jaundice and abdominal crises. No ascites. This is a rare disease.

Historical Note: First described by Requier in 1849. Later, in 1876, by Hanot, whose name has been given to it.

Etiology: Not known. Occurs in men.

Morbid Anatomy and Pathology: Much enlarged liver, of a dark greenish color. Bile ducts catarrhal. Spleen much enlarged.

Symptoms: There is nausea and vomiting. There may be hemorrhage from the stomach. There is loss of appetite and general languor. There is chronic jaundice. Bile appears in the urine. The stools are dark. There is slight fever. The pulse is slow.

The liver and spleen are much enlarged. There is no ascites.

The patient presents symptoms of toxemia, and the disease runs a long course.

Diagnosis: The long course assists in diagnosis.

Prognosis is bad.

Treatment: This must be symptomatic.

BILIARY CALCULI.

Synonyms: Hepatic Colic. Gall Stones. Cholelithiasis.

Definition: Formation of calculi in the gall-bladder. Pain is caused by their efforts to pass through the common duct.

Etiology: Much more common in women after middle life. In women who have borne children. Other causes are sedentary habits, tight lacing. Constipation. Depressing emotions.

It is believed that micro-organisms are the starting point, causing a lithogenous catarrh.

Morbid Anatomy and Pathology: There may be one large stone or many, even hundreds, of small ones. They may be more or less round or polyhedral, showing signs of rubbing together. Small ones are like grains of sand. They are light or dark in color, varying according to the amount of bile pigment. There is a nucleus of bile pigment surrounded by crescentric layers of bile pigment. There is catarrh of the gall bladder.

They are found most often in the gall bladder, sometimes they have lodged in the bile ducts, occasionally they are found in the liver.

Symptoms: Sometimes there are no symptoms, stones being found on the operating or autopsy table that were entirely unsuspected.

In other cases the symptoms are mild, simply slight attacks of indigestion. Middle aged or elderly women presenting symp-

toms of frequent attacks of mild dyspepsia should always arouse suspicion of gall stones.

In other cases again definite attacks of gall stone colic occur. These attacks may be preceded by slight indisposition, chilliness, eructations, and so on. More often there is no premonitory sign, but a sudden attack of pain in the region of the gall bladder. The pain may be mild or severe at the start. It is referred to the epigastrium and to the right side, sometimes to the left. At the height of the paroxysm there is usually chilliness and vomiting with eructations. In severe cases there may be convulsions or delirium. The expression is anxious or agonizing. Sometimes there is considerable fever. Jaundice may or may not appear. One patient of mine, an old lady, has had several attacks in the past few years, but only once has jaundice appeared.

The attacks vary in duration from an hour or two to several days. If they last for a considerable length of time the pain varies from a dull ache to irregular attacks of acute and agonizing pain.

The entire abdomen may become more or less sore and tender. Usually the tenderness is very acute over the region of the gall bladder.

As a rule, the acute pains do not last very long, but the lesser pain and soreness take several days to disappear after the acute paroxysms have subsided.

The urine is scant, high colored. There may be much sediment. In other cases there may be a large amount of colorless urine.

Complications and Sequelæ: A stone may become impacted, causing obstruction of the cystic duct and consequent dilatation of the gall bladder. There may be a cholecystitis. A stone may become impacted in the common duct, causing cholangitis.

Perforation in rare cases occurs into the gastro-intestinal tract, into the portal vein or hepatic artery, into the peritoneum, or even extends through the abdominal wall. Sometimes gall stones cause intestinal obstruction.

Palpitation during an attack may occur; rarely fatal syncope.

Diagnosis: Sometimes mistaken for renal colic. In the latter the pain starts from the back. Intestinal colic is located about the navel. To seek stones pass the feces through a sieve with water.

Prognosis: From a given attack good; as to recurrence, doubtful. Gall stones may start cancer.

Treatment: Morphine may be necessary during an attack. Chloroform may be inhaled.

Applications of heat help sometimes.

Regulate the diet between attacks. Curtail meats, coffee and alcohol.

Calcarea carbonica 200 will relieve some cases very promptly. Given in water at five minute intervals it has never failed to stop the pain within half an hour in the old lady mentioned above.

Arnica is useful for the soreness left after the acuteness has worn off.

China. Pain comes and goes even during a paroxysm.

ABSCESS OF THE LIVER.

Synonym: Acute Suppurative Hepatitis.

Definition: One or more abscesses in the liver.

Etiology: May be caused by traumatism. Osler says injury to the head may be followed by liver abscess. It may be caused by entrance of bacteria through the blood or through the bile duct. By amebic dysentery; by emboli from other organs; by suppuration by way of bile ducts secondary to gall stones; by foreign bodies.

Morbid Anatomy and Pathology: There may be one large abscess, or more commonly the abscess may be multiple. When due to gall stones these are sometimes found in the pus.

The abscess may open into the abdomen, pleura, kidney, etc.

Symptoms: Abscess of the liver may exist without symptoms.

In other cases there is evidence of a septic condition, and septic temperature with chilliness at times and rapid pulse; sweating. Anemia and leucocytosis occur. Hiccough, vomiting, and, finally, emaciation. There may be diarrhea.

Physical examination shows enlarged liver, usually upwards. Sometimes there is fluctuation and bulging. Pain usually appears in the right hypochondrium or through the right shoulder and back. The patient prefers to lie on the right side. The complexion is sallow or yellowish.

Complications and Sequelæ: The abscess may break through to other parts. Osler says it perforated into the lung in nine of twenty-seven cases. This caused coughing up of pus or reddish-brown expectoration. In other cases the perforation may be into the peritoneum, or even externally.

Diagnosis: Gall stones may cause pain similar to that of abscess. It is more apt to be paroxysmal.

Hydatid tumor, cancer, even empyema, may simulate abscess of the liver.

In doubtful cases the aspirating needle should be used. Insertion should be made in the seventh mid-axillary space.

Prognosis: This is bad. Cases may recover spontaneously.

Treatment: Surgical treatment is indicated once the diagnosis is made. Until then symptoms must be met as they arise.

CANCER OF THE LIVER AND BILE DUCTS.

May be carcinoma or sarcoma.

Etiology: Cancer of the liver is usually secondary. It may follow cancer of any part of the body, though it most often follows cancer of the digestive organs.

Cancer of the bile ducts is more often primary. It sometimes follows biliary calculi.

Cancer of these parts occurs late in life—from forty to sixty years—and in men more often than in women.

It may follow trauma. It is sometimes hereditary.

Morbid Anatomy and Pathology: The liver may become much enlarged. A patient of mine at the Flower Hospital had a liver that weighed nine pounds. It extended below the umbilicus. There are cancerous nodules throughout the mass. All types of cancer are found, usually carcinoma if secondary to cancer of the digestive organs; sarcoma if secondary to sarcoma of the eye or skin.

Symptoms: There may be none for a long time. Late in the disease there is the cancerous cachexia, emaciation, sallow complexion, sometimes jaundice. There is anorexia, then nausea and vomiting. There may be fever. There may be edema or ascites, or both, due to pressure.

Pain is always present in advanced cases.

One should always be suspicious of cancer of the liver when there is cancer of the stomach. It usually appears sooner or later.

The liver is enlarged, irregular in outline, and moves with respiration.

Diagnosis: Cancer of the liver must be differentiated from other enlargements of the liver by the general symptoms.

Prognosis: Always fatal.

Treatment: Can only be palliative.

AMYLOID LIVER.

Synonyms: Waxy Liver. Lardaceous Liver.

Etiology: Amyloid liver is part of a general condition due to suppuration or to tuberculosis in some other part of the body.

Morbid Anatomy and Pathology: The liver is large, light in color, anemic. It is firm under the knife. Iodine changes the color of cut surfaces.

Symptoms: There are no special symptoms. The liver is enlarged and painless. The spleen is enlarged. The kidneys are also large. The urine contains albumin and casts. There is a focus of suppuration somewhere in the body.

Diagnosis: Amyloid liver may be suspected when there is progressive painless enlargement with a suppurative process going on somewhere.

Prognosis is doubtful.

Treatment: This cannot be direct, but must be of the underlying condition.

FATTY LIVER.

Etiology: Fatty liver occurs in obesity; in heavy drinkers; in tuberculosis; in cancer.

Morbid Anatomy and Pathology: The liver is enlarged, is firm, and presents a fatty infiltration.

Symptoms: There are none specific except the enlargement.

Diagnosis: Must be made from the history.

Treatment is that of the original disease.

SYPHILIS OF THE LIVER.

Etiology: This is sometimes a part of general syphilis. It may be acquired or congenital.

Morbid Anatomy and Pathology: The liver is enlarged, hard and uneven. It may contain gummata. The vessels show a syphilitic endarteritis.

Symptoms: Special symptoms are due to the enlargement. There may be anorexia, vomiting, etc. The liver is large and tender. Ascites may occur.

Diagnosis: This is made from the history.

Prognosis: Doubtful.

Treatment: Antisyphilitic. (See chapter on syphilis.)

ANOMALIES OF THE LIVER.

The liver may be *transposed.*

It may be *movable.*

It may be *furrowed* from tight lacing.

ACUTE PANCREATITIS.

There are three forms of acute inflammation of the pancreas —*hemorrhagic, suppurative* and *gangrenous.*

Etiology: The symptoms in all three forms are very similar. Men are more often affected than women, and stout persons more often than thin ones. Most of the patients give a history of repeated attacks of indigestion, indefinite pains about the epigastrium, and so on. Among the exciting causes of acute pancreatitis are impacted gall-stones,. extension of inflammation from neighboring structures, general infection, immediate bacterial infection, gastro-duodenal catarrh, and traumatism.

Morbid Anatomy and Pathology: In *hemorrhagic pancreatitis* the pancreas varies in color from a light to a deep or bluish-red color. It is enlarged in whole or in part. The hemorrhages may be few, small, or numerous; diffuse or localized. If only a part of the gland is affected it is most frequently the head.

In the *suppurative* form there are abscesses; there may be but one, there may be a large number. Thrombosis of the portal and splenic veins is a common accident.

In the *necrotic* or *gangrenous* form of pancreatitis, the suppuration proceeds to gangrene. Sometimes portions of the pancreas slough off and are discharged through the intestines. At other times the suppurating process extends to adjoining structures, as suppurative peritonitis.

Fat necrosis is frequently present in pancreatitis. It is evidenced by minute whitish areas scattered throughout the gland tissue.

Symptoms: Fitz says: "Acute pancreatitis is to be suspected when a previously healthy person, a sufferer from occasional attacks of indigestion, is seized with violent pain in the epigastrium, followed by vomiting and collapse, and, in the course of twenty-four hours or more, by a circumscribed swelling, tympanitic or resistant, with slight rise of temperature."

Acute pancreatitis is probably more common than was formerly supposed. Its onset is sudden. Acute pain and distention develop in the epigastrium. There is vomiting, perhaps collapse. Death may occur in a very few hours. Where hemorrhage is severe, pressure on the solar plexus is supposed to cause the alarming symptoms.

In cases going on to suppuration there is the irregular fever usually associated with pus. Other cases may or may not have an irregular temperature—rarely over 100° F. In some cases the temperature is subnormal. Constipation is more frequent than diarrhea. Jaundice appears, and if the disease persists, there is rapid loss of weight. Delirium may supervene in severe cases. The pains are paroxysmal in protracted cases. Tender spots develop, simulating a localized peritonitis.

Examination of the stools frequently shows the presence of

fat and of undigested muscle fibres; rarely, in the gangrenous form, shreds of pancreatic tissue. Examination of the urine may show albumin or sugar. The presence of sugar is said to indicate that the islands of Langerhans are involved.

Diagnosis: The conditions most likely to be mistaken for acute pancreatitis are: (1) Intestinal obstruction. The sudden onset is less acute in the latter. Fecal vomiting appears sooner or later. Constipation is absolute, no gas even passing through the bowel.

(2) Biliary colic, and (3) gall-stones give a different history. (4) Perforating gastric ulcer also has a different history. The pain has been more or less constant for a considerable time, and hematemesis is a common symptom. (5) Irritant poisoning shows some external evidence and gives a different set of symptoms.

Prognosis: The prognosis in acute pancreatitis is unfavorable. The patient may die in a few hours, more rarely the symptoms may persist for three or four weeks. The suppurative form may become chronic, and diabetes may develop.

Authentic cases of recovery are very rare. Osler reports one case where obstruction of the bowel was diagnosed. On operation a dense mass of fat necrosis of the head of the pancreas was found and removed. The patient recovered. He returned with similar symptoms four years later, but refused operation and was lost sight of.

Treatment: The treatment of acute pancreatitis is the treatment for collapse. Absolute quiet and stimulation are indicated. *Morphine* may be necessary. In the light of Osler's case operation may be advisable.

So far as homœopathic remedies are concerned, the totality of the symptoms, as always, must be considered.

Phosphorus has fat in the stools; and may be tried if other symptoms correspond.

Iodine has the general symptoms of pancreatitis.

Hale recommends *mercurius* and *iris versicolor*. He says both these drugs have produced it experimentally.

CHRONIC PANCREATITIS.

Etiology: Chronic pancreatitis may result from closure of the gall duct by impacted gall-stones; by chronic catarrh of the gall duct; by extension of inflammation from neighboring parts. It occasionally follows acute pancreatitis. We may have a sclerosis due to syphilis or alcoholism.

Sometimes chronic pancreatitis is associated with or follows ulcer of the stomach, tumors of the stomach or suprarenal bodies, aneurysm of the aorta, etc.

Morbid Anatomy and Pathology: There is an overgrowth of fibrous tissue with atrophy of the cellular tissue, especially at the head of the organ. The pancreas is grayish in color. It may be cartilaginous, granular or smooth in consistency. Calcareous spots appear throughout in some cases. In others white spots showing fatty necrosis. The duct of Wirsung is usually tortuous or dilated.

Symptoms: The symptoms of chronic pancreatitis are usually obscure. Those that we have are of the digestive system. There is more or less deep-seated pain in the epigastrium, with tenderness and faintness. The pain is apt to be paroxysmal. There is flatulent dyspepsia, diarrhea, slight jaundice, aversion to sweets, fatty foods, and meats. There is loss of weight. Fatty stools are common.

Erdmann (*Medical Record,* May 12, 1912) says the stool is characteristic, it "is voluminous as to the intake of food; not only one stool a day, but several, the color being of a peculiar gray-yellow character, rarely with formation or shape, and resembling a 'raising of bread' in which the fermentation bubbles of the yeast are exploding on the surface. The impression is also made that the large stool weighs far less than an ordinary movement. There is also frequently seen evidence of free fat globules to large collections of fat floating on the surface or mixed with the mass. Muscle fibres are in evidence in many stools."

There may be albuminuria or glycosuria. When the disease involves the islands of Langerhans diabetes is the result, although it must not be forgotten that we may have diabetes with-

out the involvement of the pancreas, or chronic pancreatitis without diabetes.

If the disease takes the form of abscesses we have the symptoms of a suppurative process.

Diagnosis: This is difficult always, and may be impossible.

Prognosis: Prognosis is doubtful at best. If glycosuria develops the prognosis is grave. A very small portion of normal pancreatic tissue may be sufficient for the needs of the body; or extensive disease may be found at autopsy which was unsuspected during life.

Treatment: This is mainly dietetic. Fats and starches should be restricted. Only such food should be taken as can be disposed of without pancreatic digestion. Carbonated waters are of service.

Pancreatinin will supply the deficiency in pancreatic juice.

Surgical interference may prove of benefit, especially in the suppurative form.

Homœopathic remedies must be used on the totality of symptoms.

Hughes mentions *iodine* as his choice in chronic pancreatitis.

The digestive symptoms might be covered by such drugs as *carbo vegetabilis, lycopodium, nux vomica* and *phosphorus*.

PANCREATIC CYSTS.

Etiology: Pancreatic cysts may be the result of traumatism, inflammation, or of unknown causes. They may result from the closure of Wirsung's duct.

Morbid Anatomy and Pathology: Various forms of cyst may occur—namely: retention cysts, apoplectic cysts, hydatid cysts, congenital cysts, and cysts caused by or associated with cancer.

There may be one or more cysts. They are situated behind the stomach. They contain a grayish or brownish, viscid, alkaline liquid—of a specific gravity of 1.010 to 1.024. There may be only a few ounces of this fluid or there may be many gallons. In some cases the fluid acts like the pancreatic secretion. When cyst of the pancreas is suspected aspiration is indicated.

If fluid is obtained its properties should be tested to see if it contains the pancreatic juice.

Symptoms: The symptoms are variable. We may have epigastric pain, with nausea and vomiting. There may be either constipation or diarrhea. Occasionally intestinal hemorrhage takes place. Associated with the digestive disturbance is loss of flesh. Free fat and undigested muscle fibre may appear in the feces, and sugar in the urine. If the cystic tumor becomes of any size an elastic fluctuating mass can be palpated in the epigastrium and to the left. Aspiration may draw off fluid that will emulsify fat, turn starch into sugar, and digest albumin. This is the action of pancreatic juice. It must be remembered, however, that cyst of the pancreas may exist and the content have no digestive power.

Diagnosis: One method of differentiating cysts of the pancreas from cysts of the other organs consists in testing the fluid withdrawn as above.

Ovarian cysts extend and increase from below upward, there are no intestines below them. Pancreatic cysts extend and increase from above downward, and intestines do lie below.

Cyst of the kidney is one-sided.

Treatment: Surgical interference is necessary. After the fluid is removed remedies may be given to endeavor to stop any further development.

CANCER OF THE PANCREAS.

Etiology: Cancer of the pancreas is said to occur more frequently in men than in women, and usually late in middle life.

Morbid Anatomy and Pathology: As a rule, cancer attacks the head of the pancreas. Sarcoma is more often of secondary, carcinoma of primary origin. Nearly always the disease extends, and secondary infection of neighboring organs occurs.

Symptoms: The symptoms are more or less obscure till the disease is far advanced. There is digestive disturbance. There is deep-seated epigastric pain which may be constant or paroxysmal. The patient may collapse with the pain. There is jaundice, due to pressure on the bile duct. Ascites may set in later from pressure on the portal vein.

Glycosuria is only occasional. Free fat in the stools is rare. Fitz says undigested muscle fibres in the stools in the absence of diarrhea is suggestive.

As the disease progresses we have the cachexia of malignant disease—rapid loss of flesh and strength, and anemia.

As the tumor grows it may become palpable.

Diagnosis: Cancer of the pancreas may be mistaken for intestinal or gastric tumor. The pancreatic tumor is deeper. Moreover, jaundice is usual with pancreatic cancer, rare with gastric or intestinal. Pain is more severe in pancreatic cancer.

Prognosis: The prognosis is bad. A few operative cases have been cured.

Treatment: Surgery is the only sure means of relief.

CONCRETIONS OF THE PANCREAS.

Synonym: Stone in the Pancreas.

Etiology: Stone in the pancreas is due to altered secretion. There is first stasis and then formation of concretions. Most of them are found to be composed of carbonate and phosphate of lime. They vary in size and number.

Symptoms: The symptoms, when there are any, are symptoms of colic. The colic is more to the left than in gall-stone colic. There is no jaundice. There may be glycosuria, and fatty stools. Carbonate and phosphate of lime may be found in the stools.

Diagnosis: The cases are so similar to hepatic colic that the diagnosis is rarely made and then more rarely confirmed.

Prognosis: The prognosis is uncertain.

Treatment: Surgical.

The various remedies of service in stone of the kidney and gall-stone should be of service here.

Calcarea carbonica, berberis, baryta carbonica, etc.

HEMORRHAGE OF THE PANCREAS.

Synonym: Hemorrhage of the Pancreas is sometimes called Apoplexy of the Pancreas.

Etiology: Hemorrhage into the pancreas may be due to traumatism, to passive congestion, to gall-stones, to chronic gastro-duodenal catarrh, to hemorrhagic diseases, or it may accompany or follow the acute infections. It is more common beyond middle life and in stout persons. Alcoholism is considered a predisposing factor.

Morbid Anatomy and Pathology: The hemorrhage may be confined to the head, the body, or the tail of the pancreas, or it may be general. As a rule, the source of the hemorrhage is not found as there is no rupture of a large vessel. The organ may be enlarged, or it may be of normal size.

It is usually of a dense purple color and soft and friable. The subperitoneal fat and lesser omentum are also frequently infiltrated with blood. When the hemorrhages are old, the hemorrhagic spots turn yellow. In stout persons the pancreas and omentum show fatty infiltration.

The interstitial tissue of the pancreas may be fibrous.

Symptoms: The symptoms are very variable and uncertain. In rare cases, according to Strümpell, there may be none. As a rule, however, the onset is sudden and severe. There is intense distress in the epigastrium, accompanied by vomiting and tympanites. There is a peculiar cyanosis and lividity. Hiccough is a frequent symptom. Pain in back. There may be dyspepsia and collapse. According to Da Costa the disease is sometimes ushered in by chills. In less severe cases the pain may be slight. Pressure reveals tender spots. There may be either constipation or diarrhea with or without fat in the stools.

The temperature may be subnormal, or it may be 99° to 100° F. The most important physical sign is an abdominal tumor.

Glycosuria may appear, also albuminuria.

Examination of the blood shows a polynuclear leucocytosis.

Complications and Sequelæ: Hemorrhage into the pancreas may be a symptom of arteriosclerosis. It may be accompanied or followed by pancreatitis, cysts or by cancer of the pancreas. It may cause peritonitis.

Diagnosis: The diagnosis of hemorrhage of the pancreas is rarely made before death.

Some of the conditions mistaken for it follow.

Peritonitis with perforation will give a history of fever and abdominal tenderness preceding signs of collapse.

Appendicitis has the localized tenderness over McBurney's point, and rigidity of the right rectus muscle.

Intestinal obstruction causes distention of the bowel, complete constipation, and later fecal vomiting.

Ulcer of the stomach, as a rule, occurs in younger subjects, and is associated with anemia.

Impacted gall-stones cause pain radiating to the right.

Prognosis: In the majority of cases death ensues in twenty-four to thirty-six hours. It is supposed to be due to pressure on the cardiac plexus or semilunar ganglion. A few hours after apoplexy of the pancreas inflammation ensues.

Recovery is rare. Sometimes the history of a patient and the appearance of the pancreas after death show that at some time hemorrhage into the organ has taken place.

Treatment: The indications for treatment are the relief of pain and stimulation. *Morphine* may be necessary. An operation may be of service.

SECTION X.

Diseases of the Kidneys and Bladder.

CONGESTION OF THE KIDNEYS.

Etiology: Congestion of the kidneys may occur during Bright's disease, during the course of the acute infections, in chronic heart and lung affections. It may be caused by exposure. It may be caused by pressure of abdominal tumors, or ascites, or during pregnancy. Certain drugs may cause it, as *turpentine, cubebs, cantharides,* etc.

Morbid Anatomy and Pathology: The kidneys are large, firm in consistency, and of a dark bluish-red color. The medullary portion is darker than the cortex.

Symptoms: The urine is diminished, dark, and of high specific gravity. Urates are present. Hyaline casts may be found. There may be a slight albuminuria.

Diagnosis: Congestion of the kidneys is nearly always part of another condition, a venous stasis due to some influence outside the kidneys.

Treatment: Rest in bed is usually indicated. Treatment must be directed to eliminating the cause.

INFARCT OF THE KIDNEY.

Etiology: Infarct in the kidney is an end result of valvular heart lesion.

Morbid Anatomy and Pathology: Wedge-shaped infarcts are found, due to sudden cut-off of the circulation by floating particles of detritus. The infarct may disintegrate, it may be absorbed.

Symptoms: There may be no symptoms in some cases. In others there is sudden more or less acute pain like renal colic. The urine may become bloody.

Diagnosis: In heart cases, sudden pain in the region of the kidney, followed by bloody urine, is almost pathognomonic.

Treatment: There is none. If pain is severe palliatives may be necessary.

BRIGHT'S DISEASE.

Synonym: Nephritis.

Definition: "A disease characterized by degeneration of kidneys whereby the excretory function is so impaired that urea is not sufficiently eliminated by the blood; any disease of the kidneys marked by the persistent presence of albumin in the urine and attended by disintegration and wasting of the organ."

Historical Note: In 1827, Richard Bright, one of the attending physicians to Guy's Hospital, London, first called attention to the association of albuminous urine, dropsy, and diseased kidneys. This report is one of the most interesting monographs in medical literature. His description of the clinical symptoms observed and of the macroscopic appearances of the kidneys found after death will forever remain a classic. Bright reported a number of cases of dropsy with death. Post-mortem examination of some of the bodies showed diseased kidneys. In studying the clinical histories in conjunction with the lesions found on the autopsy table, Bright discovered that when the urine of dropsical patients had been coagulable by heat, there were found at the necropsy extended pathological changes in the kidneys; whereas, on the other hand, when the urine of dropsical patients had not been coagulable by heat, the kidneys, as a rule, were found to be healthy. He summed up these observations in the following words: "I have never yet examined the body of a patient dying with dropsy attended with coagulable urine, in whom some obvious derangement was not discovered in the kidneys."

Heretofore, dropsy had been recognized as due to impeded circulation, either from disease of the heart or of the liver, or as due to the mechanical interference of tumors. Bright's observations led him to believe that dropsy might also be due to impeded circulation from the disease of the kidneys; for in many of his cases the heart and liver were but little, if at all, diseased, and the kidneys only were at fault. Nevertheless, he did not overlook the fact that hypertrophy of the heart seemed

sometimes to be associated with degenerated kidneys. In some of his cases Bright also noted variations in the amount of albumin in the urine from day to day; in other cases he found albuminous urine and degenerated kidneys, but no dropsy.

The publication of his series of cases by Bright directed special attention to the study of diseased kidneys, and his name was, by common consent, given to the disease first described by him.

Theories as to Its Origin: It will be well to stop for a moment to consider theories as to the origin of Bright's disease. Bright himself ventured the following proposition: "It is probable that the altered action of the kidney is the result of the various hurtful causes influencing it through the medium of the stomach and the skin, thus deranging the healthy balance of the circulation, or producing a decidedly inflammatory state of the kidney itself." In other words, he considered the changes in the kidneys to be the result and not the cause of disease.

The disease is general with specially marked degenerative changes in the kidneys. Dickinson, Millard, and others, consider the changes in the kidneys to precede the appearance of albumin in the urine and deterioration of the blood. On the other hand, Semmola maintained the contrary. By experiment, and deduction therefrom, he concluded that the blood always first became deteriorated so as to contain an increased amount of albumin. This deteriorated blood may be due to direct poisoning, as in the acute infectious diseases, or in the ingestion of certain drugs. Otherwise, Semmola believed the deterioration to be due to interference with the respiratory function of the skin, thus causing the retention in the blood of noxious substances that would else be eliminated.

To me Semmola's views seem to be the correct ones. His experiments certainly are convincing. In one series he varnished half the bodies of several dogs: in a few days albumin appeared in the urine, subsequently followed by pathological changes in the kidneys.

Bradford has been unable to find any direct relationship between the blood-vessels of the kidneys and of the skin. It can be seen, however, that when the respiration of the skin is in-

hibited the excrementitious matters thus kept in the blood are perforce required to circulate through the kidneys. It is this impure blood which causes the pathological changes in the kidneys in Bright's disease.

Classification: As the changes that take place in the kidneys in Bright's disease have become better understood they have been found to be due to a true inflammation. Therefore the word *"nephritis"* has come to be applied to it. Different observers have made different classifications of nephritis. From a clinical standpoint the best classification is Flint's division of the disease into acute nephritis and chronic nephritis. Strümpell describes: (1) acute nephritis, (2) sub-chronic and chronic forms of nephritis, with the exception of the genuine contracted kidney, (3) contracted kidney. This is the classification most generally followed. Although typical cases of this so-called subchronic nephritis and of the contracted kidney differ in essential particulars, yet frequently it is clinically impossible to distinguish them. Moreover, the treatment of Bright's disease is mainly symptomatic, and as the same symptoms may be present in all its forms the same methods of treatment may hold good in all its forms.

From the pathological point of view again there is the acute and the chronic nephritis, but with many and varying anatomical divisions according to the part of the organ mainly affected. Bright described three forms of diseased kidney: (1) The organ loses firmness, it looks yellow and mottled. The kidney may be slightly or much enlarged. (2) The surface is granulated as though it had been sprinkled with grains of sand. The interstitial tissue is affected; it is opaque, white and occasionally no anasarca accompanies it. (3) A contracted kidney with less interstitial inflammation. Later in his report he said it was possible that each of these forms was merely a different stage of the same progressive degeneration.

This idea, that varying forms of kidney found in Bright's disease were merely stages of one disease, has been advocated by some of the later students of the subject, notably by Frerichs, who divides nephritis into, (1) the stage of hyperemia

and commencing exudation, (2) the stage of exudation and commencing change of the exudation, and (3) the stage of degeneration and atrophy. From this simple classification there are numerous others varying all the way to Councilman's fourteen anatomical divisions of parenchymatous nephritis alone. Delafield and Prudden include congested kidneys under the generic term Bright's disease. This is a mistake, as, although albumin and dropsy may occur, the dropsy at least is usually due to other causes, and the kidneys, after death, show none of the changes recorded by Bright.

A careful review of much of the literature of Bright's disease, together with a close study of clinical cases and an opportunity for post-mortem work, suggest to me the desirability of dividing the subject into *acute Bright's disease* and *chronic Bright's disease.*

Roughly speaking, the kidney is composed of the parenchyma or secreting portion, and of the connective or interstitial tissue. The entire organ is richly endowed with blood-vessels. It is subject to all the changes of inflammation from simple congestion to exudation and degeneration of plastic material. In some diseased kidneys the principal damage done seems to be in the parenchyma of the organ; in others the greatest change is in the interstitial tissue. Bright's disease has therefore been divided into *parenchymatous* and *interstitial nephritis.* Anatomically, this is convenient. *It must be borne in mind, however, that all subjects of Bright's disease present kidneys that are more or less damaged in every part. The terms parenchymatous and interstitial are merely relative.* Clinically, it is often impossible to differentiate between these forms.

ACUTE BRIGHT'S DISEASE.

Synonym: Acute Nephritis.

Etiology: The most frequent direct exciting cause of acute Bright's disease is scarlet fever. The specific poison probably acts directly on the kidneys. Smith reports an acute nephritis in a nurse who had cared for a case of scarlatina, but who at no time presented any other of the effects of the specific poison.

Flint also reports that he has seen acute nephritis occur in several attendants on scarlet-fever patients. He says that most of these patients had had scarlet fever years before. Scarlatinal nephritis may develop at any stage of the original disease. It is most apt to come on toward the close of the third week. It may come on as late as a month after convalescence is established. Smith says nephritis occurs most often in mild cases, and usually follows exposure to cold. This tendency of scarlet fever to produce Bright's disease makes it imperative on the attendants that all possible exposure of the patient be guarded against. Even then it may develop in the most carefully cared-for patient.

Acute Bright's disease may occasionally follow the other infectious diseases, as diphtheria, pneumonia, typhoid fever, typhus fever, relapsing fever, small-pox, measles, erysipelas, cholera, dysentery, and so on. Malaria occasionally produces acute Bright's, but is more apt to cause the chronic form.

Exposure to cold associated with dampness is a frequent cause of the acute nephritis. Cold alone is not so bad. Bright's disease is not a disease of cold countries.

Alcohol has long been considered to be a potent cause of acute nephritis. It is more probably due to the carelessness of alcoholic habitués in exposing themselves, and to their well-known lessened ability to react against malign influences than to the alcohol itself.

Pregnancy is another cause of acute nephritis. It occurs more often in primiparæ. It is most apt to develop toward the end of gestation.

Certain poisons, if ingested in sufficient quantity, will cause acute nephritis. The most common ones are arsenic, carbolic acid, chlorate of potash, turpentine, and some of the mineral poisons. Millard reports a case caused by essence of ginger that had been taken to cure dyspepsia.

Extensive burns are very apt to cause acute Bright's disease. Certain skin diseases are also said to be responsible for a few cases.

Morbid Anatomy and Pathology: *Acute parenchymatous ne-*

phritis. Acute parenchymatous nephritis has been variously called acute tubular nephritis, acute glomerular nephritis (Klebs), acute diffuse nephritis, acute croupous nephritis (Millard), acute degeneration of the kidneys (Delafield and Prudden).

On post-mortem examination the kidney is usually found to be enlarged. The increase in size may be slight, or it may be so great as, in a case reported by Dickinson, to burst the capsule. The whole organ may be enlarged, but the principal increase is in the cortex. The capsule is non-adherent. The surface is smooth and hyperemic.

On section the cut surface exudes blood. The kidney substance is hyperemic. The Malpighian bodies are injected and show in the cortex as minute red spots. The cones are decidedly reddened, in some cases purple in color. The pelvis of the kidney also shows a heightened color. In fact, the whole organ may be so congested as to be almost of a chocolate hue. There is a fine illustration of this form of kidney in Bright's original memoir. Other kidneys are paler than those above described. On washing the cut surface the distended tubules, containing exuded fibrin, fatty and destroyed epithelium, and so on, show themselves as yellowish streaks, thus giving the washed section more or less of a buff color.

Congestion and then inflammation take place. There is cloudy swelling of the epithelium lining the tubules. This has a tendency to become loosened and to fall off into the tubes. After the epithelium has become impaired there is an exudation of serous fluid, leucocytes, and red corpuscles into the tubes, and to a slight extent into the interstitial tissue. The tubules are now filled with a mass of epithelium, granular matter, leucocytes, red corpuscles, and fibrin, so great as often to obliterate the channels. This diminishes the secretion of urine. It is the mass of débris which, as before stated, gives the gross appearance of a pale infiltration. Fatty degeneration, according to Coats, appears in portions of the epithelium. Casts are found in the convoluted and straight tubules and in the loops of Henle. Sometimes fibrin is found next the basement membrane. It

probably gets there through the tubules. This is said by some observers to seal the tubules and thus to cause a diminution of urine.

In more protracted cases, and in scarlatinal nephritis the interstitial tissue becomes involved. Dickinson says that when this point is reached the process is at the dividing line between the acute and the chronic forms.

Acute interstitial nephritis: Councilman has described what he calls an acute interstitial nephritis. He defines it as "an acute inflammation of the kidney characterized by cellular and fluid exudation in the interstitial tissue, accompanied by, but not dependent on, degeneration of the epithelium; the exudate is not purulent in character, and the lesion may be both diffuse and focal." It accompanies or follows the infectious diseases, as scarlet fever, diphtheria, and so on. Councilman believes it to be the most common form of kidney found in the infectious diseases of children. It is also found in some of the other infections. This, together with its focal character, leads Councilman to consider the disease to be bacterial in origin, although as yet he has been unable to demonstrate that as a fact. It is only fair to other observers to state that many of them have noted the great interstitial changes in nephritis following the acute infections, especially scarlet fever.

Ryffel says acute nephritis is almost certainly not due to bacterial infection of the kidney itself, but to toxic substances which reach the kidney by the blood stream. It is, therefore, to be regarded rather as the result of disease occuring in other parts of the body than as a disease in itself.

To the eye, Councilman's acute interstitial kidney shows an enlarged organ. It may be very much enlarged, or it may be slightly so or not at all. The capsule is thin and easily removed. The kidney is pale, grayish, and mottled. In some cases the surface is irregular. On section there is found to be an increase in size of the cortex. The glomeruli are not injected but have a grayish appearance. The kidneys are soft, lax, and friable. They exude a milky fluid, possibly degenerated epithelium. Councilman says it is not pus. Occasionally the macroscopic changes may be slight.

Under the microscope there is found to be an intense cell infiltration of the interstitial tissue. This is both general and local. It is most marked just beneath the capsule and at the base of the pyramids. These cells are packed in the middle of the cortex and around the glomeruli. They resemble the plasma cells of Unna. The tubular epithelium may be destroyed, or its change may be slight. The blood-vessels are always changed, and the plasma-like cells found in them.

General Symptoms: There are three symptoms associated with all forms of Bright's disease, the pathology of which will be considered at this point. They occur in both the acute and the chronic forms of nephritis. I refer to the appearance of *albumin* in the urine, *dropsy,* and *uremia.* It is this trio of associated symptoms that individualizes Bright's disease. It is this trio of associated symptoms that, found in every form of the disease, frequently makes clinical differentiation of the varying pathological forms of kidney impossible. As a rule, all three symptoms appear at some time during the history of each case. Many exceptions, where one or another of these symptoms was absent, have been reported. Of the three *albumin* in the urine is the most frequently found. Occasionally a case will run its course without the detection of albumin. When this happens I believe that it has been overlooked, either by too infrequent examination of the urinary secretion, or by faulty methods of analysis. Cases of Bright's disease in which albumin is actually never present in the urine I believe to be extremely rare. It may be absent for a time, but not during the entire course of the disease.

The albumin found in the urine of nephritis is from the blood. Under normal conditions this is not found in the kidney secretion. The cause of its appearance is still a mooted question. Cohnheim's theory is the generally accepted one. He claims that it is due to change in the epithelium of the glomeruli, thus allowing the albumin to pass through. According to Semmola (*vide supra*), this change in the renal epithelium is produced by the deteriorated blood. That change in the glomerular epithelium alone allows of the passage of albumin is corrobor-

ated by the fact that in chronic interstitial nephritis, when the glomeruli are largely destroyed, albumin is scant and sometimes absent.

Dropsy is the next most constant symptom of Bright's disease. It is nearly always found in the acute form, and is found in the majority of cases of the chronic disease. It may not be evident for a long time in some of the latter, but if carefully searched for it will usually be found to a varying degree, especially toward the end, in nearly every case. Dropsy consists of an infiltration into the tissues and serous cavities of a watery fluid. This dropsical fluid is pale or colorless, of low specific gravity—1.006 to 1.012—and composed of water, salts, and extractives. It contains albumin. The fluid comes from the blood. Cohnheim's theory, that dropsy is due to changes in the walls of the blood-vessels, is to-day generally accepted. Renal dropsy, according to Lazarus-Barlow, exhibits itself first in the loose subcutaneous tissue. He claims this to be a proof of Cohnheim's theory. Dropsy due to defective heart's action is found in dependent portions of the body. So is dropsy due to obstructed circulation. If the heart recovers its tone or if the obstruction is removed, the dropsy promptly disappears. Further than the above the pathology of dropsy is not understood.

The third of the trio of symptoms is *uremia*. This is found in any form of the disease, but is most often associated with chronic interstitial nephritis. The pathology of uremia is obscure. In the Goulstonian lectures delivered before the Royal College of Physicians in March, 1898, Bradford devoted some attention to the subject of uremia. In conclusion Bradford asks: "Is uremia due to a retention of some body or is it due to the formation of something? Experiment and clinical evidence is against the retention theory. The only uremia of the laboratory is the latent uremia of the clinic. It is probably due to some abnormal product of disordered metabolism." In summing up he says, "(1) Uremia is not due to sudden retention. (2) In kidney disease and after experiment there is evidence of great tissue disintegration; and it is probable that uremia depends on this."

That uremia acts as a powerful poison to the nervous sys-

tem is well known. It exhibits itself as headache, drowsiness, stupor, coma, convulsions, reflex nausea, and vomiting. Before an outbreak of uremia there is a diminution in the amount of urea excreted by the kidneys. There is an increase in the percentage of urea in the blood. These few facts comprise the sum total of our knowledge of the pathology of uremia.

To this I might add that the patients I have seen die from bichloride of mercury poisoning have not presented the typical symptoms of uremia. One man I observed closely during the duration of his illness—five days—in August, 1914. He had complete suppression. There was no sign of uremia.

Symptoms: Acute Bright's disease is a self-limited disease running a course of two weeks to two months, and ending in recovery, chronic nephritis, or death. The onset is usually sudden. It may be ushered in by a chill or chills, accompanied in some instances by a slight rise in temperature, which rarely goes beyond 103° F. Children are more subject to increased temperature than are adults. The skin is dry. This last is characteristic. There is loss of appetite, possibly with nausea and vomiting. The respiration is quickened. Pain in the loins in the region of the kidneys is often present, especially on deep pressure. The pulse rate is increased. Sometimes there is hypertrophy of the heart, but this is not common. A more frequent symptom is rapid dilatation. The percentage of urea in the blood is increased. Anemia develops early. This, with the edema, gives the patient a peculiar waxy look. Epistaxis is quite common. Purpura may appear.

In the vast majority of cases the first symptom noted is a sudden appearance of edema in one or more parts of the body; usually it is a general dropsy or anasarca. This is one of the earliest and most frequent symptoms of the acute disease; it is rarely absent. After exposure to cold anasarca may set in within twenty-four hours. It usually shows itself first in the loose areolar tissue about the orbit. It may show itself first about the ankles. As a rule, however, it develops suddenly and is general. Nearly always it is a marked symptom; it may even be so great as to break the skin. Quite often it will exhibit it-

self as edema of the lungs. If there is any reason to suspect acute nephritis, edema must be most carefully searched for, otherwise it may be overlooked. I have seen two such cases. Both patients were emaciated. In one edema was not suspected. At the necropsy I found several quarts of fluid in the abdomen. The other case showed a slight puffiness about the eyes, but ascites was said to be absent. On most careful examination I was able to demonstrate it. My diagnosis was later confirmed at the necropsy. Favorite places for the edema to develop are in the loose areolar tissue about the eyes, about the genitals, about the ankles and legs, in the abdomen, and in the lungs. Edema and diminished urine are said to hold a reverse relation to each other; as one increases the other diminishes. That is, when the dropsy is greatest the amount of urine passed is the least. When the dropsy begins to disappear, the quantity of urine increases.

In acute nephritis the urine may be entirely suppressed. As a rule, it is much diminished, of high color, and with increased specific gravity. Frequent micturition is a leading symptom. On examination it is found to contain a large percentage of albumin, more, I believe, than in any other form of the disease. On very rare occasions albumin may be absent. The quantity of urea and of phosphates is diminished. The urine is clouded and deposits a heavy sediment. Under the microscope this is found to be composed of epithelium, casts, and blood. In acute Bright's epithelial and blood casts predominate; hyaline and other forms are not so prevalent. Casts are composed of débris deposited and compressed in the tubules. Some authorities claim that those passed with the urine come only from the straight tubules, casts found in the convoluted tubules not being able to get through the narrower straight ones. Coats, on the other hand, says that the casts are more or less compressible and flexible, and that those formed in the convoluted tubules can accommodate themselves to the smaller diameter of the straight tubules and thus pass through. Leucocytes and red blood corpuscles are found in abundance in the sediment. In fact the heightened color of the urine is due to the quantity of blood contained.

Eye changes are rarely present in acute Bright's disease. Sometimes sudden blindness develops. This usually disappears in a few hours. No retinal changes are found to account for it. The symptom is probably of central origin and may be due to uremic poisoning or to the pressure of fluid in the brain.

Acute Bright's disease may announce itself in the form of convulsions. This is especially true of the nephritis of pregnancy and of scarlatina. If careful watch is kept over the kidneys during gestation and during the progress of infectious diseases, this accident will not be so apt to take the attending physician unawares, for analysis will nearly always show that trouble is brewing. The convulsions of pregnancy are nearly always due to nephritis. These are known as "eclampsia." According to Lusk this occurs about once in every five hundred pregnancies. Convulsions, coma, and other profound nervous disturbances are probably due to uremia. Except as just noted uremia is not so common in acute as in chronic Bright's disease.

Complications: The complications of acute Bright's disease other than those enumerated in the symptomatology are not numerous. Bronchitis is nearly always present. In fact it is usually classed as a symptom. Pericarditis, pleuritis, pulmonary congestion, pneumonia, may occasionally develop.

Diagnosis: In typical cases the diagnosis is easy. The symptoms are rapid pulse, increased respiration, dry skin, edema, frequent micturition, suppressed or diminished urine. If any urine is passed it is found to contain albumin, casts, and blood. If there is a history of convalescence from scarlet fever or other infectious disease, or of exposure, the diagnosis is certain.

Sometimes an acute exacerbation of a chronic nephritis will give the same combination of symptoms. Then, without the previous history of the case, it is impossible to distinguish it from the acute form. I have had one such case in hospital practice. A woman, aged fifty-two years, came in with a history of exposure followed by all the classical symptoms of acute Bright's. The quantity of urine passed voluntarily and by catheter varied slightly from day to day, but it averaged less than half an ounce for three weeks, when she died. At

the necropsy the kidneys were found to be of the typical contracted kind. They had evidently been a long time in developing. The case was one of an acute exacerbation of a chronic condition.

Acute tuberculosis has sometimes been confounded with acute nephritis. The history of the case is of value in differentiating the two diseases. In the former there is extreme emaciation and lack of edema. The two diseases may coexist.

Acute Bright's disease may have to be distinguished from the acute congestion of inflammatory disease. In nephritis there is abundance of albumin and of casts, in congestion only a trace. If nephritis is induced by acute inflammatory disease, it follows that disease, and symptoms of the kidney affection thereafter cease to bear any relation to it. If acute congestion of the kidneys accompanies an infectious disease, the kidney symptoms increase as the original disease grows worse, and decrease and disappear when convalescence is established.

Suppurative nephritis is differentiated from Bright's disease by the history. It originates from external violence, by metastasis from suppuration in some other part of the body, by irritation from calculi. Suppurative nephritis is usually accompanied by pain and swelling over the region of the diseased kidney, and affects but one. In Bright's disease enlargement can rarely be demonstrated and both kidneys are alike affected.

There is a transient albuminuria that is found after eating certain things, after violent or exhausting exercise, and sometimes in adolescence. Cases of this nature are classed as cases of physiological albuminuria. Millard, in the last edition of his book, denied that there was any such thing as physiological albuminuria. He claimed that he never had found albumin except in diseased kidneys. While not quite willing to deny the existence of physiological albuminuria myself, yet I believe that apparently healthy individuals whose urine occasionally contains albumin need close watching. They should once in a while be thoroughly examined for other evidences of Bright's disease.

It is well known that albuminuria is a frequent symptom in pregnancy. It may be due to congestion of the kidneys caused by pressure, it may be due to acute nephritis, it may be due to

chronic nephritis. Lusk makes the statement that eclampsia is more apt to occur in patients who have developed the albumin during pregnancy than in those who were suffering from well-marked nephritis beforehand. Nevertheless, he advises against pregnancy in subjects of Bright's disease. The albumin of pregnancy appears in the later months and is associated with hyaline and granular casts. These symptoms usually disappear after labor. When the first symptom noticed is dyspnea, the lungs will, of course, be examined. If edema is present, examination of the urine will show whether or not nephritis is also present.

Convulsions or coma may first call attention to the disease. If due to uremia, these conditions are usually preceded by a diminution of urine. Other premonitory signs are vertigo, headache, dulness of the intellect, impaired hearing, and blurred vision. There are also apt to be nausea and vomiting. When these symptoms are present, careful analysis of the urine is in order.

If the patient is seen for the first time in convulsions or in a state of coma, but without data on which to base a clinical history, the differential diagnosis is almost impossible. Subsequent developments will help very materially to clear up the case. If the coma lasts for any length of time, the patient must be catheterized and an analysis made of the urine. The importance of this is obvious.

Uremic convulsions are epileptiform in character and usually pass into coma. The coma is profound and there is stertorous breathing. The temperature does not rise, the pulse is rapid, the skin is dry. In true epilepsy the convulsion is followed by a condition of stupor. If roused, the patient's mind is dazed. If not roused, the patient usually falls asleep and sleeps quietly for a time.

After cerebral hemorrhage the temperature rises and the pulse tends to go slower. Some form of paralysis is produced; this may be detected even when the patient is unconscious. The pupils are apt to be uneven. If convulsions are present, they follow and do not precede the coma.

Ordinarily syncope is too transient a symptom to be confounded with uremic coma.

In coma produced by poisoning, unless the dose is immediately fatal, the development of the symptoms is gradual. Irritant poisons disfigure the lips and fauces; this should lead to their detection. Narcotic poisons cannot always be distinguished from uremia.

In unconsciousness from the effects of heat there is a better chance for differentiation at the beginning, because it occurs only in hot weather or in hot places. The skin is dry, the temperature is very high. If the patient is a victim of sunstroke, the pulse will be full and bounding and the face red and congested. If the case is one of heat prostration, the pulse is very weak and rapid and the face excessively pale.

In surgical unconsciousness, concussion of the brain, fracture of the skull, and shock, the testimony of the bystanders is usually available.

Prognosis: The prognosis of acute Bright's disease is usually good. The majority of cases get well. If the acute symptoms subside, the edema disappears, the albumin becomes less, and the quantity of urine passed increases, the patient is improving. The edema disappears long before the albumin does.

Ordinarily acute nephritis lasts from two to six weeks. If the symptoms persist for two months or more the disease is becoming chronic. This is not a common course for it to take. From one-quarter to one-third of the victims die. The patient may die in a few days in coma or convulsions, or he may live for some time and eventually die from exhaustion or from some complication, as edema of the lungs and so on. Death may be caused by extension of the inflammation to other internal organs.

Treatment: The patient ill of acute Bright's disease must be put to bed and kept there until all acute symptoms have subsided. The room must be kept warm and at an even temperature day and night. The patient must be protected from any possible danger of exposure to draughts of air or other chilling influences.

Milk is the best food. To make it more palatable it may be flavored with coffee, salt, or soda water. There are persons who will be upset by milk. Thoroughly shaking the milk before

use will make it more digestible. Diluting it with lime water or carbonated water has the same effect. Some patients will take more kindly to buttermilk or to kumyss than to plain milk. Gruel may be sometimes used to advantage.

The use of tea, coffee, alcohol, and spiced foods must be stopped until after the patient is thoroughly convalescent. In exceptional cases very dilute whiskey and water may be given if the patient is excessively weak. The drinking of large quantities of pure water—several quarts daily—is essential. Lemon juice added to the water is very beneficial and may be taken *ad libitum*. Water holding in solution benzoate of sodium or citrate of potassium is used by some. My own preference is for a pure spring or distilled water with the juice of half a dozen to a dozen and a half lemons a day. This acts not only as a diuretic, flushing out the kidneys, but also has a direct effect on the kidneys, soothing the inflammation and diminishing the amount of albumin excreted.

As the acute symptoms subside and convalescence is established, meat broths, soft-boiled eggs, and fruits may be added to the bill of fare. A full diet of the character usual to the patient will have to be adopted slowly and with caution, all the time closely watching and noting the slightest changes in the urinary secretion.

The failure of the kidneys to excrete the usual quantity of urine, and the stoppage of the respiratory action of the skin as evidenced by its dryness, together cause the retention in the system of a lot of excrementitious matters that would otherwise be got rid of. To help eliminate this material as far as possible, diaphoretic treatment is resorted to. The best method is the application of heat in various forms.

The patient may be wrapped in dry hot blankets and given hot drinks. This method of diaphoresis can be employed with almost every patient. A more effective way, but one that is dangerous in cases with heart lesions, is to give the patient a hot-air bath. This can be done by completely covering the patient, except the head, with blankets, in such a way as to form a sort of air chamber, and devising some apparatus to heat the air underneath. The hot-air bath ought to last no longer than

twenty minutes. Care must be used lest the patient take cold; a rub-down afterwards with alcohol and water will help to prevent that.

The hot pack is another method that lends itself to quite general application. This consists in wrapping the patient in a sheet wrung out in hot water and wrapped round about with blankets. This hot pack may be kept up for from a half to one hour. Finally the hot-water bath may be utilized. This may last for half an hour. In utilizing any one of the methods described above, close observation of the patient will have to be kept up to guard against exhaustion or distress of the vascular system. It is an advisable procedure to rub the patient thoroughly with dilute alcohol both before and after either the hot-air or the hot-water treatment.

Pain over the kidneys may sometimes be relieved by dry cupping. Poultices do not seem to do so well.

If the bowels are constipated and the urine is scanty, it is claimed that a good deal of the excrementitious matter may be eliminated by means of cathartics. For a mild cathartic I know nothing better than Rubinat water. Senna or gamboge may be used. A more popular remedy is calomel. This may be given in eighth to quarter grain doses, repeated every hour or so until free catharsis occurs.

If uremic eclampsia or coma is impending the treatment by means of hot air or hot water, as outlined above, must be resorted to. Active depletion may be used. This is usually done by the use of pilocarpine hypodermatically, a quarter to a sixth of a grain at a dose. This relieves the dropsy as well as the uremia. Other drugs used in this way are jalap, drop doses of elaterium, and croton oil.

Loomis speaks very strongly in favor of the use of morphine hypodermatically in uremic convulsions. He claims that it not only quiets the patient, but that it has a beneficial effect on the kidneys as well.

Inhalation of chloroform is a frequently used method for controlling uremic convulsions. It has no curative effect, but it serves as a temporary relief until other means can be employed.

In uremic delirium my only success has been with *hyoscyamus*. I have seen that put the patient to sleep. After a few hours he wakened in a normal state of mind.

In eclampsia Lusk recommends the use of *veratrum viride* in doses of fifteen drops of the tincture to a teaspoonful of water, repeated in a half-hour if necessary. If beneficial in eclampsia there is no reason why it should not be of service in any case of uremic convulsions. I prefer a smaller dose.

For the lesser manifestations of uremia other methods are of service. In uremic headache I use *glonoin*. Bartholow and others mention its use. In the third to the sixth potency it has repeatedly relieved this condition.

Belladonna is another remedy that may be called for in this condition.

For the nausea sometimes found in slight uremia, bits of cracked ice held in the mouth frequently do much to relieve.

The edema of nephritis will often respond promptly to treatment. My favorite drug, if edema is general, is infusion of *apocynum*. Ten or fifteen drops may be given every hour. The quantity of urine passed is increased, the edema is held in check or reduced, and the patient feels generally stronger.

Another remedy is *apis mellifica*. I have verified it many times in practice.

Another remedy that may fit the dropsical symptoms, and is good for the anemia and debility of Bright's disease, is *arsenic*. I sometimes use Fowler's solution, and push it until the physiological effects are produced. *Arsenic* in potency may also be used.

When the edema becomes so great as to threaten to break through the skin, and medicinal and hygienic measures have no effect on it, it is necessary to resort to operative measures. The skin of the legs may be punctured under strictly antiseptic precautions, if the edema is great there. In ascites tapping the abdominal cavity and drawing off the fluid is sometimes necessary. Or it may be necessary to tap the chest to relieve the pressure there.

Aconite in minute doses frequently repeated will be found of service in reducing the hyperemia of acute Bright's disease.

Cantharides has a beneficent action on the kidney of acute nephritis. It is best given in drop doses of the tincture. It also seems to diminish the dropsy.

Later on, when the dropsy has gone down but albumin still remains, *mercurius corrosivus* will be found to be of service.

If the gastro-intestinal disturbances are marked, if there is much flatulence with scanty urine, *terebinth* will help very much.

Cactus grandiflorus is the remedy for a lesser degree of flatulence causing palpitation of the heart.

The anemia of acute Bright's is treated with the indicated remedy. Inhalations of oxygen are also of value for this symptom; the oxygen also acts on the blood in such a way as to cause a diminution of the amount of albumin passed.

CHRONIC BRIGHT'S DISEASE.

Synonym: Chronic Nephritis.

Etiology: Many cases of chronic Bright's disease are of insidious onset and are first discovered by accident. Cases of this nature cannot always be accounted for. Occasionally the acute disease runs into a chronic form.

Diabetes mellitus causes a few cases. Malaria is held by some observers to be an important factor in the production of chronic Bright's disease. Heredity is a predisposing cause. It is common for a number of cases to occur in the same family. Grief and worry undoubtedly cause chronic nephritis in some subjects. Lead is another potent cause of chronic Bright's. Workers in industries in which lead is used are exceedingly liable to develop the contracted form of kidney. In England gout causes many cases. In the United States gout is an unimportant factor. Habitual overeating may cause it. Some cases are due to the irritation of impacted calculi. Others are caused by the upward extension of cystitis or inflammation of other of the urinary organs.

Exposure is probably the most common causative factor.

Chronic Bright's disease is one of temperate climates. The varying changes in temperature are held to account for many

cases. Persons who are exposed to sudden changes of temperature in their work are prone to the disease. It is most common in the poorer classes among those who are exposed to hardship and privation. Habitual users of alcoholic liquors are frequent subjects of chronic Bright's disease. Many observers believe that the alcohol itself is responsible. Dickinson, however, considers the subject at length and combats this theory. It is well known that the habitual drinker is less able than others to withstand or resist disease. He is also exceedingly liable to expose himself and thus to produce nephritis. Experimenters have never been able to produce nephritis by the use of alcohol in animals. Tuberculosis causes some cases. Syphilis may produce the disease, especially the contracted kidney.

From one-third to one-half more men than women are affected. Roberts holds that this disproportion is not so great during the child-bearing period, when the sexes are more evenly attacked. He asserts that this is a proof that pregnancy is a real causative factor.

The disease is rare in infancy and in extreme old age. It is most common during late middle life.

Morbid Anatomy and Pathology: In describing the morbid anatomy of chronic nephritis I shall describe two forms, parenchymatous and interstitial. I must again call attention to the fact that frequently these two forms are clinically indistinguishable. There are typical cases that may be differentiated before death, but there are more that are on the border line and cannot. In all forms of chronic Bright's disease both the parenchymatous and the interstitial tissues are affected. The varying anatomical divisions merely imply that one part of the kidney structure is more damaged than is another.

Chronic parenchymatous nephritis, otherwise known as chronic tubular nephritis, chronic diffuse nephritis, with exudation (Delafield and Prudden), chronic croupous nephritis (Millard), chronic desquamative nephritis, chronic catarrhal nephritis, the large white kidney, and, later on in the disease, the small white kindney.

The kidney of chronic parenchymatous nephritis differs but

little from that of the acute form. The changes are merely slower in taking place. Usually the organ will be found to be enlarged. This is called the large white kidney. It may be as much as three times its normal size. The capsule is non-adherent. The surface is smooth and pale. There may be patches of congestion. On section it is found that the increase in bulk is mainly in the cortex. The cones are less enlarged and retain their original color.

This form of kidney must not be confounded with the amyloid, lardaceous, or waxy kidney, which is also large, white, and smooth. This latter is due to a general amyloid degeneration, and is always found associated with waxy liver and waxy spleen. It is never found except after a wasting disease. An application of a watery solution of iodine simply discolors the parenchymatous kidney; with the amyloid kidney it gives a mahogany color to the waxy part and a yellowish tinge to the remainder. Amyloid degeneration may occur or coexist with any form of nephritis.

In chronic parenchymatous nephritis the secreting portion of the kidney is specially involved, the interstitial portion less so. The diseased condition is general or diffuse—that is, the tubular and the inter-tubular tissues are both affected. There is cloudy swelling of the epithelium, which later loosens and becomes detached. The tubules are distorted and obstructed with the débris until they look varicose. Fatty degeneration of the epithelium occurs, showing as yellowish streaks and giving the organ its light color. The packing of débris into the tubules may be so extensive as to obscure the interstitial tissue. The blood-vessels may be crushed. Blood and casts are found in the tubules. Later, as the case progresses there develops a new formation of interstitial tissue with an infiltration of leucocytes. This new connective tissue extends out to the capsule, which may become thickened. In time this may distort the organ. There are slow contraction and granulation. The shrinking of the new formation causes the cortex to sink in, and we have as a result the small white kidney, which somewhat resembles the contracted or chronic interstitial kidney. The latter, how-

ever, begins its change at the surface, whereas the parenchymatous form begins its change in the interior. Even when firmly contracted the small white kidney is smooth, although it may have an irregular surface. It is soft and flabby and the capsule is non-adherent. The cortex may become thin and the glomeruli sclerosed. The organ retains its original buff color. Much of the cortical excess in the deeper parts remains.

Chronic interstitial nephritis: Chronic diffuse nephritis without exudation (Delafield and Prudden), contracted kidney, granular kidney, granular atrophy of the kidney, cirrhosis of the kidney, renal sclerosis, gouty kidney; these are some of the names given to inflammation of the connective tissue of the kidneys. It must not be forgotten that the parenchyma of the organ is also involved.

This form of kidney is generally recognized as chronic from the beginning. It is usually much reduced in size, hence its name, contracted kidney. The capsule is thickened and firmly adherent, its removal tearing off portions of the kidney substance. When the capsule has been stripped off the surface, the kidney is seen to be covered with fine granulations. These are of a lighter color at the tips and darker at the bases. Small cysts are visible and characteristic. There is an excess of fat about the organ. On section the kidney substance is found to be tough. All parts of the organ show gray radiating striæ which are found to be new formations of connective tissue. The cortex is thinned and irregularly defined. The Malpighian bodies are many of them obliterated. The medullary rays are obscured.

There is a formation of round cells in the cortical part of the kidney so that a kind of granular tissue is found. From this the new connective tissue is developed and gives a great increase in the interstitial substance. This change first occurs about the Malpighian bodies. It encroaches on the glomerular walls, these in turn become thickened and encroach on the glomeruli themselves until there is nothing there but a mass of connective tissue. The tubules are also encroached upon and obliterated. There is cloudy swelling of the tubular epithelium

followed by waxy or fatty degeneration. The epithelium is shed and forms casts as in the kidney of parenchymatous nephritis.

In the interstitial kidney cysts are formed by the squeezing and crowding of the tufts and tubules by the new tissue, and the consequent retention of the urinary contents in these little sacs. The blood-vessels of this form of kidney are thickened. There is an hypertrophy of the long and circular fibres of the muscular coats. In the glomeruli the blood-vessels may be destroyed by the advancing sclerosis. Thoma has found that the outflow of blood under a given pressure is much lessened in the contracted kidney.

Vascular changes: In chronic Bright's disease of the kidneys changes of the vascular system take place in parts of the body other than the kidneys. Roberts divides the vascular changes into three classes: (1) Simple hypertrophy of the left ventricle without valvular lesion. Bright was the first one to point out this class in his original communication. (2) Valvular defects coexist with Bright's disease. The valvular lesion is usually secondary, although it may exist separately. (3) Chronic nephritis may be secondary to the heart lesion.

There is frequently an hypertrophy of the heart. This is most often found with the contracted kidney. It may accompany parenchymatous nephritis. The general tendency of belief is that the obstruction to the circulation in the kidneys causes an increased pressure in the arteries. The heart and arteries, therefore, have constantly to exert more pressure to force the blood through. This in turn causes hypertrophy of the left ventricle, and a thickening and hypertrophy of the muscular coats of the arteries result.

Dickinson says cardio-vascular changes are constant, especially in the contracted form of kidney. Arterio-capillary fibrosis, or arterio-sclerosis, as it is also called, is a fairly constant accompaniment of chronic interstitial nephritis. When this disease (arterio-sclerosis) was first described by Gull and Sutton, they advanced the proposition that the disease was general, and that the contracted kidney was merely the result of a general process. This idea has not received the unqualified sup-

port of other observers. Osler says he believes that arteriosclerosis precedes the contracted kidney oftener than is generally supposed. He says the disease of the arteries may follow the kidney lesion.

Ocular changes: The only other constant change found in chronic Bright's disease is in the eye. The patient complains of impaired vision. On examination the eyes are found to be affected. There is a haziness of the retina and optic disc. The optic papillæ are swollen and have a clouded and indistinct outline. Extravasations of blood are seen. The veins are tortuous. Numerous white spots are seen, varying in size and scattered about the neighborhood of the optic disc. These are found to be foci of fatty degeneration.

A rarer form of eye trouble is sudden and transient blindness, most often found in the acute nephritis of pregnancy or of scarlet fever. This must be of central origin, as no retinal changes are found.

Symptoms: Chronic nephritis is an affection that may last from one month to many years. A few patients recover, the majority eventually die. The onset is most often insidious and the disease is usually discovered by accident. Patients really ill of Bright's disease will present themselves with symptoms pointing to the nervous, vascular, or digestive systems so often that it is always wise to resort to a urinary analysis before making a positive diagnosis. It is my rule to examine the urine of all patients in private practice no matter what the ailment.

In examining the urine too much emphasis cannot be laid on the necessity, (1) of knowing the total quantity of urine passed in twenty-four hours, (2) the specimen examined must be a portion of the entire mixture, (3) the specific gravity must be noted. Other findings are of lesser importance. A diseased kidney will cause a change in the total quantity of urine and in the specific gravity sooner or later and more or less regularly. Conversely, a urine of constantly normal quantity and of constantly normal specific gravity proves the kidneys to be normal.

In chronic nephritis the urine varies in amount from day to day. In the parenchymatous form it is usually diminished in

quantity, is dark in color, of slightly increased specific gravity, and contains albumin. It resembles the urine of acute nephritis. In typical cases of the interstitial variety the urine is increased in quantity and is of low specific gravity; this may be as little as 1.010. Albumin may be present only to a slight extent, or it may even be absent for a time. As the disease draws to a close, however, albumin rarely is absent altogether. In all forms of chronic nephritis, toward the end of life, the quantity of urine is diminished. In one case of an acute exacerbation of chronic interstitial nephritis under my care during the summer of 1898, the patient passed an average of less than half an ounce of urine for each twenty-four hours for three weeks before death. Micturition is more frequent than is normal. The patient, as a rule, is obliged to pass urine during the night. This, indeed, may be the first symptom for which the patient seeks relief. The urine is very apt to irritate the parts. Besides the appearance of albumin in the urine, as noted above, in all forms of chronic Bright's disease there is diminished excretion of urea and of the mineral salts. This was first noted by Bostock, who made the chemical analyses of the urine in Bright's original cases. Various observers have since that time added to the knowledge extant concerning the chemical constituents of the urine of chronic nephritis. When the urine is scanty and the specific gravity increased we may have a larger percentage of urea, but the total quantity for the twenty-four hours will fall below normal. When the urine is increased in quantity and the specific gravity is low, the percentage of urea is low, but the total amount for the twenty-four hours may not be greatly lessened. Toward the end the excretion of urea is greatly reduced. I have seen it as low as eight grains in the twenty-four hours.

Purdy has called attention to the diminution of the phosphates in chronic Bright's disease, especially in the urine from the contracted kidney. Laidlaw has gone so far as to say "that a deficient excretion of phosphates in the urine is the only constant urinary symptom of chronic nephritis with interstitial changes; it is more constant than albumin, more constant than

the low specific gravity or increased quantity of the urine." Laidlaw suggests for this symptom the name of "oligophosphaturia."

In the sediment granular and fatty casts predominate. Epithelium from the bladder and kidneys is found. Blood corpuscles and pus cells are also frequently found. The sediment in chronic interstitial nephritis is less than that in the parenchymatous form.

A good many cases of chronic Bright's disease discover themselves first through a puffiness about the eyes, or through edema in some other part of the body. Occasionally cases progress to the end without edema. This does not often happen. As mentioned when discussing the edema of acute nephritis, edema will usually be found if carefully searched for. The chronic parenchymatous kidney is the one that produces dropsy to the greatest extent. It develops slowly and constantly increases, until toward the end of life it may cause dyspnea by edema of the lungs, or by pressure in the pleural or abdominal cavities. Ascites sometimes develops to an astonishing extent. I have several times removed fifty and sixty ounces of fluid from the abdomen.

The dropsical fluid accumulates not only in the cavity of the abdomen, but there may also be an effusion into the walls. The dropsy may become very great, and yet, under appropriate treatment, almost entirely disappear. If the case happens to be one of the rare ones that end in recovery, the edema may cause no further trouble. As a rule, however, the dropsy recurs again and again until the point is reached where medicinal treatment has no further effect. Then the only resort is to palliative measures until death closes the scene. As a rule, it diminishes at the end. The favorite places for the appearance of edema have been indicated under the symptomatology of the acute form of the disease. It begins in the loose areolar tissue about the orbits and genitals. Flint says a good place to detect slight edema is over the sternum. When the heart's action fails, the dropsy appears in dependent portions of the body. The labia and scrotum may become swollen to the size of a

man's head. The face may lose all semblance of a natural appearance and look more like a balloon than anything else.

The third of the great trio of Brightic symptoms is uremia. This may manifest itself as a dull throbbing headache—a very frequent symptom in chronic interstitial nephritis. In my experience the common form of headache is occipital. Other observers have noted it more frequently in the frontal region. We may get cerebral nausea and vomiting as a result of the uremic poisoning. Uremic vomiting is said to have an ammoniacal odor. If the vomited matter is acid it is said to evolve ammonia on the addition of caustic potash. The patient may be drowsy or stupid, or he may be talkative and start and twitch. Uremic coma and convulsions are comparatively frequent. Either condition may be the first to call attention to the disease. Delirium is rare. I have seen one case of it. It appeared suddenly and would disappear after a quiet sleep induced by *hyoscyamus*. This occurred a number of times during the last few weeks of life.

If the brain symptoms are slight, there may be blurred vision, light flashes, and other disturbances of the sense of sight. Cases have been reported of toxic dyspnea due to uremia. My own experience has been that dyspnea is always associated either with edema of the lungs or with pressure from hydrothorax or ascites.

Vascular changes are marked in chronic nephritis. Hypertrophy of the heart or valvular lesion is associated with nearly all cases. The pulse is characteristic of the vascular condition it is associated with. In parenchymatous nephritis there is apt to be a weakened pulse with increased rate. Valvular lesions are often found with this form of kidney. In the interstitial form of the disease the pulse is full and strong. Owing to the sclerotic changes that take place in the walls of the arteries the pulse continues to feel hard to the end. The blood pressure is much increased. The heart sounds are strong. The second sound is accentuated. The apex is low and to the left. Palpitation is common.

Anemia accompanies chronic Bright's disease. During tran-

sient disturbances of the circulatory system cyanosis may develop. In far advanced cases, when the heart is unequal to the continued strain, cyanosis of the dependent parts of the body is common. I have seen but one case in which the cyanosis was general for any length of time. The patient was waterlogged and cyanosed when brought to the hospital. He lived about a week in that condition. Cyanosis and anasarca were more marked than in any other case that has passed under my observation. Another case in my service at the Flower Hospital in 1914 presented marked cyanosis, but the edema, though general, was not very great. Angina pectoris is occasionally present as a symptom. I have had one such case. Purpura may also occur. Cerebral hemorrhage is quite a common symptom, especially in chronic interstitial nephritis.

In the lungs bronchitis is commonly associated with the chronic as well as with the acute Bright's disease. There may also be cough and dyspnea. The dyspnea may be due to uremic poisoning or to pressure of the dropsical fluid. Pleurisy is often found with chronic Bright's disease. It occurs most often in connection with a heart lesion. The symptoms found in the gastro-intestinal canal are numerous. They often cause a mistake in diagnosis. I have seen nausea and vomiting occur often in chronic Bright's disease. They may be due to uremia, or they may be due to actual gastric disease. Burning pains in the stomach are sometimes complained of. One patient of mine complained of soreness across the abdomen. Diarrhea often occurs, especially in dropsical subjects. This is sometimes due to the swollen condition of the intestines which causes them to act abnormally. It may be accompanied by cramps. There may be a great deal of flatulence.

The symptoms found in the nervous system are numerous and varied. Some are due to the deteriorated blood, others are due to uremic poisoning. Headache frequently occurs. Sleeplessness is another distressing symptom, especially toward the end, when it is apt to be aggravated by dyspnea. The patient may be drowsy, dull, or in a profound stupor. This latter is due to uremia. Uremic convulsions are similar in their manifestations to epilepsy and are quite common. Coma also is quite

common. These two symptoms, convulsions and coma, are often preceded by amblyopia. There may be lack of coördination between the eyes. The patient sees double. They may be preceded by vertigo, numbness of the hands and feet, cramps in the muscles. This last group may for a long time be the only manifestation of uremia.

One patient of mine first consulted me because of this peculiar numbness. Later she complained of "sinking spells," which she brooded over. Then she got the notion that her family was neglecting her. Noises annoyed her. Music, which she had formerly been very fond of, caused her to weep. In a word, the condition simulated one of melancholia. Another patient, already referred to, developed violent delirium. He believed a conspiracy had been formed by his wife and physician to kill him and to get his property away from him. It required considerable diplomacy to manage him. I hope never to see another case like it.

One of the diagnostic signs of chronic nephritis is albuminuric retinitis. A patient will complain of blurred vision. An ophthalmoscopic examination will show the characteristic changes. These consist in the appearance of an indefiniteness in the outlines of the optic papillæ. The retina is puffy-looking. The veins are engorged and tortuous. Little white spots of different size are scattered about the optic disc.

Another eye symptom that has been mentioned several times is sudden blindness. This augurs ill. It sometimes precedes the onset of acute uremia. The blindness is only temporary in any case. No anatomical changes have been discovered in association with it.

The temperature of chronic Bright's disease is most frequently below normal a very little. During the final weeks it may vary from 99° to 100° F. The appetite is lost. The skin is dry and pale. There may be intense itching, accompanied or not by a deposition of urea on the surface of the body As life draws to a close the patient exhibits all the signs of profound disorganization. There are twitchings and picking at the bed-clothes. The patient slides to the foot of the bed. Urine and feces are passed involuntarily. The patient is unconscious.

Complications: An acute nephritis may develop in the course of a chronic condition. This may present such a classic picture of the acute disease that the chronic condition remains undiscovered until the necropsy. Chronic nephritis is subject to all the complications of the acute disease. Hemorrhages from the mucous membranes may occur, as epistaxis and hemoptysis. Alternate constipation and diarrhea is more or less common; this may be due to catarrhal conditions, to uremia, or to edema. Secondary inflammations of the pleura, lungs, pericardium, peritoneum, and even of the integument may complicate the chronic nephritis. Numerous nervous, digestive, and vascular changes are associated with it; most of them have been noted in the symptomatology. Chronic Bright's disease and pulmonary phthisis are often found associated.

Diagnosis: The diagnosis of chronic Bright's disease, early in its history, is frequently overlooked. The onset is insidious, and the disease may progress for months and years before it is even suspected. In the majority of cases the discovery of the existence of chronic nephritis is made by accident, many such being found on examining candidates for life insurance. It is quite a common affection in this country. It is important that chronic Bright's disease be discovered early, for if promptly and properly treated much can be done to prolong life. It is wise, therefore, to make analysis of the urine a routine measure in all cases, whether nephritis is or is not suspected. For several years I have made it my rule to examine the urine of every new patient and to keep a record of the result on file for future reference. A number of times I have in this way discovered that a patient was suffering from chronic Bright's disease, and in each instance I believe the patient has been the gainer.

Typical cases of chronic parenchymatous nephritis, in which the urine is scanty and contains albumin, and there is edema, are not difficult to diagnose. Nor are cases with a history of profuse urine of low specific gravity, slight edema, accentuated second sound of the heart, indicating the contracted kidney, at all confusing.

Acute exacerbations of a chronic nephritis can be mistaken

only for the acute disease. Without a history these cases cannot be differentiated. This makes no difference in the treatment, as both groups of cases require the same. It does make a difference in the prognosis, because the rule is for the acute case to result in recovery and for the chronic case to be fatal.

The varying symptoms of chronic nephritis may simulate those of any of the conditions already noted in discussing the diagnosis of acute nephritis. That section must be read before going on to the consideration of the other diseases that may be mistaken for chronic Bright's. The urine must be carefully studied in all doubtful cases, not once, but many times. Even then a positive diagnosis can be made only by carefully weighing all the evidence.

Chronic indigestion is often treated without the fact being detected that it is caused by Bright's disease. Patients with gastro-intestinal troubles should always be carefully examined for further evidence of nephritis, as this is one of the characteristic methods of its development.

Other patients will exhibit symptoms of neurasthenia, of melancholia, of neuralgia, of rheumatism. All such patients must be watched for the development of Brightic symptoms.

Anemia is one of the most prominent symptoms of nephritis. Any case presenting anemia as a symptom needs to be watched.

It is unnecessary, perhaps, to particularize further. A patient presenting with constant disturbance of the digestive system, of the nervous system, of the vascular system, or of the respiratory system, should put the physician on his guard. Groups of symptoms of any of these parts, especially if of obscure origin, should arouse the suspicions of the medical adviser. Even if changes cannot at first be found in the urine, that does not imply that they may not later develop. If this fact is always borne in mind and acted on, mistakes will not be so apt to occur. Finally, some cases will escape the most careful diagnostician until help is impossible.

Prognosis: The prognosis of chronic Bright's disease is bad. Nearly all patients die of it. The disease may last from several months to ten years or more. I had one case under constant observation for twelve years before she died. A few ob-

servers have reported cases of cure. I have never seen or personally known of an actual cure. I have seen many patients much benefited by treatment and kept alive for varying lengths of time. Acute exacerbations may be met and subdued with appropriate treatment. But a time will come in nearly every case when that becomes no longer possible.

Death may occur from any of the causes that effect a fatal termination in acute Bright's disease. Cerebral apoplexy is the cause of death in quite a percentage of cases.

Treatment: When acute symptoms develop during chronic Bright's disease, the patient has to be treated in all respects as if it were an acute case.

If the patient is found to be suffering from chronic Bright's disease in a quiescent state—that is, if he is in fair general health—he must be cautioned against catching cold. It would be preferable to have him remove to a warm climate. Wherever he lives he ought, at all times, to wear wool next the skin —woollen underwear by day, woollen pajamas by night. Great care must be used to keep the skin functionating. For this purpose there is nothing so good as a sponge bath, followed by a good rub-down. This kind of bath is best taken with moderately cool water on rising in the morning. Every other day is often enough. One or two hot baths—tub baths—should be taken through the week, preferably at night just before retiring.

The diet of the chronic Brightic in apparent fair health is a mooted question. My opinion is in favor of a liberal diet. Red meat is permissible once a day. Chicken, eggs, most kinds of fish, vegetables, fruit, milk, all may be used. Fat, greasy foods, and rich, heavy pastries must be avoided. Tea and coffee may be used if desired. In the way of alcoholic liquors, I like my patients to stop their use altogether. If something of the sort must be used, claret or diluted whiskey—at the strongest one part whiskey to four of water—are the least harmful. I prohibit all malt liquors.

A few years ago Edebohls advocated surgical interference in chronic interstitial nephritis. He decapsulated the kidneys. In 1913 Lloyd reported 41 cures out of 121 cases.

If there is a history of syphilis, *iodide of potassium* will almost invariably benefit the patient.

Besides the remedies mentioned under the treatment of acute Bright's disease, there are one or two that need special notice here. *Mercurius corrosivus* was mentioned before. It is one of the best of remedies in chronic nephritis. It will diminish the amount of albumin and may cure the patient.

I have also found *plumbum* to be a good remedy, especially in the contracted kidney.

Arsenicum album 6x, continued over a long time is a very useful drug in chronic nephritis.

Phosphorus is indicated in cases of sudden onset with vomiting and gas. Spots appear before the eyes.

Toward the end of life, when the heart begins to fail, there are a number of remedies that may be of service. Infusion of *digitalis* is one of the most frequently used. It ranks high as a diuretic and as a heart stimulant. My own experience with it, however, I must confess to have been disappointing.

I like *caffeine*, 3x to 6x, better and have seen it do good work. The heart's action becomes stronger, the urine increases in quantity, and the patient is less nervous.

Strychnine is a good heart stimulant, but its use must not be continued for too long a time. If it is, the patient gets into a neurasthenic condition that is distressing. The bowel becomes paralyzed. It is a dangerous remedy to give for continued periods. It will make the patient worse if care is not used.

For emergency use in threatened heart failure, *camphor* in drop doses, repeated every five minutes, if necessary, is my first choice. It is one of the most prompt and diffusible of heart stimulants. For hypodermatic use at such times brandy or ether may be used.

One of the most distressing symptoms in advanced cases of Bright's disease is sleeplessnesss. *Morphine* is usually recommended. I have so often seen it excite instead of soothe the patient that I do not use it any more. If I use *opium* at all I prefer *codeine*. I give it in one-eighth of a grain doses. It is safer and does the work more effectively for me. The *bromides* and *chloral* I object to on general principles. If the sleeplessness is not caused by actual physical pain, but is more of a nervous phenomenon, I sometimes use *trional*. I give it in the

usual fifteen-grain dose. In such cases it has proven itself very satisfactory.

ALBUMINURIA AND UREMIA.

These two conditions, albuminuria and uremia, have been sufficiently discussed under Bright's disease. Practically always albuminuria, always if constant, is due to some form of nephritis. Uremia means nephritis, except in certain cases of diabetes.

AMYLOID KIDNEY.

Synonyms: Lardaceous or Waxy Kidney.

Etiology: Amyloid degeneration of the kidney is a part of a general process affecting other organs as well. It may follow suppuration in some part of the body; it may accompany syphilis, tuberculosis, leukemia, lead poisoning, or gout.

Morbid Anatomy and Pathology: The kidney is large and pale. The cut surface glistens. The glomeruli are distinct, the pyramids deep red in color. There may be a fatty degeneration. There may be spots of atrophy. A dilute solution of *iodine* changes the color of the waxy part to a deep red. The liver will also show amyloid degeneration.

Symptoms: There is an increase in the quantity of urine passed. It is clear and of low specific gravity. There is albumin. Hyaline and fatty casts are found, but are not numerous.

Dropsy often occurs. There is sometimes diarrhea.

With the above symptoms there is no increased arterial tension, no enlargement of the heart.

Further investigation will show a suppurating focus somewhere in the body, or else the patient will be suffering from some wasting disease or other of the conditions noted in the etiology. There will be found amyloid degeneration of the liver and other organs.

Diagnosis: The amyloid kidney is a part of a general process, the urinary symptoms are incidental. The diagnostic point to be remembered is that the patient has a polyuria with much albumin and few casts.

Prognosis: The prognosis is grave.

Treatment: The treatment must be directed first to the underlying disease. Special measures for the urinary condition must be purely symptomatic.

NEPHROLITHIASIS.

Synonyms: Stone or Gravel in the Kidney. Renal Calculus. Renal Colic.

Definition: Osler's definition is "The formation in the kidney or in its pelvis of concretions, by the deposition of certain of the solid constituents of the urine."

Etiology: Nephrolithiasis is due to faulty metabolism. Certain constitutions seem prone to it. Those of sedentary habits or of a gouty tendency seem most often affected. Men develop it more often than women.

Morbid Anatomy and Pathology: Various salts that normally are carried off in the urine are deposited in the kidneys. At first sand-like particles are thrown down and known as gravel. Later these particles may become larger from concretions deposited on the surface when they are called calculi. This increase may go on till the stone or stones become of considerable size. Different calculi are composed of different salts. The uric acid calculi, according to Osler, are most important. Then those of oxalate of lime. Third, phosphatic calculi. An occasional one is found composed of something else.

Symptoms: Nephrolithiasis may produce no symptoms. The particles may be so small that they are washed out with the urine without causing irritation. In others they may cause a dull ache in the back. In some cases again the concretions may cause no symptoms because they remain quiescent.

In still other cases calculi may become engaged in the ureters, or they may pass through the ureters with effort causing that most agonizing pain known as *renal colic.*

Renal colic is a symptom that develops suddenly. It is evidenced by acute pain in the region of one kidney. There is tenderness on pressure. The pain is so great that the patient cannot keep still, but seeks relief by constant change of position —a relief that cannot be found. I know from personal ex-

perience. The pain of renal colic is said to be the most severe that mankind is subject to! It may be localized in the region of the affected kidney; it may extend down the line of the ureter. In men it is sometimes referred to the inner side of the thigh and to the corresponding testicle. This last is given as one of the classic symptoms of renal colic. In my cases it has been an exceptional symptom. Occasionally the pain extends across the abdomen as though the whole peritoneum had become involved. The attack may last a few minutes or hours. Usually it comes and goes for hours.

Nausea is often present, sometimes even to vomiting. The patient gets in a profuse perspiration.

The stone may drop back into the kidney, or it may enter the bladder. After the attack is over there is often a soreness that persists for a day or two. There may be recurrent attacks for days with periods of rest in between.

A stone that finally finds its way into the bladder may cause much irritation there before it is gotten rid of.

Complications and Sequelæ: If the stone becomes lodged in the ureter there may be hydronephrosis, later there may be pus.

The irritation may cause bloody urine.

A stone that gets as far as the bladder may set up a cystitis.

Diagnosis: A typical case of renal colic is characteristic. That it is caused by stone can be inferred if the urine becomes diminished, high colored and of high specific gravity.

Renal pain may be caused by hemorrhagic infarct of the kidney. There is usually history of heart disease with hemorrhagic infarct. I have verified this form of renal colic at autopsy.

Gall stone colic may be hard to differentiate sometimes. The location of the pain is different in the two.

Prognosis: Usually good for a given attack. Recurrence of colic is always a possibility till the stone is passed. Even then there is always the possibility of a new formation and recurrence later. I have known fourteen years to elapse between attacks.

Treatment: Rest in bed as quietly as possible is the first thing necessary. Sometimes hot applications may afford a certain amount of relief.

Chloroform may be administered to dull the sensibilities.

Morphine is recommended but care must be exercised not to give too large a dose. It must not be forgotten that attacks of renal colic may end as suddenly as they begin. Morphine enough to mask the pain, immediately the pain is gone, might overpower the patient.

In 1894 I prescribed *calcarea carbonica* 200 for a case of renal colic. Since then I have used it many times with satisfaction. It is indicated by the pain and its location, by the associated symptoms of nausea and vomiting when they occur. I put the remedy in water and give it every five or ten minutes until the patient is relieved. This usually occurs within half an hour. Then at longer intervals for some days. *Calcarea carbonica* not only relieves the pain of renal colic, but I believe it stops the tendency to the formation of calculi.

Berberis, sepia and other remedies are recommended.

Arnica will relieve the soreness left after the acute pain has subsided.

Opium in potency may be necessary if constipation results.

HYDRONEPHROSIS.

Definition: Dilatation of the pelvis and calyx of the kidney, with atrophy of its substance, caused by the accumulation of non-purulent fluids the result of obstruction. (Osler.)

Etioloy: The condition is caused by a blocking of the ureters or other portion of the urinary tract by stricture, by tumor, by calculi, or by moveable kidney, causing a damming back of the urine. In some cases there is alternate damming and releasing. The pelvis becomes dilated, and the kidney substance atrophies.

Symptoms: There may be none. In other cases a palpable tumor may be the first evidence of trouble. This may change from time to time as the urine accumulates or is released. When the urine passed is scant the tumor enlarges. There may be pain, chills and vomiting. When the urine passed is plentiful the symptoms disappear.

The trouble is usually unilateral.

The heart is often hypertrophied.

Complications and Sequelæ: The kidney may rupture causing alarming symptoms. It may lead to uremia. It may cause pus kidney.

Diagnosis: Exploratory puncture may be necessary.

Prognosis: This is bad.

Treatment: Surgical.

PYELITIS.

Synonyms: Pyelonephritis. Pyonephrosis. Consecutive Nephritis. Suppuration of the Kidney.

Definition: Inflammation and suppuration of the kidney.

Etiology: It is due to pus organisms. It may come by way of the blood current, or it may be due to extension of disease of the lower part of the urinary tract. If by way of the blood current it is often part of a general septic condition. Foreign bodies in the kidney, as calculi, or clots of blood, may eventually cause suppuration. It may occur in the lying-in woman.

Bacteriology: Pus organisms, colon bacilli and tubercle bacilli have been found.

Morbid Anatomy and Pathology: The condition may occur in any part of the kidney. There is inflammation, then ulceration. The kidney structure may be affected, presenting minute abscesses, or the pelvis may be just a dilated sac filled with purulent fluid.

Symptoms: The symptoms may be slight. They may be masked by the primary disease. There is often a slight ache or tenderness over the region of the kidney. The urine is increased in quantity, of low specific gravity, acid in reaction. It may contain albumin. Under the microscope pus casts may be found. In cases due to calculi the urine may be bloody.

When the disease is the extension upward of a cystitis both kidneys are apt to be affected.

Diagnosis: The diagnosis must be made from the history of the case. It may be necessary to catheterize the ureters to make sure the pus comes from the kidneys and not from the bladder.

In some cases the x-ray may be of help.

Prognosis: The prognosis is bad.

Treatment: First, get at the cause if possible and eliminate that.

The patient should be encouraged to drink water to flush the kidneys. Warm baths will be of service.

Surgical treatment may be called for.

Urotropin, 10 grains, three times a day in water, may help to cleanse the kidneys and neutralize the urine.

Such remedies as *arsenicum album, hepar, echinacea* may be useful for the septic condition.

PERI-NEPHRITIC ABSCESS.

Definition: Suppuration in the neighborhood of the kidney.

Etiology: It may be due to trauma; to extension from the kidney; from the intestinal tract; from caries of the spine; from the pleura. It may be a sequel to the infections.

Morbid Anatomy and Pathology: The kidney is in a pus sac.

Symptoms: May be absent. There may be indefinite pain in the loins. The legs may be flexed when lying down to relieve this pain. When the abscess is large a swelling appears in the lumbar region. It finally points.

The patient usually shows evidence of pus by a septic temperature. He may have chills and sweats. He loses flesh and becomes weak. It hurts to walk.

Complications and Sequelæ: Pus is not usual in the urine. The abscess may rupture in the chest or in the abdomen.

Diagnosis: The location of the abscess will show it to be in the region of the kidney. The aspirating needle will reveal the pus. Absence of pus in the urine will show that the trouble is not in the kidney itself.

Prognosis: The prognosis is doubtful.

Treatment: Treatment is surgical.

FLOATING KIDNEY.

Synonyms: Movable kidney. Nephroptosis.

Definition: Floating kidney is one that can be palpated and is movable.

Etiology: Floating kidney is nearly always acquired. It is much more common in women than in men. The right kidney is more often at fault than the left, in the proportion of about six to one. Both kidneys are occasionally movable. The condition may be caused by tight lacing, by sudden physical strain, by frequent pregnancies, by relaxed abdominal walls, by the rapid loss of flesh.

Floating kidney is frequently a part of a general ptosis of the abdominal organs.

Morbid Anatomy and Pathology: The morbid anatomy consists in the abnormal position of the kidney.

Symptoms: In many cases there are no symptoms, the condition is discovered by accident. In others there is lumbar pain at times, and a pulling sensation. The patient is neurasthenic. She suffers more or less from indigestion.

In others again there are attacks of discomfort known as Dietl's crises; that is, the pedicle is supposed to get twisted in some way causing intense pain in the kidney, with nausea and vomiting; the patient has chills, fever and symptoms of collapse. In one patient it caused attacks of angio-neurotic edema.

The urine is scant at such times, and there may be an intermittent hydronephrosis.

Physical examination will reveal a tender, movable kidney. If the obstruction causes hydronephrosis the organ will be enlarged. Different examinations will find the kidney in different positions.

Diagnosis: The diagnosis is made from the physical examination. The possibility of gall stones must be thought of.

Treatment: Many patients are kept comfortable by wearing a specially prepared bandage which is applied when the patient is lying down or is in the knee-chest position.

Surgical interference may be necessary.

TUMORS OF THE KIDNEY.

Tumors of the kidney may be primary or secondary. They may reach a large size. There may be carcinoma, sarcoma or cysts.

Etiology: Tumors of the kidney occur more often in childhood. The left is more often affected than the right.

Morbid Anatomy and Pathology: The tumor may affect a part or all of the kidney and may be of great size. In cancer the destruction of tissue may cause severe hemorrhage.

Symptoms: The symptoms are usually indefinite until the tumor becomes big enough to be discovered. There is a dull pain in the lumbar region. As the tumor grows it causes symptoms by pressure and by displacing the diaphragm upward. The abdomen becomes large and distended. The veins become prominent. There is a rapid pulse. Later, in case of malignant growths, the patient develops the cancerous cachexia.

Physical examination shows a tumor that does not move with respiration. It can be moved with the examining hand. The colon covers it. Strümpell says that in girls there is an early growth of pubic and axillary hair.

The urine may remain normal. It may contain blood, blood clots, and casts.

Diagnosis: An abdominal tumor in children is suspicious. Kidney tumor does not move with respiration, it can be moved with the examining hand. Enlarged liver or tumor of the liver moves with respiration. Intestinal tumors, or stomach tumors, fall away from the back when the patient is in the knee-chest position. Abdominal aneurism remains stationary in all positions and is pulsating.

Prognosis: The prognosis of kidney tumor is bad.

Treatment: Surgery, as a rule, holds out the only hope.

TUBERCULOSIS OF THE GENITO-URINARY ORGANS.

Etiology: Practically always tuberculosis of the urinary organs is secondary to tuberculosis elsewhere. It rarely exists alone.

Morbid Anatomy and Pathology: Tuberculosis may attack the kidneys, bladder, prostate or testicles in men. It may attack the kidneys, bladder, uterus or ovaries in women. It may attack any of these organs first, and then extend to the others. Cheesy

nodules appear which break down and ulcerate. The disease may be unilateral or bilateral.

Symptoms: As noted above, tuberculosis of the genito-urinary organs is usually a part of a general tuberculosis. There is, therefore, fever, anemia, anorexia, emaciation and weakness.

There may be pain in the affected part. In tuberculosis of the urinary organs tubercle bacilli may appear in the urine. They must be differentiated from the smegma bacilli.

Vickery says the smegma bacillus can be bleached by staining with carbol fuchsine, decolorizing with 20 per cent. nitric acid, washing in water, and decolorizing again with 70 per cent. alcohol.

The kidney is not enlarged, as a rule.

The prostate and testicles become hard.

Diagnosis: This is often difficult. In suspected cases guinea pigs should be inoculated with the discharges.

Prognosis: The prognosis is bad.

Treatment: Krauss, of Boston, in a paper read before the American Institute of Homœopathy in 1913, says that a mild, bland, fluid diet is indicated. He believes the indicated homœopathic remedy is the best drug treatment.

Surgical interference may be of service.

CYSTITIS.

Synonym: Vesical Catarrh.

Definition: Inflammation of the bladder.

Etiology: Cystitis may be caused from without by carelessness in the use of injections, or by unclean catheters. By invasion of the colon bacillus—this occurs most often in children. It may occur in childbed. It may be caused by cold or by traumatism. It may be due to the travels of the gonococcus. It may occur from incomplete emptying of the bladder due to stricture or to paralysis. It may be caused by certain drugs, as *cantharides*.

Morbid Anatomy and Pathology: The mucous membrane of the bladder is swollen and inflamed. There is more or less dis-

charge of mucus, and, later, of pus. The membrane may ulcerate. Abscess may form. The bladder may become dilated and the walls hypertrophied.

Symptoms: The symptoms may be indefinite. As a rule there is frequent micturition with more or less pain on emptying the bladder. There is tenesmus of the bladder. The urine may burn and excoriate the parts. There is often more or less constant pain or feeling of weight in the bladder, just above the pubes and deep seated. The pain and frequency of urination are worse on moving about, better lying down.

In severe cases there may be systemic disturbances as shown by headache, some fever, flushed face, possibly slight nausea.

Urine: The urine is apt to be dark in color. The quantity, as a rule, is normal. The specific gravity may be normal. The reaction is alkaline, and the urine may have a disagreeable or an ammoniacal odor. Microscopical examination shows bladder epithelia, pus, sometimes blood. There are crystals of phosphates and urates.

Complications and Sequelæ: Extension of the inflammation upward may cause a pyelonephritis. In cystitis due to paralysis a general septicemia may result.

Diagnosis: The diagnosis is made on the symptoms enumerated above.

Prognosis: The prognosis depends on the cause. In case of infection from without the prognosis is usually good, but the course of the disease is long and tedious and there may be many relapses.

Treatment: Rest is essential if an early cure is desired. The patient should be placed on a bland, nourishing diet. Alcohol, coffee and spices should be interdicted. The patient should be encouraged to drink freely of water.

Warm baths will afford comfort and allay irritation. Warm applications over the bladder will often give relief.

It may be necessary to irrigate the bladder. A saturated solution of boric acid at body heat, or a little warmer, can be used every day, continuing the irrigation till the water returns clear. I had one case that I washed out every day for six weeks. The

cure was complete. In severe infection stronger solutions are sometimes used; namely, nitrate of silver, protargol or argyrol.

In chronic cases occurring in paralysis, locomotor ataxia, or similar conditions, the patient must be regularly catheterized every eight or twelve hours, and the bladder washed out every day, or every two or three days.

In some cases *urotropin*, ten to fifteen grains after meals, may be given.

Homœopathic remedies have a beneficial effect often.

Belladonna will relieve mild cases with a feeling of weight in the bladder; milky urine worse on moving about, and burning pains on urinating.

Cantharis is useful in violent, acute cases with much vesical tenesmus.

Mercurius corrosivus is similar to *cantharis*, except the case is more chronic.

Lycopodium is useful in cases with associated gastric symptoms.

CANCER OF THE BLADDER.

Cancer of the bladder is usually secondary to cancer elsewhere. The local symptoms are those of cystitis. The urine contains much blood. Tumor can sometimes be made out. In doubtful cases examination of the bladder with the cystoscope is necessary to make a diagnosis.

ENURESIS.

Synonyms: Involuntary Urination. Wetting the Bed.

Etiology: The exact cause is unknown in many cases. It occurs most often in children. There seems to be an abnormal weakness of the sphincter.

Some cases seem to be caused by stone in the bladder, by foreign bodies, by worms, by phimosis.

Symptoms: The main symptom is frequent wetting the bed. There may be no other.

Prognosis: The prognosis is good.

Treatment: Cases due to ascertainable causes should have the cause removed.

Children should be taught to urinate the last thing before going to bed. No drink should be permitted for two or three hours beforehand.

Many homœopathic remedies have been recommended.

Belladonna, involuntary urination due to weakened sphincters. This is probably most frequently indicated.

Causticum, wetting of bed in first sleep.

Conium is useful in the dribbling of old men.

Sepia, the urine is offensive.

SECTION XI.

Skin Diseases.

INTRODUCTION.

This section is intended simply to give a general idea of some of the more common skin diseases. The eruptive fevers, measles, scarlet fever, and so on, have been described in the section on infectious diseases. The more common skin diseases are here arranged alphabetically.

Some skin eruptions are not contagious, others are very much so. To be on the safe side, towels, dishes, anything coming in close contact with a skin patient, should not be used by anyone else till after a thorough cleansing. A well person handling a patient with skin disease should use the utmost care in keeping his own hands clean. He should thoroughly wash both before and after attendance on such a case, especially if the eruption has been touched.

HYGIENE.

The skin is an important organ and it is essential that it be kept in good condition. If not properly cared for the various secretions and epithelial cells tend to accumulate and clog the pores. Dirt sticks to the surface and adds to the trouble. Cleanliness is essential to prevent skin disease.

The food should be plain, nutritious, sufficient, and yet not too much. Impaired digestion is the source of many skin diseases. A proper amount of exercise must be taken to keep the various functions in good condition. The clothing must be sufficient for comfort. It should never be enough to cause undue sweating, and yet sufficient to prevent chilling in cold weather. It must keep the body dry, and yet allow for the insensible perspiration necessary to health. Highly colored underwear may

irritate the skin on account of the dyes they contain. Either wool, linen, or cotton in its natural color is best. Some persons find wool irritating, they should wear one of the other two materials.

A warm bath should be taken once or twice a week for cleanliness. A warm bath opens the pores of the skin, softens any excess of sebaceous secretion that may have accumulated, and gets rid of it. This is best done at night on retiring to prevent catching cold. In the morning a cold bath is desirable. This acts as a stimulant. It causes an increased flow of arterial blood to the skin, and an increased flow of venous blood and lymph away from the skin, carrying off deleterious substances. It also tones up the nervous and elastic tissues of the skin and conduces to the general health. After the bath a rub with a bath towel till the skin is in a glow is beneficial. The first effect of cold water is to close the superficial capillaries and drive the surplus blood away from the surface. This is promptly followed by a reaction and by the return of the blood to the surface. The cold bath should always be followed by a feeling of comfort. If not, the person is either not in good health, or the water has been too cold. A few individuals can never accustom themselves to the cold bath on account of lack of reaction. Most persons, however, can gradually accustom themselves to it. Once accustomed to it it becomes a necessity and a pleasure.

ACNE.

Synonym: Pimples.

BLACKHEADS.

Synonym: Comedones.

Acne—pimples, comedones—blackheads, are closely related and must, therefore, be placed in the same chapter.

Symptoms: Of all the skin diseases that may attack the face the most common is acne. Less often it attacks the back and shoulders. This is an inflammation of the sebaceous glands. The glands become swollen, hard, and often maturate forming

small pustules. It is frequently preceded by or associated with the condition known as blackheads, technically called comedones. Where blackheads exist they come first. They are simply a plugging up of the openings of the glands—the pores—with a thickened secretion. The dust and dirt adhering to the surface give the black appearance, hence the name blackheads. They can be removed by pressure, either with the fingers, or by means of an old fashioned watch key. They should always be removed. Blackheads usually appear in sluggish, oily skins. After their removal massage is an excellent measure.

The plugging of the pores by comedones may result in inflammation of the glands—acne or pimples. Pimples are most apt to appear about the time of puberty, when there is rapid growth and development of all the functions of the body.

Treatment: Attention should be paid to the habits and general health of the patient. The digestion must be looked after, the patient avoiding rich, fat, or stimulating food. Plenty of water should be drunk. The bowels must be kept regular.

In both comedones and acne applications of cloths wrung out in hot water will relax the skin and loosen the plugged secretions. After hot applications cold ones should be made for stimulating purposes.

Dilute alcohol may be used as a lotion after the acute condition has subsided.

Aconite has seemed to help some cases with considerable inflammation.

Nux vomica is of service in cases due to high living or dissipation.

Sulphur has helped in indolent cases.

Kali hydroiodicum is of benefit in some cases.

Hepar will benefit if the spots tend to maturate.

Mercurius solubilis is of service when the pimples tend to become bluish-red nodules.

Phosphorus follows *mercurius,* and may be necessary to complete the cure.

Iodium, conium or *clematis,* long continued, will help the indurated form.

Silica is sometimes of service when the case is very chronic.

ALBINISMUS.

(Latin, *albus*, white.)

Albinismus—the name given to the condition causing an albino. It is an absence of coloring matter from the skin, hair and eyes. The patient is born that way. The general health is unimpaired. The condition is incurable.

ALOPECIA.

Synonym: Baldness.

Baldness, falling out of the hair, technically known as alopecia, may be partial or complete.

Symptoms: Alopecia is quite common in elderly persons who are otherwise healthy. The hair sometimes falls out after severe illness. In other cases baldness is due to seborrhea and dandruff of the scalp. Baldness occurs in comparatively young members of certain families.

Alopecia areata—partial baldness—may attack the scalp or the bearded portions of the face. The cause is not always known.

The baldness of elderly people is not curable. Premature baldness may be. Baldness from fevers is usually only temporary, and the hair returns of itself.

Treatment: On the scalp, frequent shampoos will help in some cases. Bald spots of the beard may be benefited by massage. Stimulating lotions will benefit. Alcohol is one of the best.

To massage the scalp it should first be thoroughly cleansed and dried. Then the scalp should be thoroughly massaged with the dry hands. First manipulate the skin from before backwards and from the median line to the sides. Then work the scalp and underlying tissue back and forth over the skull. Finally, rub in thoroughly some oily substance.

Graphites has caused the hair to reappear in a number of cases of alopecia. In one patient of mine who had been nearly bald a long time a full crop of hair appeared after a course of *graphites* given primarily for chronic constipation.

BOILS:

(Anglo-Saxon, *Byl.*)

Synonym: Furuncle—a little thief, steals slowly.

Definition: A boil is a localized inflammation of the skin attended by the formation of pus.

Historical Note: Boils are as old as human kind. The patience of Job was tested with a crop of boils 3,500 years ago.

Etiology: Boils appear more frequently in young persons than in old; more frequently in men than in women. The furuncle that appears in the ear is a particularly atrocious punishment.

It was formerly supposed that boils appeared only as the result of a deteriorated condition of the blood. It is now believed they are due to infection from without. They are undoubtedly auto-infectious.

Symptoms: The first symptom usually noticed is a painful point at which the skin becomes raised, red and hot. The pain is throbbing and acute. The boil increases in size and painfulness for three or four days, when the summit of the tumefaction breaks down and pus exudes. By pressure around the opening a thickened plug or slough of broken down tissue, covered with pus and frequently blood-streaked, can be forced out leaving a cavity. If the cavity is thoroughly emptied healing takes place in a few days more. If not it may fill up again. Occasionally a boil does not break down, but gradually dries up and disappears.

Boils have a tendency to appear on the back of the neck and on the buttocks. They may appear anywhere on the surface of the body. As the pain and tenderness depend on the tension, these symptoms are more severe when the boils appear where the skin is naturally drawn tight, as in the ear, or at the nostril.

There is marked local elevation of temperature over a boil. I recall one case where a boil appeared in the axilla of a child, and there was a difference of more than two degrees between the two sides.

Complications and Sequelæ: Boils often appear in crops. This is probably due to auto-infection. Boils may be a complication

of Bright's disease and of diabetes, and occasionally of typhoid fever.

Diagnosis: This is self-evident.

Prognosis: This is good.

Treatment: Cleanliness is the best prophylaxis against boils When one appears it and the surrounding skin should be kept aseptically clean. This will provide against auto-infection. If the patient is not in good condition he should be built up.

The knife is indicated after the formation of pus. It should not be used too soon else it will aggravate the case. When the boil is opened the cavity should be thoroughly emptied. It may be washed out with hydrogen dioxide. If large it may be swabbed out with pure carbolic acid, followed immediately by pure alcohol.

The indicated homœopathic remedy will do much.

Belladonna is useful in the beginning for the heat, redness and throbbing pain.

Arnica is indicated where there is a tendency to recur. It may be used locally.

Hepar is frequently of service after the formation of pus. It may be given low to hasten the process.

Phytolacca is indicated if the neighboring glands are swollen.

CALLUS: CORNS

Etiology and Symptoms: Callus spots, thickened epidermis, result from the constant use of certain tools, or from constant pressure on one spot. They come, as a rule, on the hands and feet.

A corn is a special kind of callus on the foot due to badly fitting shoes.

Callus tends to get well if the cause of the trouble is removed.

The best **treatment** is first to soak the part affected in hot water, and then cut or shave off the excess of epidermis.

CANCER OF THE SKIN AND MUCOUS MEMBRANES.

Cancer of the skin and mucous membranes may occur in many forms. Any sore, no matter how insignificant, that persists for a few weeks despite treatment should arouse suspicion.

Etiology: Cancer of the skin and mucous membranes is most often due to traumatism, or to some constant local irritation, as smoking.

Morbid Anatomy and Pathology: There is first a nodule which may not be very large in the beginning. This, after a time, ulcerates, giving off a slight thin discharge. Still later there may be induration about the edges, and finally the neighboring glands may swell.

Symptoms: The local symptoms are as described in the preceding paragraph. The leading characteristic is persistency. There may be some tenderness; occasionally there is itching or burning. I have met with such cases where the lesion was on the lip, and where it was on the hand.

Some cases may go on for weeks or months without much change. Then rapid and extensive ulceration may take place. The disease invades the system. Cancerous cachexia develops, and the patient dies of exhaustion or of hemorrhage from an eroded vessel.

One patient, a man of fifty, with cancer of the right side of face, noticed the first swelling at the age of fifteen. This grew slowly. At forty-seven he had his first operation, and has had two since.

Complications and Sequelæ: There may be general involvement from systemic infection. Hemorrhage may occur from an eroded blood vessel.

Diagnosis: A case that presents an insignificant nodule or pimple that ulcerates, that lasts a long time despite treatment with drugs or external applications, should arouse suspicion.

Prognosis: Is good if correctly treated before there is general infection. It is bad if not stopped early.

Treatment: Complete removal with the knife is the best treatment if diagnosed early. The x-ray will cure some super-

ficial cancers. One patient of mine was cured of a small cancer of the lip by the x-ray. Radium is used to advantage sometimes. Three exposures, at intervals of a week or ten days, cured a cancer on the back of the hand. This patient had been operated on twice, but the wound refused to heal. The condition had lasted for several years.

Phytolacca will sometimes hold a case in abeyance, even improve it. It kept one patient of mine with cancer of the right side of the face comfortable for months. I think it helped to prevent a recurrence of an intestinal cancer.

Arsenic in potency, *arsenic* in the form of Fowler's solution, *arsenic* externally, has helped many cases. Locally it is used for its escharotic effect.

Dearborn mentions *condurango* in epithelioma of the lips or anus or from a mole, with fissure-like ulceration.

Kreosote, ulcerating cancer with offensive secretions.

CANITIES.

Synonym: Gray Hair.

Etiology and Treatment: Gray hair is common in old people. Occasionally patches of gray hair are met with in young persons. It is said that great mental shocks, grief, and so on, sometimes change the color of the hair very suddenly.

The condition may be disguised with dyes, always a dangerous procedure on account of the ingredients used. It cannot be cured otherwise.

CARBUNCLE.

(*Carbunculus,* diminutive of carbo, *a live coal.* A genius must have named this condition.)

Synonym: Anthrax.

Definition: A carbuncle is a hard, circumscribed, deep-seated, painful inflammation of the subcutaneous tissue, accompanied by chill, fever and constitutional disturbance (Gould).

A carbuncle is of the same nature as a boil, except that it covers a larger area and presents several openings instead of one. It most often occurs on the back of the neck.

Etiology: Carbuncle is more frequent in middle aged or elderly men who are in a run down condition. Many carbuncle patients are also sufferers from diabetes.

Bacteriology: The pus organisms are found.

Morbid Anatomy and Pathology: A diffuse redness appears over a more or less extensive area of skin. This becomes infiltrated, raised, and hard to the touch. After a few days numerous openings appear exuding, first, a watery fluid, then pus. Extensive sloughing takes place. This is thrown off after a time leaving an ulcer which in favorable cases heals over, leaving a scar.

Symptoms: With the local condition outlined above, systemic symptoms develop, chills, fever, and general run down condition.

Complications and Sequelæ: Bad cases may develop general septic poisoning.

Diagnosis: Carbuncle is differentiated from a boil by its larger area and the numerous openings. From erysipelas by its more localized area, thickening and hardening of the skin, formation of pus and location.

Prognosis: The disease is sometimes fatal. The vast majority of cases recover.

Treatment: A carbuncle should be treated surgically by free use of the knife, and by wet antiseptic dressings. Hydrogen dioxide may be used as a wash after slough has been thoroughly removed. The cavities may be swabbed out with pure carbolic acid, followed immediately by pure alcohol.

The patient should be built up with a nourishing diet.

Hepar is useful in cases with much pus.

Silica in sluggish cases.

Arnica may be of service where there is much tenderness with a bruised feeling.

Anthracine, a nosode, may help.

The snake poisons may be necessary in general sepsis, as *lachesis, crotalus,* etc.

CHLOASMA.

Synonyms: Melanoderma. Liver Spots.

Definition: Chloasma is the name given to increased pigment in the skin.

Etiology: This may be due to certain diseases—Addison's disease, disease of the uterus, and so forth. Certain medicines make an increased deposit in the skin; for example, arsenic and various silver preparations.

Others have larger or smaller spots of discoloration which appear at birth and persist through life.

Chloasma, due to disease of other organs, tends to disappear when the original disease gets well. Local treatment in other forms may clear up the condition for a time, but the spots tend to recur.

Treatment: One of my patients, a girl of eighteen, had a large number of brown spots on the lower abdomen. *Mercurius solubilis* 6x cured it in three weeks. The patient had had the condition for a year, and came to New York from Illinois for treatment. She had tried many local applications at home.

Congenital cases do not respond to treatment, in fact, they need none as a rule. A woman of twenty-eight in the Flower Hospital with typhoid fever had a beautiful skin of normal texture, but half the lower chest, abdomen and one thigh were of a brown color, the rest of the body and face were white. The condition was congenital.

DERMATITIS.

(Greek, δερμα, skin.)

Definition: Inflammation of the skin.

Etiology: Dermatitis may be caused by cold—frost bites, by heat, by traumatism, by various irritants.

Symptoms: The inflammation may vary all the way from a simple redness, like in a mild case of sunburn, to the formation of vesicles which later become pustular, as happens from a bad burn.

There is a form known as *dermatitis exfoliativa,* or *pityriasis rubra,* which is quite generally distributed over the body, and is

followed by extensive desquamation. It occurs mostly in gouty or rheumatic subjects. The cases I have seen have all been at the Metropolitan Hospital and occurred in alcoholics.

Diagnosis: Localized spots of dermatitis offer little trouble in diagnosis.

Dermatitis exfoliativa must be differentiated from psoriasis. The former is general over the body. Psoriasis attacks all parts of the body at the same time, but the eruption is in isolated spots.

Prognosis: Mild cases recover. Severe ones may last a long time. They may become septic and cause death.

Treatment: The cause must, of course, be removed. The patient must be put in good general condition. Mild cases will require no special local treatment.

Burns may have unguentine applied, a most excellent remedy. Or carron oil may be used. A solution of picric acid is also of service.

Blisters should be opened at the most pendant portion and emptied, then the application made and the part kept from the air.

In dermatitis exfoliativa carbolized vaseline may be applied to relieve the irritation.

Of the many remedies that can be used *belladonna* is the best for a simple dermatitis of any type.

Arsenicum album is sometimes useful in run down patients.

Cantharis is one of the best remedies in burns. It is strictly homœopathic, as *cantharides* is used to produce blisters.

Picric acid, internally as well as externally, may also be tried.

Agaricus is especially useful in dermatitis due to frost-bite.

ECTHYMA.

(Greek, εκθυμα, pustule.)

Symptoms: Ecthyma is an eruption that occurs in weak and run down, individuals. There may be but one or two spots, there are never very many. They are about the size of a dime, well-defined, usually pustular, that is, a crust forms, and they contain matter.

Diagnosis: The spots of ecthyma are deeper than those of impetigo. The eruption in impetigo is very superficial and occurs mostly in children. It may be epidemic.

Treatment: The patient must be put in a healthy condition. Scratching must be prohibited, because the disease may be carried to other parts of the body otherwise. Cleanliness is a necessity. The spots usually heal without much trouble.

ECZEMA.

(Greek, ἐκζέω, to boil over.)

Definition: Eczema is the most common of all skin diseases. The forms of its eruptions are manifold. In appearance it may simulate any known skin disease. Eczema is a catarrhal affection of the skin. At some stage of its development it must have had an oozing or weeping surface, although temporarily it may be dry. Another distinguishing mark of eczema is that the eruption shades off into healthy skin, it is not sharply defined like many other conditions.

Eczema is rarely an acute disease. It is usually chronic, coming and going for years.

Etiology: There are many forms of eczema. Some of the more common are redness and chapping of the hands due to certain occupations, as those of bakers, bricklayers, washerwomen, and so on. The chapping and cracking due to cold weather. The so-called salt rheum of the hands is eczema. The leg ulcer of old people is eczema. The crusts found on babies' heads and cheeks is still another form of this same disease.

Any condition that lowers the vitality may, after some local irritation, be followed by eczema.

Symptoms: Any part of the body may be affected. In infants and young children it appears most often on the face and scalp. In adults the hands and arms are favorite places. Usually it is more or less localized.

It may begin with a simple redness which rapidly develops papules, vesicles or pustules. These may desquamate or form scabs. In short, all forms of skin lesions may present themselves at the same time in different places; or a given case may

show different lesions at different times. Whatever the type, or wherever the lesion, at some stage of its existence it shows a moist exudation. Another point to be remembered is that the lesions present no definite outlines, but merge gradually into normal skin.

Many forms of eczema have been described, according to the leading characteristic of the eruption. For example, *eczema erythematosum, eczema papulosum, eczema vesiculosum, eczema pustulosum, eczema squamosum,* etc.

Diagnosis: As noted above, eczema must, at some stage, exhibit a moist exudation. The skin lesions fade off into normal skin. These points are characteristic and diagnostic.

Prognosis: The prognosis is usually good, although the disease may be very chronic.

Treatment: For chapping, applications of cold cream, camphorated vaseline, or a wash of glycerine and water, half and half.

Soap and water aggravates acute eczema. It is useful in removing scabs in chronic cases. Water with bran can be used for cleanliness.

The patient suffering from eczema must be put in good general condition. Attention must be paid to the diet. Meats, all rich and spicy foods, tea, coffee and alcohol should be interdicted. Plenty of water should be drunk.

The number of local applications recommended for eczema is legion. Crusts may be removed by applying oiled rags to soften them. Then some mild lotion to keep the parts clean.

Graphites is indicated in cases where there is considerable exudation and itching of the skin; the patient is also constipated.

Apis is indicated if the eruption is vesicular.

Mezereum is especially useful in infants with large crusts on the head. The change wrought in a few days is sometimes marvelous.

Nux vomica cured a case of eczema of the scrotum and groin.

Many other remedies may be indicated as *arsenic, sulphur, antimonium tartaricum,* etc.

ELEPHANTIASIS.

(Greek, ελεφος, an elephant.)

Symptoms: This is a disease where the skin and subcutaneous tissue is much thickened due to blocking of the lymphatics. It occurs most often in one leg, next in the scrotum or pudenda, very rarely in an arm. In tropical countries, where the disease is comparatively common, the lymphatics are found to be blocked by the filiaris sanguinariæ homenis. The sporadic cases in this country are mostly of unknown origin.

The affected part becomes of enormous size. The cases I have seen have been of the lower extremity, from the groin to the foot.

Treatment: Bandaging and massage are said to be of benefit.

ERYTHEMA.

(Greek, ερυθημα, a blush.)

Symptoms and Treatment: Erythema is a simple reddening of the skin. It is usually due to external causes, though certain drugs will produce it in some persons. One form with which everybody is familiar is sunburn. Cream is a good application for that form. Anointing the skin with vaseline before and during exposure is a good preventive.

Another common form of erythema is *intertrigo*—redness of the skin due to friction of adjacent parts, or between folds of the skin, as under the breasts in very stout women. Sometimes the skin is red and raw looking from constant perspiration. Chafing is another name for it. The best preventive is cleanliness and keeping the parts separated. When it exists separate the parts and dust with starch. Keep clean but do not wash too frequently as that will aggravate.

Arsenicum album and *rhus toxicodendron* have seemed the most frequently indicated remedies in many cases.

ERYTHEMA MULTIFORME.

Definition: This is a more or less generalized erythematous condition attacking, by preference, the extensor surfaces of the hands, arms and legs, although no part is exempt.

Etiology: The cause is supposed to be gastro-intestinal toxemia. It occurs most often in rheumatic subjects.

Symptoms: The disease runs an acute course. There are generally chills, fever, and rheumatic pains here and there. The skin lesions consist of reddish macules, papules, sometimes nodules. The macules usually clear up first from the center leaving rings. Rings may appear within rings. The eruption lasts a few days to a week or two.

Treatment: The diet must be regulated, cutting out stimulating food and drink.

Such remedies as *belladonna, rhus toxicodendron* and *phytolacca* are indicated.

ERYTHEMA NODOSUM.

Etiology: This seems to be a rheumatic condition. It appears mostly in children and young adults.

Symptoms: The disease is ushered in by malaise, fever and rheumatic pains. I have seen the temperature go to 102° F. The rash attacks the front of the legs by preference, although it may appear on the arms, and in one case of mine a nodule appeared on the nose. The eruption consists of red, swollen, tender spots, which may be as large as a hickory nut. At first they are red, then begin to fade out like black and blue spots, changing their color as they go. They desquamate.

Treatment: The condition lasts two or three weeks. The patient is best in bed. A liquid diet should be given, at least one free from stimulating food or drink.

Rhus toxicodendron is indicated for the rheumatic pains and in the skin lesion. I have used it successfully.

Arsenicum album may be needed to complete the cure.

Other remedies may suggest themselves.

FIBROMA.

(Latin, *fibra oma,* a tumor.)

Symptoms: Fibroma is a condition of the skin characterized by hypertrophy and outgrowth of the connective tissue from the

deeper layers of the corium or subcutaneous tissue. Some patients present one or two large tumors, others present many small ones. I have seen but one marked case; the patient was a young negress who eventually died of tuberculosis. When she entered the Metropolitan Hospital she had more than a hundred of these small tumors, varying in size from a hickory nut to a duck's egg. They were on all parts of the body, front and back, on the arms, thighs, and legs, even on the forehead and chin. The skin covering them was normal; the tumors felt soft like lumps of flesh.

Treatment: There seems to be no treatment for the condition other than removal. Where one or two exist they can be cut out. Where there are many, like in my case, removal is, of course, out of the question.

FRECKLES.

Synonym: Lentigo.

Definition: Freckles is a special form of chloasma. The technical name is lentigo.

Symptoms: Freckles are small brownish or blackish spots appearing most often on the face and backs of the hands.

People with reddish hair and white skins seem to be most susceptible. Heat and exposure bring out the eruption.

Treatment: The spots can be removed by treatment, but the measures needed are rather harsh, and the spots almost invariably return.

Mercurius solubilis occasionally seems to be of benefit.

HERPES.

Symptoms: Herpes are little blisters that appear on the lips or near them. They are frequently found with ordinary colds.

Herpes also appear with many of the severer forms of fever. They are of no special importance, and disappear when the patient gets well.

Treatment: Camphorated ice—a preparation of vaseline—is the best application.

HERPES PROGENITALIS.

Symptoms: Herpes sometimes appear on the penis, less often on the vulva.

Treatment: Cleanliness is essential. *Boric acid* may be used as a dusting powder.

Arsenicum album cured one case of mine.

HERPES ZOSTER.

Synonym: Shingles.

Symptoms: Herpes zoster is quite a severe disease. It is a collection of vesicles or small blisters following the course of some nerve. It may occur on any part of the body, and is almost always on one side. The most common location is around one side of the chest following the line of an intercostal nerve, or down the back of the thigh over the sciatic.

The patient usually has shooting pains over the affected area, with fever for a day or two before the vesicles appear. The pains and the fever persist until the vesicles begin to fade out.

One patient, a woman, aged fifty, had shooting and burning pains that were excessively severe. The fever rose to 100° to 101° F. every day. The vesicles appeared on both sides from the buttocks down the backs of the thighs and legs to the ankles. It took several days for them to travel down. Later, there was dermatitis between the vesicles. The patient was ill six weeks.

Complications and Sequelæ: Sometimes the pain will persist after the eruption goes. There may even be a temporary paralysis afterwards.

Diagnosis: The diagnosis is easy, because the vesiculation following the line of a nerve is characteristic.

Prognosis: The prognosis is good. Most cases run a course of two to four weeks.

Treatment: The patient is best in bed. A non-irritating or liquid diet is indicated until all acute symptoms have subsided.

Rhus toxicodendron is the closest remedy to the average case. It has the nerve pain and the vesicular eruption.

Aconite may be of service in the very beginning.

Arsenicum album is indicated if the pains are burning in character and the vesicles tend to become pussy.

Plumbum cured a following paralysis in one case.

HIRSUTIES

(Latin, *hirsutus,* shaggy.)

Symptoms: Hirsuties is the name given to hair growing in an abnormal place, or to a general excess of hair.

Women occasionally have it in the form of an incipient moustache or beard.

Treatment: The best method of removal is the electric needle. This should be used only by an expert.

HIVES.

Synonym: Urticaria.

Symptoms: Hives is an eruption of white or red blotches that usually appears suddenly after some indiscretion in eating, and may disappear just as suddenly. I have seen some cases following salt water bathing. As a rule, it lasts but a short time. It may last several days. The spots may be uniform or irregular in size and shape. They are usually accompanied by intense itching, sometimes by burning.

The eruption is usually distributed over the whole body and causes much annoyance. In one case it caused delirium.

Some persons always have hives after eating fish, others after eating strawberries, still others after some other particular food.

Treatment: Lotions of alcohol and water, half and half, or of vinegar and water in the same proportion, are useful in allaying irritation.

Apis is almost a specific.

ICHTHYOSIS.

Symptoms: Ichthyosis means fish-like, scaly. This is the name given to a rough, scaly skin. The scales may be very fine, or they may be comparatively coarse. Bad cases are rare in this country.

The milder form is sometimes met with. The disease may be no more than a roughness and dryness, which flakes off readily. It appears very early in childhood. The scaliness is most marked on the extensor surfaces of the limbs. It is always worse in cold weather.

Treatment: The disease is incurable. It may be helped by frequent warm baths with soap and water.

IMPETIGO CONTAGIOSA.

Symptoms: Impetigo is an eruption that presents one or more pustules varying in size from a pea to a finger nail. This pustule or pimple fills with pus and a crust forms outside. It occurs usually in well nourished children and in young people. Individual spots tend to disappear in a week or two. Cleanliness is essential. Scratching must be forbidden, as, otherwise, the patient may transfer the infection from one part of the body to another. The disease is also contagious from one person to another.

Diagnosis: Impetigo has been diagnosed when the disease was small-pox.

Treatment: In one family a child at school became infected, and before the disease ended I had three boys aged, respectively, thirteen years, four years, and ten months, and one girl aged fourteen years, under my care.

The first boy, when I saw him, had had a succession of spots for six weeks. They appeared on the head, arms, legs and neck in that order. The child had chills and some fever. This case cleared up under soap and water for cleanliness, together with *arsenicum* as a remedy. The spots looked not unlike vaccination marks, and when the scabs came off they left red areas of skin which gradually disappeared.

The other patients caught the disease from the first one, and showed the spots from two to four weeks later. The skin and hands seemed to be the points of preference. Under external cleanliness and *arsenicum* all got well in two to three weeks.

KELOID.

(Greek, χηλη, a claw.)

Definition: Keloid is a new growth of the connective tissue of the skin that forms masses or bands of scar tissue.

Etiology: Keloid starts from a scar or cicatrix from some wound, it may be a vaccination mark or burn or any wound that leaves a scar.

Symptoms: The growth may form nodules or masses or bands that reach out into the surrounding tissue. This irregular shape gives the disease its name, keloid, like a crab's claw. The growth is composed of very fine connective tissue. It is of a reddish hue or may be pigmented.

In only rare cases is the growth painful or tender.

Treatment: From the very nature of the case, an overgrowth of scar tissue, surgical removal is useless because the disease usually recurs in the new scar. Some cases result in spontaneous cure.

Daily use of the Bier hyperemic cups has been recommended by some.

Hypodermic injection of fibrolysin has cured some cases. Fibrolysin has an affinity for scar tissue.

Dearborn suggests *calcarea fluorica, fluoric acid, graphites, nitric acid.*

LEUCODERMA.

(Greek, λευκος, white; δερμα, skin.)

Synonym: Vitiligo.

Definition: Leucoderma is a discoloration, or lack of color of the skin due to absence of pigment.

Symptoms: The condition is said to be acquired, and once the process begins is apt to slowly continue.

The affected spot loses its normal pigment, and in contrast to the surrounding skin looks very white. The sharply outlined borders may, indeed, have an increased pigmentation still further accentuating the contrast. There is no change in sensation or function of the affected area.

It sometimes occurs in debilitated subjects, or following some

disease. It is supposed to be due to some defect of the nervous system. The discoloration is often symmetrical.

I have seen the disease in negroes. In a white man, aged fifty, suffering from pulmonary tuberculosis, the white areas were on the backs of the hands and fingers, on the under side of the chin, the front of the chest, the back of the neck, on the scrotum, and on the under side of the penis. The patient spent most of the time out of doors on account of his tuberculosis, and a curious feature was that the white spots did not sunburn as did the normal skin. He reported that his son, aged twenty, suffered from the same condition.

Treatment: This is usually unsatisfactory though Sibley reports cures by the use of Bier's cups together with the x-ray.

Staining with walnut juice may be used for its cosmetic effect.

Dearborn specially mentions *arsenicum album, sulphur, nitric acid,* and *zinc phosphide.*

Some cases tend to recover spontaneously.

LICHEN PLANUS.

Definition: Stelwagon calls lichen planus an inflammatory disease characterized by pin-head to small pea-sized flattened, glistening, crimson, or violaceous papules, with often a slight central depression, and often an irregular or angular base; tending to coalescence and the formation of areas with a roughened or scaly surface.

Symptoms: The papules are small and flat. They are very slightly raised, solid, and slightly scaly, varying in color from violet to purple. They tend to run into irregular outlines. The common seat of the lesion is on the fronts of the forearms, although they may appear anywhere except on the head and face. There is always more or less itching. The disease is very chronic.

Treatment: Many patients are in a run-down condition. The first indication for treatment, therefore, is to build up the system. A change of climate is often of service.

Bran or starch baths are soothing.

Arsenicum album is probably the most useful drug in this condition.

Arsenicum iodatum may sometimes help where *arsenic* fails.
Mercurius solubilis is useful in cases with intense itching.
Natrum muriaticum is another drug that may be indicated.

LICHEN SCROFULOSUS.

Symptoms: This condition occurs mostly in scrofulous children. It is characterized by an eruption of miliary papules that are similar in color to the normal skin, or pale yellow, or a dull red (Tilbury Fox). They are usually limited to the trunk. They sometimes simulate acne. There is rarely any itching.

The **treatment** must be directed mainly to the building up of the patient.

LIPOMATA.

These are fatty tumors that may be few or many scattered over the surface of the body.

LUPUS ERYTHEMATOSUS.

Symptoms: This is a superficial new growth that may start from sun-burn, chilblain, seborrheic eczema, or acne. There is, first, an erythema, then the sebaceous follicles become plugged, and the lesion becomes slightly scaly. The disease is found most often in women of early adult or middle age, and in those of a strumous diathesis. It occurs most often on the face, less often on the scalp and backs of the hands. The disease may appear in spots that run together over the nose, the so-called "butterfly lupus." It runs a long course, and then tends to disappear, leaving a slight scar.

Treatment: Consists in building up the patient. The parts should be frequently washed with soap and water. Alcohol and water, or pure alcohol may be used as a lotion afterwards.

The x-ray is of service.

Belladonna is indicated if there is much scaling.

Graphites is indicated in seborrheic cases.

Calcarea carbonica may be needed as a constitutional remedy.

LUPUS VULGARIS.

Symptoms: This is a form of tuberculosis of the skin. It occurs most often on the face. It usually begins with soft papules or tubercles that run together and form patches of a reddish color, slightly elevated. The disease is very chronic, and may last for many years. It tends to spread at the periphery and heal at the center, leaving scar tissue that atrophies. The extending parts tend to ulcerate. It may reach to the mucous membrane about the eyes or mouth, and as scar tissue forms distort the features.

Diagnosis: This form of lupus must be distinguished from the erythematous form, and from cancer.

Prognosis: The disease is very chronic and tends to recur.

Treatment: During my service as acting dermatologist at the Metropolitan Hospital in 1904 a German, aged sixty-three, entered with the following history. It is quite typical of lupus vulgaris.

The disease appeared first when the patient was eight years old. After several years' treatment he was pronounced cured; at the age of thirty the disease reappeared. From then on he had gone from clinic to clinic, from hospital to hospital. Sometimes he was better, sometimes he was worse. When he entered the Metropolitan the entire left side of the face was affected. He was given a number of x-ray treatments, which apparently aggravated the condition and set up an acute inflammation. Various applications were made and numerous remedies given. All this time some part of the affected area presented an ulcerating surface. Finally it occurred to me to treat the ulceration as an open wound, and in December I ordered wet dressings of *bichloride of mercury*, 1 to 4,000, applied. *Iodoform* 3x was given as a remedy. In February *rhus toxicodendron* was given in place of the *iodoform*. The wet dressings were continued until July when the area was completely healed over and the patient discharged. A year or two later I found mention of *bichloride* dressings in Stelwagon's book on Skin Diseases. I was not aware until then that they had ever been used by anyone else. My own use of them was because it seemed the logical thing to do.

The Finsen light helps some cases it is said.

The solid *carbon dioxide* has been used of recent years. Other treatment has been sufficiently outlined above.

MEDICINAL ERUPTIONS.

Certain drugs may cause eruptions in specially susceptible persons. The physician should know this and be on his guard. The homœopathist, with his more accurate knowledge of drug action and his use of infinitesimal doses, is not so liable to be led astray. I shall quote largely from Tilbury Fox.

Arsenic may cause brown pigmentation, blotches, and urticaria.

Belladonna may cause erythematous rashes and desquamation. Every homœopathist has used it to cure these indications.

Bromides may produce eruptions from acne to boils; all, according to Fox, connected with inflammation of the sebaceous glands.

Chloral sometimes causes the following: "A diffused redness of the face with puffiness and conjunctivitis sets in, followed by well-defined and slightly elevated erythematous patches on the face and neck, extending and coalescing, and spreading to the shoulders, trunk, neighborhood of large joints, dorsum of hands and feet, etc. There is great irritation present, and the rash on the face is more crimson than elsewhere. It fades in a few days with occasional slight desquamation." There is dyspnea and palpitation of the heart.

Copaiba sometimes causes erythema and urticaria. There is irritation and a characteristic odor. It disappears without desquamation when the drug is stopped.

Iodides may cause eruptions similar to the bromides. They also cause purpuric spots and urticaria.

Opium and **morphine** may cause a scarlatiniform rash followed by desquamation, and accompanied by puffiness of the face.

Quinine sometimes causes itching and stinging with an erythematous or urticarial rash. There may be desquamation. There are constitutional symptoms.

Salicylic acid may cause erythema or urticaria. There may be temporary deafness.

MOLE: NÆVUS: BIRTHMARK.

Symptoms: Mole, nævus, birthmark, may be simply an increase of pigment, or increase of pigment with hypertrophy. The cause is unknown. These marks vary much in appearance and in size.

Treatment: They are best left alone. Some of them can be removed by the knife or cautery.

Solid *carbon dioxide* is sometimes used. Treatment of these conditions must be left to the specialist.

MOLLUSCUM CONTAGIOSUM.

Symptoms: This occurs mostly in undernourished children about the eyes and mouth. The eruption consists of discrete growths in size from a pin-head to a pea. They are whitish or pinkish in color, umbilicated, and filled with milky contents. The disease is contagious. It tends toward spontaneous recovery.

Treatment: The contents may be squeezed out and touched with caustic.

Kali iodide is the only remedy mentioned in the books.

NAIL AFFECTIONS.

The nails may become brittle, or grooved, or clubbed, or what not, as a result of general malnutrition.

There may be an inflammatory condition, *onychia*, inflammation of the matrix; or *paronychia* (whitlow), inflammation of the folds of the nail. There is acute inflammation with local heat, redness, swelling and severe pain.

Treatment: Soaking in hot water, as hot as can be borne usually gives some relief. There may be suppuration, then the knife is indicated.

Hepar low will hasten the case.

PEMPHIGUS.

(Greek, πεμφιξ, a blister.)

Symptoms: Pemphigus is a comparatively rare disease which consists of the successive formation of blebs or blisters on various parts of the body. These may be few or many, widely scattered or close together. The cause is unknown. The trouble may be acute or chronic. Sometimes the disease is so severe that death ensues. Most of the cases are not so very sick, but the succession of blisters extends over a long time. I have seen a number of cases.

Treatment: *Apis mellifica* is most often indicated.

Hepar is useful in cases with formation of pus.

PRICKLY HEAT: MILIARIA: SUDAMINA.

Symptoms: Prickly heat is a mild inflammatory disease of the skin due to heat; hot weather, occupation in hot places, or too much clothing may cause it. The eruption is very fine and is scattered over the body and face. The eruption is usually more profuse on some parts than on others. It is usually associated with itching and burning.

Treatment: The first essential is to avoid the cause if possible. Next put the patient in good general condition. Local applications for relief of the discomfort are powder, starch or talcum or lotions of vinegar and water. Prevent scratching as that aggravates.

Lemon or lime juice drinks are permissible.

Apis mellifica is indicated in this condition when the rash is profuse and the itching excessive.

Aconite if there is much inflammation of the skin and the patient is nervous.

Bryonia and *rhus toxicodendron* are other remedies frequently indicated.

PRURIGO.

(Latin, *prurio*, to itch.)

Symptoms: This is a rare condition consisting of isolated papules, of a color lighter than the skin or pinkish, mostly on ex-

tensor surfaces of the extremities. Scratching may make the eruption eczematous. The disease is more common in the poorly nourished, begins in childhood and persists throughout life.

Treatment: The patient must be built up.

Arsenicum is probably the most useful remedy.

PRURITUS.

(Latin, *prurio,* to itch.)

Synonym: Itching.

Symptoms: Pruritus, itching of the skin, is a disease without visible change in the skin structure, characterized solely by itching. A few cases of pruritus are found in association with other diseases, as diabetes. Other cases have no known cause.

Fortunately the symptom is limited usually to one part of the body. It comes on in paroxysms and is very distressing.

Treatment: Lotions of *carbolic acid,* a teaspoonful to the pint, sometimes give relief. The general health should be carefully attended to.

Bran baths may be used.

Mercurius solubilis will relieve some cases. I have prescribed it solely on the itching.

Apis mellifica, rhus toxicodendron, sulphur, are other remedies that may be tried.

PSORIASIS.

(Greek, Ψωρα, the itch.)

Etiology: The cause is not known.

Symptoms: Psoriasis is a disease characterized by clearly outlined red spots which are covered with scales. These spots appear mainly on the trunk and on the extensor surfaces of the limbs. They are covered with scales that are easily rubbed off, and may be very profuse. They may be small or large, regular or irregular in outline. Usually the body is profusely covered with the eruption.

Psoriasis usually appears first in adolescence. After its first appearance it comes and goes, usually for many years. An

attack lasts for a few weeks. The intermissions may last several months. There are few or no constitutional symptoms.

Diagnosis: The leading characteristic is the definite outline of each spot. The scales are superficial, bleeding does not occur when they are removed. There is no moisture. Patches of eczema do not have a definite outline. There is a moist exudation. Dermatitis exfoliativa is general, the face included.

Treatment: A nutritious but simple diet is indicated. Bulkeley insists on a vegetable diet.

Alcoholic drinks must be prohibited.

Daily baths in warm water using plenty of soap will help to get rid of the scales.

The x-ray will clear up the eruption quickly, leaving a brownish pigmentation. The cases I have observed treated that way usually recurred in a short time.

I believe the best results are gotten with the indicated remedy.

Arsenicum album is easily first. The 6x cured one case in 1894 that has not recurred up to this writing—1914—a period of twenty years. The patient, then a young woman of twenty-eight, had had recurring attacks for years. Old school treatment by external applications and Fowler's solution had failed. The *arsenic* in potency cured.

Belladonna has cleared up some of my hospital cases. Whether they recurred or not I do not know.

RHUS POISONING.

Synonym: Dermatitis Venenata.

Definition: This is an inflammation of the skin caused by contact with the rhus plant. The irritation seems to be caused by an essential oil. Actual touching of the plant may not be necessary, as some persons are so susceptible that they are affected by air-borne particles. Other persons can handle the plant with impunity.

Symptoms: There may be a period of incubation of a few hours to a few days. There is itching, burning, sometimes a crawling sensation of the parts affected. Then the eruption

appears. It is usually not deep. There may be a simple erythema. Other cases show edema of the skin. There may be a serous infiltration, with vesicles, which later become pustular. The face and hands are most often attacked, sometimes other parts of the body. One of my patients, a young woman, must have sat on the plant because her trouble started about the genitals. She was very ill, ran a high temperature and was delirious.

Treatment: Cases tend to recover. Some persons are extremely susceptible and become infected every year.

Guernsey, of Philadelphia, says those who are susceptible should immediately after exposure wash the parts with 50 to 70 per cent. alcohol, or with ether, to dissolve the essential oil which is the cause of the trouble, as a preventive.

Keeping the inflamed parts constantly wet with a solution of epsom salts will allay the irritation in some.

Helmuth, Senior, in his Surgery, recommended *sanguinaria canadensis* as an external application, and internally as a remedy.

Rhus toxicodendron, high, will clear up some cases.

Arsenicum album has relieved cases with much redness and swelling.

Apis mellifica is indicated when vesicles form.

Sanguinaria acts well in cases with intense itching.

I have verified each of the above remedies.

ROSEOLA.

Symptoms: This is an erythematous eruption which usually covers a large part of the body. Sometimes it is little more than diffuse redness, at others it may look not unlike measles. The rash is the only symptom in most cases. The conjunctiva and throat may be a little red.

Diagnosis: I have met with the condition a number of times, usually in infants. Lack of symptoms other than the rash will help to differentiate the cases from measles and scarlet fever.

Treatment is usually unnecessary.

SCABIES.

Synonym: The Itch.

Symptoms: Scabies, the itch, is a disease due to a minute animal parasite that burrows under the skin. Frederick M. Dearborn says the gypsy moth causes some cases. Favorite locations are the arm-pits, groins, and between the fingers. The eruption produced consists of raised red marks. The intense itching and consequent scratching produce marks and excoriations which only add to the trouble. It gives a characteristic appearance to the affected part. The disease is contagious between different individuals by contact, and it is transferred from one part of the body to another by scratching.

Treatment: Most of the cases occur in uncleanly persons, although others may contract the disease. Soap and water is a good remedy. The patient should take a hot bath, scrub thoroughly with a brush, then rub in a mild sulphur ointment. This should be done three nights in succession. It must be done thoroughly else one small spot overlooked may start the trouble all over again. I have had success with the following:

℞. Sulphur præcip. .....ʒj.
Balsam Peru .....ʒj.
Adipis Petrolati aa .....ʒiij.
Mx.
Sig. Apply externally, night and morning.

Remedies cannot cure so long as the parasite persists. They may allay some of the irritation.

Croton tiglium may relieve itching when scratching is painful.

Hepar may help when the patient is very susceptible to cold.

Sepia, the itching is not relieved by scratching.

SCLERODERMA.

(Greek, σκληρος, hard.)

Definition: A localized or general induration of the skin. It may be general or circumscribed (*morphea*).

Etiology: It is more frequent in women and between the ages

of fifteen and forty-five, though no age is exempt. It is probably a neurosis. Exposure, rheumatic conditions, and thyroid disease have all been given as causes.

Morbid Anatomy and Pathology: There is thickening of the connective tissue of the skin. The subcutaneous fat atrophies. The blood vessels are diminished in caliber.

Symptoms: There is first a slight stiffness of the part affected. It is slightly swollen or indurated. There may or may not be some brownish pigmentation. Late in the disease the affected skin is hard and leathery to the touch. The induration affecting, as it does, the subcutaneous fat as well as the skin makes the part more or less immobile. There may be no subjective symptoms.

In severe cases of more acute onset the affected area may be very considerable. It is nearly always symmetrical. When it occurs in the limbs the underlying muscular tissue may become atrophied and the whole part become welded together. In these cases ulceration is apt to occur over the joints.

In the circumscribed form—*morphea*—the spots are small, at first pinkish, but eventually reaching the sclerosed condition. Sometimes the spots are like scars.

Diagnosis: The typical hardness with pigmentation is diagnostic. The edematous stage may be mistaken for myxedema. It may be confounded with angioneurotic edema. The latter presents only a transient swelling where scleroma is more or less permanent.

Prognosis: General scleroderma may be fatal. As regards a cure, any given case is doubtful.

Treatment: Build up the patient is the first need.

Continued warm baths and inunctions of oil are of great help.

Arsenicum album, or *arsenicum iodide*, would seem to be the most frequently indicated remedies.

SEBORRHEA.

(Latin, *sebum*, fat; Greek, ῥέω, I flow.)

Definition: This is an excess of secretion from the sebaceous glands.

Symptoms: In the scalp this excess dries and accumulates dust. The dried material becomes scaly and is then known as *dandruff*.

The skin may be oily or greasy. In some cases a mild inflammation, due to the disturbed function of the sebaceous glands, takes place. This is called *seborrheic eczema*. The affected areas become slightly reddened. There may be patches scattered here and there of round or irregular outline, and in size up to a half inch or more in diameter; or the patches may cover considerable areas, especially when on the forehead. Examination shows the spots to be covered with fine dry scales. When the scales are removed they leave a hyperemic base.

Next to the scalp the armpits are perhaps most often affected. Of the hairless parts of the body, the forehead and face, neck, and shoulders are most often affected. It may extend down till the entire body and legs are disfigured with patches. I believe it to be autoinfectious.

As a rule, there is very little annoyance. The spots may feel hot and burn a little. Itching is not common. Patients, particularly women, are much perturbed over the appearance, none at all over the discomfort.

Many patients are in a run down condition or are anemic.

A young married woman, aged twenty-seven, had a seborrheic eruption on both breasts, under the arms, on the shoulders and chest. It was very slight over the shoulder blades and back, and there was none at all on the throat or abdomen. There was no itching or burning, no sensation whatever. The spots were round, from one-eighth to one-half inch in diameter, very slightly raised, and dry. They desquamated leaving a red base.

Treatment: *Arsenicum album* was given internally.

Bay rum with two per cent. resorcin was used locally.

Some skins will not stand the bay rum and resorcin. Then oil may be used to soften the scales, and green soap and alcohol, half and half, to bathe the parts with.

Graphites is sometimes indicated, especially if the skin is very oily.

Belladonna may be indicated if peeling is marked.

SWEAT GLANDS.

Hyperidrosis: As has been already mentioned, the skin is kept in good condition by the secretions from the sweat and sebaceous glands. There may be an excess as well as a deficiency in these secretions. If the excess comes from the sweat glands the skin is wet and damp with perspiration. Sweats of the entire body occur in general weakness from certain diseases. Such sweats can be cured only by curing the original disease. Localized sweats sometimes occur as the result of weakness of the sweat glands themselves; for example, excessive sweating of the hands or feet. In such cases the patient should be got in good condition by correcting any errors in diet or hygiene. The offending part or parts can be stimulated by massage and by cold water baths.

Anidrosis is diminished sweat.

Bromidrosis is sweat of unpleasant odor.

SYCOSIS.

(Greek, συκωσις, fig like.)

Synonym: Barber's itch.

Symptoms: This is a disease limited, as its name implies, to the region of the beard in men. It is an acute inflammatory disease attacking the hair follicles. It is purely a local condition, although it frequently occurs in debilitated subjects.

Treatment: The general health must be improved. Shaving is usually impossible on account of the pain it causes. The hair must be kept short with scissors. A mild antiseptic wash, bichloride of mercury, one part in four thousand of water, is the best local application. Alcohol and water, half and half, may be used. Cases of sycosis are very stubborn, lasting for weeks or months. There will be frequent remissions, the patient getting almost well, then the eruption will break out again. Eventually these cases do get well. While under treatment all forms of alcoholic drinks must be forbidden.

Hepar is useful in case of pus formation.

Rhus toxicodendron is sometimes helpful where vesicles form.

TELEANGIECTASIS.

This is the name given to the little capillary dilatations that radiate off from certain skin lesions. Chronic topers with bulbous noses show it very well.

TINEA.

Synonym: Ringworm.

Definition: Tinea, or ringworm, is a vegetable parasite disease. It is contagious. It varies somewhat in appearance, according to the part of the body affected.

Etiology: Ringworm is caused by a variety of fungi. The two most common are the small spore fungus, *microsporum*, and the large spore fungus, *megalosporum* or *tricophytum*.

Symptoms and Treatment: *Tinea circinata.* On the skin tinea shows first as a number of small spots arranged in a ring. This tends to spread at the periphery, and to clear in the center. Persons of any age may develop this form. An antiseptic dressing will cure it. Applications of pure alcohol is one of the cleanest and best. Tincture of iodine may be used.

Tinea tonsurans. Ringworm of the scalp attacks only children. Broken down hairs are plentiful, leaving the affected spot denuded. This form is very stubborn and requires months of the most assiduous treatment. The x-ray has been used with some success in recent years. The patients with ringworm must be built up, for many of them are run down and debilitated.

Occasionally remedies may be of service.

Graphites may be used to induce a new growth of hair if that has been destroyed.

VERMIN.

Bed Bugs: Bed bugs are found in beds. If they once get a foothold they get in the wood work of a room, in the picture moulding, on the backs of the pictures, in chairs, and so on.

The bite of a bed bug makes a small rounded elevated spot. If such spots are found on the body search for bugs. The mark does not last long. Thorough cleanliness is the only way to get rid of the bug. Hospitals wash their beds with a poisonous solu-

tion of carbolic acid or of bichloride of mercury. The washing is repeated frequently.

Fleas: Fleas usually come from house pet animals.

The flea bite is a very small hemorrhagic spot.

Pet animals should be frequently bathed to rid them of fleas.

Pediculosis, or lice: These are small vermin somewhat resembling crabs (their vulgar name) in appearance.

Children often pick up scalp lice, *pediculosis capitis,* in school. Careful attention should be given to a child's head to keep it clean. When lice are present they tend to cling to the roots of the hairs where they can feed on the scalp.

Kerosene will cause them to quit. The so-called blue ointment, a poisonous mercury preparation, thoroughly rubbed in, will kill them off. Care must be used not to get it—the ointment—in the eyes or mouth. It is very irritating and extremely poisonous.

Body lice, *pediculosis corporis,* or *vestimenti,* live in the clothing, only going on the skin to feed. Clothing should be fumigated. Blue ointment may be used on the skin in limited areas if necessary.

Pediculosis pubis are usually found in the hair about the genitals. Treatment is the same as for other forms.

Nits—the eggs of lice—show as white spots on the hair shafts. Vinegar will remove them.

WARTS.

Synonym: Verrucæ.

A wart is a small growth developed from the papillary layer of the skin.

Warts vary much in size. They occur mostly in youth and early adult life. The favorite locations are the face and fingers.

Very small ones in great numbers sometimes appear on the neck about the edge of the collar.

The knife or the cautery or caustic is the best treatment.

Pure *nitric acid* is a good caustic.

Thuja is said to cure them.

WENS.

Synonym: Steatoma.

Wens are tumors developing under the skin. They usually start from a plugged gland.

Wens are found most often in the scalp. They are usually harmless and cause no discomfort. Occasionally one will develop large enough to cause discomfort by its size. The knife is the proper remedy.

SECTION XII.

Diseases of the Ductless Glands.

STATUS LYMPHATICUS.

Synonyms: Lymphatism. Status Thymicus.

Definition: A rare condition found mostly in children and young persons, characterized by hyperplasia of the lymphatic glands, the thymus, the spleen and the bone marrow. Sudden death appears to be common.

Historical Note: It has been known for a long time that an enlarged thymus gland is the chief discoverable lesion in many cases of sudden death. Various reasons were assigned for this. In 1889 Paltauf stated that the enlarged thymus in these cases was merely a manifestation of a general enlargement of all the lymphoid tissues of the body. In consequence there was a constitutional weakness that accounted for all the symptoms.

Etiology: The cause is not known.

Morbid Anatomy and Pathology: All the lymphoid structures are enlarged. The bronchial and peritoneal glands, the tonsils and the spleen show this enlargement as well as the thymus. The lymphoid bone marrow is red. The subjects show infantilism.

Dr. L. R. Kaufman reported a case in the *Chironian* for August, 1911.

Hawley reports a case in the *Lancet-Clinic,* August 14, 1914.

Symptoms: These are altogether vague and uncertain and very often are only realized after death. Babies may be rickety and subject to laryngismus. Attacks of dyspnea are common. There may be evidences of enlargement of the lymphoid structures.

The enlarged thymus may sometimes be percussed as a triangular area of dulness over the upper part of the sternum which moves upward when the head is retracted (Bogg's sign).

Very slight causes end the lives of the patients. Deaths may occur under anesthesia, on taking a cold bath, from injection of antitoxin, and so on.

Diagnosis: This is rarely made before death because there are no definite symptoms to call attention to the condition.

Prognosis: It is probable that all cases of status lymphaticus do not die from it. But as the symptoms are not definite and diagnosis in life not certain the prognosis is not known.

Treatment: None known. The x-ray is said to be of some service.

GOITRE.

(Latin, *guttur*, throat.)

Definition: This is a simple enlargement of the thyroid gland.

Etiology: The cause of simple enlargement is obscure. It is a not uncommon condition in young girls. In some women there is a transient enlargement during menstruation.

Simple goitre seems to be endemic in the State of Washington. In *Northwest Medicine* for July, 1914, Dr. D. C. Hall, of Seattle, reports on the examination of 3,339 students at the University of Washington. Of 2,086 men examined 374 or 17.94 per cent. had enlarged thyroids. Of 1,253 women 388 or 30.94 per cent. were affected.

Symptoms: In simple enlargement of the thyroid there may be no symptoms except the enlargement itself. The physician's advice may be sought because the patient does not like the appearance of the swelling. In other cases there is a soreness of the gland. One patient complained of a "crowding" sensation in the throat on speaking or eating. In her case the swelling was quite pronounced during the menstrual period. Between times it was hardly noticeable. I have seen a number of similar cases.

Diagnosis: This is made on the appearance of the swollen gland, together with an absence of other symptoms.

Prognosis: The prognosis is good. I have several women patients treated twenty or more years ago for simple goitre, who were relieved of the swelling at that time and have had no return.

Treatment: The patient should live a quiet, regular life, cutting out too much dancing and late hours.

Iodium 6x has been almost the only remedy used. It is almost a specific.

EXOPHTHALMIC GOITRE.

(Greek, ἐξ, ὀφθαλμος, out, the eye.)

Synonyms: Parry's Disease. Graves' Disease. Basedow's Disease.

Definition: Exophthalmic goitre is the name given to the disease characterized by acceleration of the pulse, protrusion of the eyeballs, and enlarged thyroid.

Historical Note: Exophthalmic goitre was first described by Parry in 1786, by Flajani, of Italy, in 1800. Graves redescribed it in 1835, and Basedow in 1840.

Etiology: Exophthalmic goitre occurs much more often in women than in men, and between the ages of twenty and thirty.

Heredity seems to play a part, as it is often seen in several members of the same family. One patient of mine died of it at fifty-four, her mother had died of it at sixty-two, a younger sister now has it. It is considered a neurosis by some, and is so classified by Strümpell. By others it is considered as primarily a disease of the thyroid gland, hyperthyrea, the direct antithesis of myxedema, or athyrea. It occurs more often in families with nervous disorders, as epilepsy, hysteria, and the various psychoses. Severe nervous or emotional shock often seems to be the starting point.

Morbid Anatomy and Pathology: General emaciation is common. The thyroid is enlarged and also the nearby lymphatics. The superficial blood vessels of the thyroid are dilated. The tissue is firm. Microscopically, there is an increase in the secreting part of the gland. The thymus gland can also be found.

Symptoms: The increase in the pulse rate is the most constant and often the earliest sign. It may be anywhere from 100 to 140 or even 160 per minute. There is no characteristic change in the quality of the pulse, other than its rapidity. There is no characteristic change in the heart. Hypertrophy has been

observed in some cases, dilatation in others. Palpitation is very often present, sometimes a persistent angina pectoris. The carotid pulsations are often visible in the neck.

The protrusion of the eyeballs is another characteristic symptom. This may be slight, or it may be quite marked. Even when slight it may give a peculiar staring expression to the eyes. The exophthalmos is always bilateral, although one eye may appear to protrude more than the other. The protrusion varies from time to time.

There are several peculiar signs usually present that should be remembered. (1) Stellwag's sign. This is an increase in the palpebral fissure due to retraction of the upper lid. (2) Graefe's sign. On lowering the eyes the upper lid lags in following the eye downwards. (3) Möbius' sign. In converging the eyes on a near point, one eye soon turns outward.

With the ophthalmoscope the retinal vessels may be seen pulsating.

The enlargement of the thyroid, like the exophthalmos, varies from time to time. It may be much or little. It is soft and can be felt to pulsate. Sometimes loud vascular murmurs can be heard over it. The enlargement may increase after excitement or during the menses.

Tremor is another symptom frequently found. It may be of the extremities only, or it may include the whole body. Excitement makes it more marked. Besides the tremor there is an irritability of temper. There is a haste in moving and speaking. The patient flushes and perspires easily. There is lack of attention. Sometimes complete alienation.

The respirations may be increased and the patient suffer from shortness of breath. Sometimes there is a nervous cough.

Quite often there is some degree of fever.

The skin often shows changes in pigment. There may be vitiligo or there may be chloasmic spots. Itching may be a troublesome symptom.

The appetite is capricious. It may be ravenous at times. Paroxysmal vomiting and diarrhea may occur. Frequently patients become much emaciated and anemic. The menses may be irregular or absent.

Complications and Sequelæ: Are not common. Other neuroses and psychoses may occur. Myxedema may be a sequel. Acute dilatation of the heart ended the life of one of my patients.

Diagnosis: In a marked case is easy.

Cases existing without exophthalmos may be more difficult. As most cases are chronic in their course the history must be taken into account. An excessively rapid pulse, long-continued, and without obvious cause should always excite suspicion.

Prognosis: A few cases recover, most do not. The younger the patient the better the outlook. Relapses may occur.

Treatment: The patient with exophthalmic goitre should be placed under the best possible hygienic surroundings. Rest of mind and body is necessary, especially late in the disease.

Hector Mackenzie recommends the following diet: Very little milk, exclude meat. Allow fish, bacon, chicken, eggs, vegetables, salads, fruits, cream, sugar, butter, bread, carbohydrates.

Rodagan, milk from goats whose thyroids have been removed, is recommended.

Strümpell suggests galvanism of the neck. He also claims to have had good results from *iodide of sodium* and from *ergotine*.

Sometimes removal of the thyroid gland in whole or in part gives good results. Or the cervical vessels may be ligated.

Iodium seems to be of value in some cases.

Spongia is preferred by Hale.

Belladonna is recommended by physicians of both schools.

Glonoin is useful in cases in threatened cardiac failure. It should be used in full doses.

Spigelia has many of the cardiac symptoms of exophthalmic goitre.

Lycopus in five drop doses of the tincture is recommended by Hale. Dr. James D. Miller has used it with success in a number of cases.

Aconite in full doses is recommended in the 1912 edition of Osler.

CRETINISM.

Synonyms: Cretinoid Idiocy. Infantile Myxedema.

Definition: Cretinism is the name applied to the set of symptoms produced by congenital absence or loss of function of the

thyroid gland. There is arrested development of both body and mind.

Historical Note: In certain parts of Europe cretinism is endemic, and a large proportion of the inhabitants are affected. The cretins from these districts have been known and described for years. It was only after the studies of Gull, in 1875, and of Ord, in 1879, became known that cases of sporadic cretinism, or the myxedema of childhood, came to be recognized. Before then these cases were classified as cases of idiocy.

Etiology: Cretinism is due to congenital absence of the function of the thyroid, and consequent lack of mental and physical development shows itself in early childhood.

Morbid Anatomy and Pathology: Goitre is present in sixty per cent. of the cases of endemic cretinism. In sixty cases of sporadic cretinism collected by Osler goitre was present in only seven per cent. In a case of mine the thyroid appeared to be absent. When goitre is present the swelling is evidently in the connective tissue and at the expense of the glandular substance.

Barker, of Johns Hopkins, examined a case and found a marked increase in the connective tissue separating the lobules of the thyroid gland. The microscope showed the acini to be separated from one another. Some were solid, some seemed to be replaced by cysts. The lumina of the acini varied in size; the epithelia were unrecognizable. The bones, especially the long bones, show the arrested growth.

Symptoms: In sporadic cretinism, the only kind to be found in the United States, the child is usually normal till two years of age or more. Then the head continues to enlarge while the rest of the body retains its infantile shape. The belly becomes protuberant, the spinal curves are exaggerated. The legs are bowed and thick. The head hangs down; the mouth is open; the lips and tongue are thick, the latter protruding and the saliva runs away. The skin is dry and harsh; the hair coarse and usually brittle and scant; the teeth are bad. The patient is very sensitive to cold. The face is expressionless, stupid, and the child does not learn to walk or talk, or may be able to walk only a little and to use a few words. The sexual organs are undeveloped.

The patients are apathetic and sleep a good deal. Other cases, of course, are more intelligent and further developed.

In the *New York Medical Journal* for February 22, 1902, I reported at length on a case of cretinism that had improved a good deal under treatment. The girl had had thyroid treatment before she came to me. When I first saw her she had been without it for two years, during which time she retrograded:

"When the patient came to me she was twenty-five years and two months old. She measured 53½ inches in height, and was rather stout. The features were small, but not clean cut; the forehead was low; the mouth rather large, showing the teeth regularly placed, but not in very good condition; the nose broad. The base and back part of the skull seemed well developed. The skull sloped forward to the low forehead; the hair was black (Rotch says the hair is usually light), coarse, and not in good condition; the eyes and complexion dark. She walked rather slowly and heavily. In going up and down stairs she went one step at a time, always putting the same foot forward, like a child too small to use one foot after the other. She menstruated irregularly. Her mental equipment was like that of a child of six or seven, without the eagerness to learn the why of things that a normal child develops. She appeared to be very shrewd and let no remarks escape her. She was very sensitive, self-willed, and irritable. Her mother told me that she had violent fits of temper. She never became violent in actions, although she did in her language.

"In watching her I found her clever in some things. For example, one day her mother took her to a dentist. She fussed around his office for four hours, but would not let him look at her teeth. She said she had perfect confidence in the dentist, but none in herself. I found her extremely egotistical; she was all self. She suffered somewhat with her stomach, complained of cramps and heartburn. Occasionally she had a vomiting attack."

This patient grew an inch in stature and became more tractable. I followed her for about seven years when she dropped out of sight.

Diagnosis: As a rule, is easy. Other forms of lack of mental development do not exhibit the physical characteristics of cretinism. Rickets may simulate the physical symptoms, but the child is, as a rule, bright mentally.

Prognosis: Before the use of the thyroid gland in treatment cretins rarely lived to be over thirty. Now, with the use of the thyroid, life may be prolonged indefinitely.

Treatment: It goes without saying that the best surroundings possible should be provided for the patient.

There was no known cure, however, till Murray suggested the use of the thyroid gland in myxedema in 1891. The use of thyroid in cretinism followed.

Thyroid extract, thyroid gland, or liquor thyroidei, may be used. It is best to begin with a small dose. Young cretins may be benefited or made normal. Older ones may be benefited. Treatment must be continued throughout life. A dose of one grain three times a day is enough to begin with. Some patients can stand as much as five grains three times a day. That is too large a dose, however, to begin with.

The particular preparation does not seem to matter, although I used *iodothyrine* of the Farbenfabriken of Elberfeld Company, in one case with marked benefit.

In beginning any form of thyroid treatment the patient is best kept in bed for a time. The treatment is contraindicated in irritable or weak heart. If there is marked increase in the pulse rate, or rapid loss of weight, reduce the dose. If diarrhea supervenes, stop the remedy. In cases where the thyroid treatment does well an occasional intermission of a week or two seems to be of benefit.

MYXEDEMA.

(Greek, μυξα, mucus; οιδεω, to swell.)

Synonym: A Cretinoid State Supervening in Adult Life.

Definition: Myxedema is the name applied to the set of symptoms produced by loss of function of the thyroid gland in adult life. There is an infiltration of the connective tissue, first noticeable about the face and upper part of the body, and gradual impairment of the mental faculties.

Historical Note: In 1875 Sir Wm. Gull described a "cretinoid state" in adults. In 1879, Ord, who had made a study of this condition, named it "myxedema." In 1892 Kocher described the "Cachexia strumipriva" a cretinoid condition developed after removal of the thyroid gland.

Etiology: Myxedema is due to loss of function or extirpation of the thyroid gland. It occurs from disease of the gland, more often in women than in men, about seven to one.

Morbid Anatomy and Pathology: The thyroid is reduced in size, pale and tough. The alveoli disappear, and there is increase in the interstitial tissue. The skin shows some changes, otherwise there is nothing characteristic. Murray, in the Johstonian lectures in 1900, gives the symptoms developed in monkeys after extirpation of the thyroid and parathyroid glands. Nervous symptoms appeared after five days. There was a fine regular tremor followed by progressive muscular atrophy. There was loss of curiosity and interest in surrounding objects and great irritability of temper. There was loss of activity and of muscular power; contractions followed, due to clonic spasms of the flexor muscles. The gait became stiff and unsteady, with a tendency to falling backward. True epilepsy appeared. The temperature was at first increased and later became subnormal. In two or three weeks myxedema became distinct, most so in the face. The skin became dry and rough. The red corpuscles diminished, the white corpuscles increased.

Symptoms: Myxedema is insidious in onset when the result of disease of the thyroid. After extirpation the symptoms may set in rapidly.

There is at first a disinclination to do things, and the patient is conscious that more effort than usual is required for the ordinary affairs of life. Then the subcutaneous tissue becomes infiltrated and swollen. The face becomes distorted and expressionless. The hands become large and clumsy. The whole body becomes enlarged. In consequence of the general swelling the finer movements of the joints are interfered with.

The skin becomes dry and scales easily. The hair falls out. The nails become cracked and discolored.

The thyroid becomes diminished in size.

The temperature is inclined to become subnormal. The hands and feet are cold and the patient is susceptible to cold.

There is gradual impairment of the mental faculties. The patient thinks more and more slowly. She may progress to true dementia. She is suspicious. She may have hallucinations.

Sight and hearing may be affected.

Loss of appetite and constipation are common. Other symptoms are not characteristic.

Diagnosis: Myxedema may be mistaken for Bright's disease. Examination of the urine will differentiate.

Obesity will not present the same mental symptoms.

Prognosis: The disease used to be slow and progressively fatal. Now the prognosis is good under thyroid treatment.

Treatment: Murray, who first suggested the use of thyroid gland in mxyedema, says the treatment may be divided into two stages: (1) To remove all symptoms; (2) to maintain the patient in health. He advocates either the liquor thyroidei or the dried gland. He prefers the liquid, but says it must be made up fresh every two weeks. He begins with five minims of the liquor, or two grains of the powder each night at bedtime. This is gradually increased up to ten or fifteen minims unless the pulse is unduly accelerated, and then maintained until the symptoms disappear. Then, usually, a smaller dose will suffice to keep the patient in health. Treatment must be continued throughout life.

TETANY.

(Greek, τανω, to stretch.)

Synonyms: Tetanilla. Intermittent Tetanus.

Definition: Paroxysmal tonic spasms, usually painful, in symmetrical and bilateral groups of muscles, usually the extremities.

Historical Note: The name was originated by Corvisart.

Etiology: By some the condition is considered to be infectious. It is occasionally epidemic. It may follow the infectious fevers. It is sometimes the result of catching cold. It

may follow pregnancy, the debility of lactation or of diarrhea. It follows removal of the thyroid gland, probably because the parathyroids are removed at the same time.

Children and young adults are most often affected. In children it is usually associated with rickets.

Morbid Anatomy and Pathology: The disease is now considered to be due to diminution or loss of function of the parathyroids.

Symptoms: There may be prodromal symptoms of general discomfort, weakness and stiffness. The spasms usually begin in the fingers and arms, then go to the toes and legs. The flexor muscles are the ones affected. The condition is almost always bilateral and symmetrical. The fingers become fixed as though holding a pen; the wrists and elbows are flexed; the arms folded over the chest; the toes are flexed and the feet extended. Sometimes the jaw and the muscles of the trunk are set. While the spasm lasts the muscles are hard and sensitive to touch, and there is pain. The condition may last from a few minutes to hours, possibly days. The paroxysms are repeated for days or weeks. I have a patient who suffers from these contractions, sometimes for days at a time, usually in the hands only. Once she was confined to the bed for several weeks, the hands, feet and trunk all being affected.

The reflexes remain normal. There is no disturbance of consciousness.

During the attack there is an abnormal sensibility of the system, and the spasms may be caused by compression or tapping the affected parts. This is known as *"Trousseau's symptom."*

The disease is apt to recur.

Diagnosis: As a rule, the diagnosis is not difficult. Hysteria may be much like it sometimes, but the spasms are more often unilateral. Multiple neuritis has more chronic spasms. Tetanus always has trismus.

Prognosis: This is usually good. An occasional death results from spasm of the diaphragm.

Treatment: The cause must be eliminated if possible.

The patient should be given simple but nourishing food.

Rest is essential.

Osler recommends *calcium lactate*, fifteen grains every three or four hours. He also reports good results with *thyroid extract*.

Aconite has helped in at least one case.

ADDISON'S DISEASE.

Definition: A constitutional disease characterized by asthenia, feeble circulation, gastric irritability, and pigmentation of the skin.

Historical Note: Under date of May 21, 1855, Dr. Thomas Addison, of London, published an original communication, entitled "On the Constitutional and Local Effects of Disease of the Suprarenal Capsules." In this paper Addison said that he had observed at various times a number of cases that were anemic, but that gave a history of none of the ordinary causes of anemia; the patients became extremely weak but did not lose flesh; they also presented a peculiar pigmentation of the skin. Addison called these cases "Idiopathic anemia." Necropsies made on a number of them gave but one common lesion, namely, disease of the supra-renal capsules. The original paper detailed the histories of eleven cases. They formed a new symptom-complex to which Trousseau gave the name "Addison's Disease." In 1875 Greenhow, in the Croonian lectures for that year, made an exhaustive study of the disease, presenting histories of nearly two hundred cases.

Etiology: Addison believed that anything that caused interference with the function of the supra-renals would cause the symptoms described by him. Others have thought that the symptoms were produced through disturbance of the sympathetic nervous system. Rolleston sums up the various theories and says: "Addison's disease is due to inadequacy of the chromaffin or adrenalin secreting cells which are chiefly situated in the medulla of the gland, but are also found in connection with the sympathetic trunks. It is possible that there is a second factor at work, namely, irritation of the sympathetic nerves, which might be, (1) mechanical and due to adhesions and inva-

sion of the pericapsular nerves and ganglia, or (2) toxemic and due either to failure of a hypothetical antitoxic function of the cortex of the adrenals, or to disturbed metabolism resulting from absence of adrenalin."

The disease is very rare. I have seen but one case and that at the Metropolitan Hospital. There was but one other case there between 1890 and 1914. It occurs more often in men according to some authorities, more often in women according to others. Adults about thirty are most often affected.

Morbid Anatomy and Pathology: The essential lesion is change in the supra-renals, most often tubercular in character. There may also be hypertrophy of the lymphoid structures. Pigment exists in the skin, and often in the mucous membranes. The heart is small.

Symptoms: The onset of Addison's disease is insidious. The patient gives a history of a gradual loss of strength, as a rule. The pigmentation of the skin may precede or follow this decline.

The most striking symptom connected with a fully developed case of Addison's disease is the pigmentation. This may vary all the way from slight exaggeration of the normal pigment on the exposed surfaces of the body to a deep discoloration of the whole surface. As a rule, the mucous membranes are also pigmented. Greenhow was of the opinion that the latter was not an essential symptom, but was due to local irritation.

Asthenia is always a marked symptom. It was well defined in my patient. He entered the hospital May 1, 1900, and had to take to his bed shortly after. He did not die till September 13th. He died from exhaustion.

Associated with the general weakness is usually found a weakened heart and low blood pressure. This was so in my case. The heart sounds were very feeble, so much so that it was difficult to tell whether or no they were otherwise normal. After death the heart was found to be soft and flabby, but all the valves were perfect. The pulse rate was about one hundred throughout.

The temperature of Addison's disease is usually subnormal. In my case it was normal during May and June. In July and August it ranged from 100° to 103° F. For the last two weeks

of life it dropped to normal again, and sometimes as low as 98° F.

At all times there was more or less gastric disturbance. The appetite was fair. Constipation was marked toward the end. There was much gas and much distress from it. These symptoms are quite characteristic.

There is depressed vitality. The sight and hearing may be dulled. The knee jerks are usually diminished. There may be twitching or rigidity.

Anemia is a frequent symptom. The red cells are usually diminished.

Complications and Sequelæ: Complications are not frequent. My patient had an interstitial nephritis, diagnosed before death and verified at the autopsy. Other similar cases have been reported.

Pulmonary tuberculosis is sometimes associated with Addison's disease.

The pleura and peritoneum were inflamed in my case.

Also edema was a marked symptom late in the disease, probably due to the weakened heart.

Diagnosis: In hospital practice the most frequent condition to be differentiated from Addison's disease is what Greenhow calls *vagabond's disease*. This latter occurs in poorly nourished individuals, and the pigmentation is due to irritation by body lice.

The pigmentation of abdominal diseases and of pregnancy can be differentiated by the other symptoms.

Continued use of arsenic may produce pigmentation.

Prognosis is bad. Whether cases are ever cured or not is a question. The disease usually extends over several months, rarely more than two years.

Treatment: Consists in making the patient as comfortable as possible, and in trying to build him up with proper food.

Supra-renal extract has been used with uncertain results.

Homœopathically, *arsenic* and *argentum nitricum* appear to be the remedies most often indicated. I used *arsenic* in my case.

OTHER DISEASES OF THE SUPRARENALS.

The supra-renals are subject to the various degenerations, as fatty, tuberculosis, hypertrophy, hypoplasia, etc., but they have no special characteristic disease entities. They may also harbor carcinoma. sarcoma, etc.

DISEASES OF THE SPLEEN.

An **enlarged spleen** accompanies many of the acute infectious diseases. In such cases the enlargement usually subsides when the patient gets well. In malaria, in syphilis, and in lardaceous disease it may become very much enlarged and remain so.

In all cases presenting chronic enlargement of the spleen the differential diagnosis is very difficult. It is only after the most rigid investigation into the patient's history and physical condition that a diagnosis of splenic leukemia (see page 294) or splenic anemia can be made by exclusion. These terms simply mean enlarged spleen without known cause. In splenic leukemia there is increase of leucocytes and presence of myelocytes. In splenic anemia red cells and leucocytes are both reduced.

Rolleston (in Allbutt's System of Medicine, 2d edition) advances the theory that some "unrecognized parasite, possibly from the alimentary canal, has reached the spleen, and there remained to multiply and cause splenomegaly and anemia."

I have seen two cases of splenic leukemia—at least they were diagnosed as such—in my service at the Flower Hospital. So far as I know I have never seen a case of splenic anemia.

Splenectomy is indicated in splenic anemia; it is fatal in splenic leukemia.

SPLENIC ANEMIA.

Synonyms: Banti's Disease. Splenomegaly with Anemia.

Definition: Osler calls it "a primary disease of the spleen of unknown origin, characterized by progressive enlargement, attacks of anemia, a tendency to hemorrhage, and in some cases a secondary cirrhosis of the liver, with jaundice and ascites."

I am indebted to Osler for what follows:

Historical Note: Banti made the first exhaustive study of this condition in 1883.

Etiology: Enlargement of the spleen takes place without known cause.

Morbid Anatomy and Pathology: The spleen is enormously enlarged and fibroid in character. The neighboring blood vessels are distended and show atheroma.

Symptoms: The spleen is enormously enlarged, sometimes without other symptoms. Later anemia develops, as shown by pallor and dyspnea.

Blood examination shows a leucopenia, the red cells may drop to 2,000,000. The leucocyte count is normal or diminished.

There is a tendency to hemorrhage from the stomach.

Complications and Sequelæ: The disease runs many years and may end in ascites. There may be jaundice and cirrhosis of the liver.

Diagnosis: Splenic anemia must be differentiated from other forms of enlarged spleen. The history of the case is the main guide, other forms are secondary, splenic anemia is primary.

Prognosis: The disease lasts a long time but usually ends in death.

Treatment: Complete removal of the spleen has resulted in cure.

WANDERING SPLEEN.

Synonym: Movable spleen.

Definition: A spleen that is unduly movable.

Etiology: Is most often found in women whose other abdominal organs are displaced somewhat.

Occasionally it is caused by a blow.

Morbid Anatomy and Pathology: The spleen is enlarged. The pedicle may be twisted.

Symptoms: Sometimes movable spleen exists without symptoms, and is only discovered by accident. Usually the patient has a heavy or dragging sensation in the back and abdomen. The displaced organ may pull down the other viscera and cause gastric disturbances. Sometimes very acute and alarming symp-

toms are produced by a twist in the pedicle. Physical examination will find the spleen abnormally placed.

Complications and Sequelæ: A twisted pedicle may cause thrombosis, necrosis, and peritonitis, or engorgement and rupture may take place.

Diagnosis: The diagnosis is made by finding the displaced organ. If it has become fixed by adhesions in an abnormal position the diagnosis may be difficult.

Treatment: A band may be used to try to hold the organ in place. Surgical interference may be necessary, either fixation or complete removal. Splenectomy, however, is comparatively safe. Three deaths in forty-three cases reported by one operator.

OTHER DISEASES OF THE SPLEEN.

We may have: (1) Capsulitis; *Ceanothus* is indicated in this condition; (2) chronic venous engorgement; (3) hemorrhage; (4) abscess, usually secondary; (5) tuberculosis, part of a general tuberculosis; (6) syphilis may affect it; (7) lardaceous disease; (8) cysts; (9) new growths; (10) infarct. This last is not uncommon in heart disease. I have seen a number of cases at the autopsy table. Sudden acute pain is one of the symptoms. (11) Rupture. None of these conditions require extended comment.

ACROMEGALY.

(Greek, ακρος, an extremity; μεγαλος, large.)

Synonyms: Marie's Disease. Pachyacria.

Definition: A nutritional disease characterized by enlargement of the bones and overlying tissues, chiefly of the face and extremities. It is due to perversion of secretion of the pituitary gland.

Historical Note: Marie was the first to clearly describe the condition.

Etiology: The disease occurs more often in women than in men. Persons of large size are the ones usually affected. There is disturbed function of the pituitary gland. In a case reported by Grinker the trouble was due to a tumor of the hypophysis.

Morbid Anatomy and Pathology: There are changes in the pituitary gland and in the bones of the face; frequently in the hands and feet.

Symptoms: There is a gradual enlargement of the features, then of the hands and feet. The condition may become quite marked before it is recognized. The eyelids and ears are enlarged. The hands and feet are big.

Kyphosis may develop later.

There is headache, sometimes disturbance of vision. The patient is irritable and cross.

Complications and Sequelæ: Diabetes. Goitre.

Diagnosis: The diagnosis is made from the appearance of the features.

Prognosis: The prognosis is bad.

Treatment: Glandular therapy may be tried.

Cushing, formerly of Johns Hopkins, now of Harvard, has done some remarkable surgical work on the pituitary gland and has added much to our knowledge of its physiology and pathology.

INFANTILISM.

This is the name given to lack of mental and physical development. There are many types and many causes. As the internal secretions are becoming better understood, conditions of infantilism are supposed to be due to some fault in them. The pituitary gland is found to be an important regulator of development. As acromegaly is known to be due to hyperpituitarism, so infantilism is supposed to be due to hypopituitarism.

SECTION XIII.

Mental Diseases and Psychoses.

MENTAL DISEASES.

It has always seemed to me as though a brief consideration of mental diseases, or, rather, an outline of the various abnormal states of mind, should be included in a work on the practice of medicine. Many physical ailments are accompanied by delirium, which is a temporary mental aberration; to distinguish the temporary from the permanent it is well to have a general idea of the whole subject. Other diseases permanently affect the mind, after a longer or shorter time, and the physician should know when that time comes.

A correct statement of subjective symptoms on the part of the patient depends on his mentality. A correct interpretation of what the patient says depends on the experience and judgment of the physician. For these reasons, therefore, this section will be devoted to a consideration of mental abnormalities and their underlying causes.

SYMPTOMS OF MENTAL ALIENATION.

Symptoms of alienation may be grouped as follows:

I. *Impairment of the higher cortical functions. Delusions.* The patient shows defects in judgment and intellect. These defects are of slow development and eventually result in fixed and insane ideas or delusions, usually on some particular subject. Otherwise the mental processes may be normal.

II. *Anomalies in the intensity and direction of the mental process.* These are shown by disorders of attention. The patient may be unable to fix the attention on the matter in hand no difference what it is, or the patient may be unable to shake off some particular idea. This last is usually some form of de-

pression, fear of death, for instance, which constantly obtrudes itself on the consciousness.

III. *Disorders of sensation. Hallucinations.* The patient has imaginary subjective sensations, hallucinations or illusions. One or more of the senses are at fault. He sees imaginary things, he hears sounds that do not exist, he feels things that are not present; more rarely he may have sensations of smell or taste that have no basis in fact. These things are real enough to the patient, but they have their origin in a disordered mind.

IV. *Disturbances of consciousness.* These may vary from slight stupor to complete loss of consciousness. This includes automatism—the doing of an ordinary act unconsciously. I have seen a patient, under severe mental stress, walk automatically, oblivious of danger when it existed.

V. *Disturbances of association.* Connected thought is impossible. The flow of ideas is incoherent and unrelated. There are anomalies of memory. Ideas are distorted. Disorientation—loss of sense of time and place—occurs.

VI. *Disturbances of volitional processes.* These involve mental reflexes, the patient is unable to carry out his intended thoughts or movements. Tics become pronounced. Stereotyped movements—movements constantly repeated—interfere with volition. Negativism, an irrational resistance to external influences, a purely mental condition, may ensue. The patient has obsessions and is liable to act on sudden impulses without reason.

VII. *Disturbances of the emotions.* The patient is subject to moods, and is gay or sad, elevated or depressed, excited or quiet, without adequate cause. The emotions are easily aroused and the result is out of all proportion to the cause.

VIII. *Anomalies of conduct.* These include moral insanity in whatever form; addiction to drugs, alcoholism, sexual perversions, and so on.

CLASSIFICATION AND CAUSES OF MENTAL DISEASE.

For this section I have adopted a modified Kraepelin classification according to Paton. Some of the diseases with psychoses are spoken of in detail in other sections of the book.

GROUP I.
MENTAL DISEASES CAUSED BY DEFECTIVE DEVELOPMENT.

The diseases of this group may be congenital or acquired.

IDIOCY AND IMBECILITY.

Definition: A condition of mental deficiency usually accompanied by physical degeneracy is called idiocy or imbecility. Idiocy and imbecility differ only in degree.

Etiology: Congenital or due to causes operating soon after birth. Of prenatal causes the most frequent are a family history of phthisis, of insanity, of neuroses, of intemperance, of syphilis on the part of the parents; of accident or shock to the mother during pregnancy. Other cases may be traced to injury due to prolonged labor or to forceps delivery. After birth the acute infectious diseases appear to be responsible for most cases.

Morbid Anatomy and Pathology: There may be atrophy of the brain associated with defective development. There may be hypertrophy of the brain, usually of the white matter, with lack of surface development. The brain may be sclerosed, in whole or in part, or it may be softened. Sometimes there is great excess of fluid, almost like hydrocephalus. There may be tumors in the brain substance. The meninges may be affected. The fontanelles may close too soon and the brain-case ossify in such a way as to prevent brain development.

Whatever change takes place in the brain or its membranes may extend to the cord as well.

Usually there is a disproportion between the head, trunk and extremities. The teeth are irregular. There are various anomalies of development of other parts of the body.

Symptoms: Idiocy and imbecility differ only in degree. There are many types laid down in the special text-books on mental diseases, but the division is more physical than mental.

The expression shows more or less lack of intelligence. Some patients will have a constant drooling from the mouth, thus adding to the silly appearance.

There is lack of attention. Sight and hearing may be defective, or the patient too stupid to use his eyes and ears. A lack of co-ordination of the muscles involved in speech will change both the character of the voice and the articulation. The patient may be able to talk a little, or words may be altogether unintelligible. Some are dull and listless, others are excitable and constantly in motion.

They are emotional, and laugh, cry or become angry without adequate reason. Milder cases are imitative. Some are easily influenced by music. They may become much attached to those who care for them, although their unstable dispositions make them totally unreliable and subject to violent outbursts of temper even against those who can ordinarily control them. Bad cases are liable to be unclean about the person. They may also be totally unmoral.

These patients show a shuffling, shambling gait. They are also clumsy with the hands.

As a rule, the skin is dry and harsh and the hair coarse. The circulation is bad and the hands and feet cold.

A large number suffer from epilepsy.

Complications and Sequelæ: Idiots and imbeciles are prone to respiratory diseases. Many have convulsions.

Diagnosis: The diagnosis is easy.

Prognosis: In mild cases something can be accomplished by long continued and skilfull training. As a rule, most of them die early of some acute disease.

Treatment: Bad cases had best be put under institutional care, the home is not the proper place. I was called in once to see a big, physically, powerful boy of eighteen, of well-to-do parentage, who was kept in an iron cage built into the house. He was very violent at times, so much so that his mother strapped his arms and legs to a chair before I was allowed to examine him. At the termination of my brief visit I left the cage first; then his mother loosened the straps and fled just in time to escape the crash of the chair hurled after her. A patient like that could not help but be a never ceasing source of depression and grief to the family; he had no place in the home, he should have been in an asylum.

Mild cases, with some intelligence, if kept at home, require the best of hygienic treatment; fresh air, moderate exercise, and easy tasks and amusements. Careful training in the care of the body as to cleanliness and neatness should come first. Then, if possible, the patient should be trained to do some light and useful work.

In certain defects surgical interference may be necessary. It is a question if medication is of any service except for intercurrent conditions.

MENTAL DEBILITY.

This condition differs from the above in that the patients may be bright in some one thing though below normal in most of their mentality. They may be clever at figures, or they may have great musical talent, or be skilled in some other one thing but deficient otherwise. Such patients are egotistical, given to excesses and lack concentration.

Here, again, good surroundings and patient training may do much.

MORAL INSANITY.

The victim of moral insanity lacks stability. He is unable to forego the gratification of his desires at the present moment, without regard to the future or to its effect on others. He does whatever appeals to him at the moment. The spendthrift, the gambler, the drunkard, the seducer, all are morally insane to the extent that they injure others regardless of consequences. Their inhibitory power is lost or in abeyance.

Development of will power is the only cure. Treatment must be psychological. The patient must be given a normal trend of mind.

GROUP II.

AUTOINTOXICATIONS.

This group comprises the manifold mental abnormalities produced by many physical diseases which are treated of elsewhere in this volume.

FEVER DELIRIA OF ACUTE DISEASES.

Any disease causing fever may cause delirium. For example, typhoid fever and pneumonia frequently have delirium as a symptom. Severe cases of measles, rheumatic fever, scarlet fever, and other of the infectious diseases, may have delirium. The delirium may be due to an overwhelming toxemia, or it may be due to idiosyncrasy. I say this because some patients will develop delirium when the other symptoms of the particular disease are slight, whereas other patients will be extremely sick yet not be delirious.

COLLAPSE DELIRIA.

Excessive fatigue, starvation, surgical shock, severe traumatism of any part of the body, all of these may cause delirium.

Disorientation is a leading symptom of this type.

SUBACUTE STATES OF DELIRIUM.

This type is caused in the same way as the preceding two, but is of lesser degree. The patients are confused. Consciousness is clouded. They are apprehensive. They are right on the border line.

KORSAKOFF'S DISEASE.

This is another name for the chronic mental defects of chronic alcoholism. It is entirely different from delirium tremens, which is an acute condition and spoken of under intoxications. In Korsakoff's disease the patient has false conceptions of time and place. He gives circumstantial details of things he has done and people he has seen that are all untrue; hallucinations in other words. He is totally unreliable in his statements.

COMMENT ON GROUP II.

There is one remarkable thing about this entire group. In marked cases, of course, the mental aberration is self-evident, but in mild cases it may not always be recognized. Many

patients ill of an acute disease, or after an accident, will, to the uninitiated, appear perfectly rational, when, as a matter of fact, they are not. Time and again I have known sick patients to say and do things that have hurt the feeling of those nearest to them, in apparently a perfectly natural way, things that they were entirely unconscious of at the time, and that they remembered nothing about later on. After accidents that seemed trivial I have seen patients try to go on with what they were doing, or heard them give wrong names or addresses in a seemingly rational way, when, in reality, the shock had made them temporarily dissociated with everything belonging to them. In either condition only the trained observer could detect anything abnormal, and even he might be fooled sometimes.

Treatment consists in the treatment of the cause.

GROUP III.

MANIC DEPRESSIVE INSANITY.

Definition: Insanity marked by periods of exaltation—*mania,* and by periods of depression—*melancholia.* There may be periods of lucidity in between. There is no mental deterioration.

Historical Note: It has long been recognized that persons suffering from mania or from melancholia had remissions, even recovered for a time. It was known also that some patients exhibited both the exalted and the depressed states at different times. We are indebted to Kraepelin for showing that whatever the form it is essentially one disease with different phases. He gave it the name, *"Manic-Depressive Insanity."*

Etiology: Usually occurs in those with bad neurotic family history. It is more frequent in women. The first attack usually comes before twenty-five. Severe mental or physical shock may act as an exciting cause. Childbirth may cause it.

Morbid Anatomy and Pathology: There is no known definite pathological lesion.

THE EXALTED PHASE, OR MANIA.

Definition: (Tuke.) A form of insanity characterized in its full development by mental exaltation and bodily excitement.

Symptoms: Mania is the opposite of melancholia. The mental condition varies from mild exaltation to wild delirium.

There may be a prodromal stage of depression, lack of attention, insomnia, digestive disturbances, and so on.

Then the patient becomes unduly excited and excitable. He may be abnormally witty and brilliant. He may become excessively active in business. Sometimes he becomes dissipated or pugnacious or both. He is egotistical. He is tirelessly active. As he becomes worse he has hallucinations and delusions. He has a "flight of ideas." That is, he will go from one thing to another in his conversation without finishing any. He is constantly in motion. He does not sleep.

The appetite is voracious, yet the patient emaciates. He may become filthy in his habits.

One patient, a woman, thought her baby would die on its birthday.

Another, a woman, imagined people were after her. She screamed; she had convulsive movements, during which she was cross-eyed, her mouth opened and shut with a click, there was opisthotonos.

Diagnosis: Usually easy. It must be distinguished from the acute delirium of other diseases.

Prognosis: Usually good. Most cases recover in a few months.

Treatment: Best carried out in an institution. The patient must be constantly watched so that he will not injure himself or others. Good hygiene is essential.

The homœopathic materia medica is rich in remedies for mental conditions.

Belladonna has violent mental excitement with flushed face and throbbing pulse.

Hyoscyamus imagines people are conspiring against him. May have a silly mania. May have lascivious mania.

Stramonium tries to run away from imaginary dangers. Sees strange people or animals.

Coffea. Disoriented. Constant talking.

THE DEPRESSIVE PHASE, OR MELANCHOLIA.

Definition: (Tuke.) A disorder characterized by a feeling of misery which is in excess of what is justified by the circumstances in which the individual is placed.

Symptoms: The mental symptoms may vary all the way from depression to acutal stupor. In the milder forms the patient is irritable and depressed. Interest in life becomes lessened. Memory is good and the patient's mentality is clear. But he is no longer interested in things that most concern him. He will not do things he ought to do on the general principle of "What's the use." He wants to die, sometimes has a suicidal tendency. Occasionally there is a homicidal tendency, as where sometimes a mother will kill her child because worldly affairs seem so hopeless. As the depression becomes greater the patient wears an abject expression. The world is dark. He wants to cry but cannot.

The patient feels as though under a cloud. Frequently he thinks he has committed the unpardonable sin and is doomed to eternal damnation. He is being punished for past wrong doing. Later he may have hallucinations, as the hearing of voices.

The digestion becomes impaired and the bowels sluggish. In extreme cases no effort is made to care for the person, and the urine and feces are passed involuntarily wherever the patient happens to be. He will not take the trouble to eat, will go hungry rather than make the effort. He will not move. There may be rigidity of muscles, sometimes a cataleptic condition.

Complications and Sequelæ: Sometimes results in permanent insanity. Digestive disturbances and emaciation may become troublesome.

Diagnosis: Melancholia must be distinguished from neurasthenia, in which the patient is often mildly depressed. From hypochondria, in which the patient constantly imagines he may

have some disease. And, most important, from the depression of other more serious mental diseases.

Prognosis: Usually good. The majority of patients recover after a year or two, some few after many years.

Treatment: If possible remove the cause or causes.

Mild cases may be treated at home, others will require a change. Institutional care may be essential.

The bodily functions must be gotten in normal condition. Rest and fresh air are of prime importance. The best possible mental and hygienic surroundings must be provided.

A simple nourishing diet is necessary. Sometimes forcible feeding is required.

Homœopathic remedies are of great value.

Arsenicum album, the patient thinks he is hopelessly ill.

Pulsatilla, the patient thinks she has committed the unpardonable sin.

Nux vomica, hypochondriacal, oversensitive to external impressions, whether of light or noise, often takes offense at what is said.

GROUP IV.

DEMENTIA PRÆCOX.

This group most often has its origin in a defective heredity. The majority of cases develop before the age of twenty-five. There is distinctive mental deterioration. Patients are highly nervous. They have imperative ideas. There are periods of mania and depression. There are stereotypies.

Historical Note: Kraepelin gave the name to this group. There are three types, which may, however, run into each other.

1. HEBEPHRENIA.

Definition: According to Kraepelin this form is characterized by the gradual or subacute development of a simple, more or less profound, mental deterioration.

Symptoms: The onset is gradual. The patient begins by having headache and insomnia. Then he loses interest in things and

lacks ambition. On the other hand, he may try various things. He may think he has talent which is not appreciated. He has emotional storms, but is without deep feeling. The entire character becomes changed. He wants to talk about his troubles. He may even simulate illness to obtain sympathy. Finally delusions and hallucinations appear.

Diagnosis: The history and lack of physical symptoms will serve to differentiate it from other mental conditions.

Prognosis: Bad.

Treatment: Institutional treatment is best.

2. KATATONIA.

Definition: According to Kraepelin this form is characterized by stuporous states with negativism, and by excited states with stereotypy, ending in mental deterioration.

Symptoms: The onset is gradual. The patient assumes peculiar attitudes. In case of stupor there is negativism. This may alternate with suggestibility. He refuses to do anything asked of him. There may be muscular rigidity. The patient refuses to eat. He is uncleanly. In cases of excitement he may repeat meaningless words or phrases, or give irrelevant answers to questions. In other cases there may be sudden acute mania with destruction of things or injury to the person. Hallucinations of hearing, sight, and smell occur. Judgment becomes impaired and disorientation takes place.

Diagnosis: The verbigeration and stereotypy are characteristic.

Prognosis: Usually the disease is progressive. A few cases recover.

Treatment: Institutional treatment is best.

3. PARANOID FORM.

Definition: This form is characterized by stereotypy and fixed ideas.

Symptoms: This form begins like one of the others. Later the patient has fixed delusions. They may be hypochondriacal or

expansive. There may be hallucinations of hearing. The patient may think he is controlled by other people. He often has delusions of persecution. He is egotistical. Sometimes the delusions take the character of attempts of others to poison him, and he applies to the police for protection or injures the suspected person.

Diagnosis: Must be made on the history of the case.

Prognosis: Not good.

Treatment: Institutional.

GENERAL PARESIS.

Synonyms: Progressive General Paralysis of the Insane. Paretic Dementia. Dementia Paralytica.

Definition: A chronic disease of the cerebrum and meninges causing gradual mental decay and progressive paralysis.

Historical Note: General paresis, as a distinct disease entity, has been recognized less than a century. Bayle and Calmeil, French alienists, writing in 1822 and 1826, respectively, gave the first clear descriptions of it.

Etiology: By far the most frequent cause is syphilis. Symptoms of general paresis are a late manifestation and come on several years after the original infection. Trauma, excessive fatigue and sunstroke are given as exciting causes sometimes. Men are more often affected than women. The disease develops usually between thirty and fifty.

Recently it has been found that cases react to the Wassermann test, thus indicating that the syphilis is still active.

Morbid Anatomy and Pathology: There is atrophy of the anterior part of the brain. The pia mater is firmly adherent. The microscope shows a degeneration of the nerve cells and fibers together with changes in the blood vessels.

Symptoms: The onset of general paresis is insidious. It is only after the symptoms have become more or less pronounced that they are recognized. The first sign is a general change in the disposition and character of the patient. He is by turns irritable and apathetic. He is inattentive to his business, or he is overattentive and constantly planning things that he never carries

out. He becomes careless of his person, and may be unconventional or indecent in his behavior. He gets queer notions in his head that he cannot rid himself of. One patient of mine felt he was never clean about the anus, and was constantly washing himself. He later began ordering goods in his business in quantity sufficient to supply the world. It was his astounding orders that got him into trouble and led to a diagnosis of his case finally. This was a "delusion of grandeur" typical of many cases. The patients become egotistical, believe they are persons of vast importance or of fabulous wealth.

The patient becomes forgetful. He is incapable of continued mental effort, he tires easily. Speech becomes hesitating, he stumbles over syllables, or he develops some form of aphasia. Sometimes he cannot read. He feels confused, mentally, suffers from diziness and fulness in the head. Later the writing becomes quite characteristic. He leaves out or puts in syllables or words, the letters are irregular, finally the writing becomes illegible altogether.

The pupils may be uneven and do not react to light. The patella reflex may be exaggerated or lost. The tongue is tremulous. There may be ataxia and loss of sensibility.

In short, the whole disease is a progressive mental and physical decay, with, sometimes, periods of remission.

Many patients have recurring attacks of aphasia and of paralysis, either hemiplegia or monoplegia. Others have attacks of vertigo or attacks simulating epilepsy or apoplexy.

Complications and Sequelæ: There may be emaciation, loss of control of the sphincters and bed sores.

Diagnosis: Loss of interest in one's affairs, change in the moral make-up, and the characteristic changes in speech and writing, make the case reasonably clear.

Tumors and gummata may produce some of the symptoms of general paresis and be mistaken for it.

The cerebro-spinal fluid should be examined. Lymphocytosis is found in it. In general paresis and in locomotor ataxia albumin is also present in the fluid.

The Wassermann test may be positive.

Prognosis: The prognosis is bad. Some cases present remissions for a time, but eventually death occurs. The disease may last for several years.

Treatment: Patients must be placed under the best possible conditions. Business must be given up; many men have ruined themselves by foolish or wrong dealings before the true reason for their actions was realized. Rest, mental and physical, must be insisted on. The whole life must be regulated, and this is best done in an institution.

Antisyphilitic treatment should be tried—salvarsan, mercurial inunctions and potassium iodide.

Baths, massage and electricity may help some.

The indicated homœopathic remedy should be given according to the symptoms.

EPILEPSY.

Synonym: Falling Sickness.

Definition: Epilepsy is a disease characterized by paroxysmal loss of consciousness, which periods are usually associated with convulsions.

Historical Note: The Greeks named the disease "a falling upon" as by a spiritual agent.

Etiology: Heredity seems to play a part. Strümpell says it is not necessary to find other cases of epilepsy in a family, simply that other members of the family have neurotic tendencies.

Epileptiform attacks occur in certain diseases and sometimes follow injuries. But none of these are true epilepsy, the exact cause of which is unknown. It is a neurosis and occurs about evenly in the two sexes.

Most cases develop before twenty years of age.

Many cases date their origin from some specific cause, as infantile convulsions, slight brain injury, following the infectious diseases, emotional disturbances, as fright, anxiety, or from intestinal reflexes.

Any of the above may precede the first epileptic seizure. Gowers says, "we regard a certain persistent condition of the nervous tissues as the most general element in the causation."

The immediate existing cause "is either like the gradual rise of temperature, which brings about explosive combustion, or a spark which immediately excites a discharge of explosive material."

After a first attack subsequent ones are easier because "every action paves the way for its own recurrence. . . . Every fit is in part, at least, the result of those which have preceded it, and in part a cause of those which follow it."

Morbid Anatomy and Pathology: Some unknown cortical disturbance.

Symptoms: There are different forms of epilepsy—*Grand mal*—the usual form; *Petit mal*—very mild; *Jacksonian*—cortical or partial—consciousness may not be lost—the spasm is local.

There is a peculiar facial expression.

Grand mal: Usually the attack is preceded by an "aura," that is, a peculiar warning symptom. This may be sensory, motor, vaso-motor, or psychical. There may be changed sensation in some part of the body. The patient may perceive a peculiar odor or taste. There may be palpitation, pain in the epigastrium, or even nausea. There may be a dreamy sensation preceding the attack, or there may be hallucinations.

The aura usually lasts but a short time, just long enough for the patient to find a place of refuge. Occasionally it comes on several hours or even days before an attack.

Some few cases have no warning. The patient becomes unconscious and drops where he happens to be. There may be an involuntary cry. The body is in a tonic spasm, respiration is suspended, and the patient becomes cyanotic. The teeth are clenched. The hands and arms flexed with the fingers tightly closed, the legs extended. The head is usually turned to one side.

After a few seconds the clonic stage begins. The muscles first tremor, then are thrown into violent convulsions. The facial muscles become greatly contorted, the eyes roll, the patient froths at the mouth. He may bite his tongue.

There may be involuntary bowel and bladder evacuations.

This lasts a few minutes then gradually quiets down, and the

patient passes into coma. He may go into a peaceful and restful sleep. If he becomes conscious right away, or his sleep is disturbed, he is confused, and, usually, is not in normal mental condition for several hours.

Petit mal: This is a mild type of the disease. There may be only momentary loss of consciousness with no recognizable spasm after; or there may be only hesitancy in speech for a moment; or a blank expression of the countenance which almost instantly passes over.

Automatic motions may occur; the patient may do ordinary things while irrational.

Jacksonian epilepsy is caused by a definite lesion of the cortical motor center on the opposite side. The seizure is limited to one side of the body, or to a limited group of muscles, as the face, arm or leg. There is more or less constant disturbance in this form.

General remarks: There may be seizures only at night during sleep. If the patient sleeps alone these may go on for years without detection. In other cases—the majority—seizures occur both waking and sleeping. The majority have seizures only during the waking state.

Women are apt to have attacks during the menstrual period. Pregnancy may or may not have any effect on the seizures. As a rule, cases that develop before puberty are apt to become more pronounced then.

The severity of the attacks varies all the way from the slightest and briefest momentary loss of consciousness to the most violent convulsions followed by hours of mental cloudiness.

The frequency of attacks varies from possibly one or two a year to as often as several every day. The majority of cases will have an attack every two or three weeks. Sometimes there will be two or three seizures within a few days, then an intermission of months.

There is a periodicity in most cases. In others a status epilepticus, frequently recurring paroxysms with no consciousness between.

Complications and Sequelæ: In some cases the mentality finally

becomes impaired. Gowers says this is more apt to occur in patients with the milder forms. As a result of treatment attacks may stop and the patient become depressed. Gowers thinks the energy is accumulating all the time, and the discharge is necessary for relief. If the attacks become frequent the patient may develop dementia.

Other patients are unaffected mentally. The patient may injure himself in his unconscious state.

Diagnosis: As a rule, epilepsy is easy of diagnosis. It is most easily confounded with hysterical convulsions. The epileptic has his seizure without special cause, apparently. Unconsciousness is sudden; the convulsive stage is short and cannot be prolonged or brought on by touching the patient. He froths and may bite the tongue.

In hysteria attacks are brought on by the emotions, by a close room, etc. They can often be repeated by touching the patient.

Mild attacks may be confounded with syncope or vertigo. No mental confusion results after these.

Prognosis: Unfavorable as regards cure. Has no tendency, *per se,* to shorten life. It may cause accident. Minor attacks are the most unfavorable.

Treatment: The epileptic must be put under the best possible hygienic measures. Exercise is essential, but it must not be excessive. L. Pierce Clark has found that games like foot ball and base ball, entailing mental excitement as well as strenuous exercise, are harmful. Inaction is equally so. Clark insists on the cold douche or the cold tub daily in the able bodied. He says this is doubly necessary if bromide therapy is followed to counteract the circulatory depression caused by the drug. He withholds bromides until the circulation, digestion, and other functions have been properly regulated.

The accepted treatment by the old school is with the bromides. They are given in doses of ten to thirty grains, one to three times a day. Cures have been reported. The treatment must be continued for two years after attacks cease.

Bromide of potassium must be well diluted. Bromide of sodium is preferable, and may be given in milk or mineral water.

The bromides may cause a skin eruption; this may be antidoted with Fowler's solution.

Jacksonian epilepsy is sometimes cured by operating on the brain.

Some homœopathic remedies suggested for epilepsy are:

Absinthum, agaricus, arsenicum, artemesia, belladonna, calcarea carbonica, cannabis indica, cicuta, cuprum, hydrocyanic acid, plumbum, silicea, according to indications.

HYSTERIA.

Definition: A psychoneurosis characterized by increased impressionability and lack of self-control and manifested by a train of symptoms of most varied character.

Historical Note: The name hysteria—ὑστερα, uterus—was originally given to the condition because derangement of the sexual function in women was supposed to be at the bottom of it. We know now, however, that men may suffer as much from hysteria as women, that it is a distinct disease entity with protean manifestations.

Etiology: Hysteria is found more often in women than in men, more often in the Latin and Jewish races than in Anglo-Saxons. It develops most often in early adult life.

Heredity plays an important part in that cases are more apt to occur in neurotic families. Faulty training is another factor.

Nearly all cases are brought on by emotional disturbance. An attack may be precipitated by some violent emotional disturbance, a "psychical trauma," as Strümpell terms it, or it may be the result of long continued anxiety, sorrow, disappointment, and so on.

Morbid Anatomy and Pathology: None known.

Symptoms: Before speaking of special symptoms I wish to quote a few paragraphs from Strümpell.

"1. All hysterical disturbances, no matter how severe the functional nervous derangement attributable to them, are without visible anatomical basis. The best proof of this is the fact that any hysterical affection, however severe, may sometimes develop and then may recover completely in a very short time.

"2. The hysterical affection is very often most intimately associated with discoverable causes of a psychical nature. Not only is its appearance and incipiency most closely linked with emotional excitement, but later on the mind is the main, if not the only channel through which causes can operate to change the condition of the patient, whether favorably or unfavorably.

"3. It is, therefore, evident that the origin of all hysterical disturbances must be sought in the most central portions of the nervous system, so far as these depend upon psychical processes or may be altered by them. The symptoms of hysteria are, therefore, more manifold than those of almost any other disease. Although so manifold, certain symptoms predominate with such frequency as to be characteristic of hysteria, and, therefore, to be regarded as especially valuable in diagnosis. Following Charcot we term such symptoms 'hysterical stigmata.' These symptoms are most constantly present, and, therefore, they can easily be found at any time.

"4. Besides the premanent symptoms—the 'stigmata'—we see in many cases of hysteria peculiar nervous attacks. These are also highly characteristic of hysteria, since they come on in forms which are seen in no other disease; but there are also patients who suffer from hysteria who, nevertheless, are never visited by attacks. The possibility of the occurrence of an attack is, however, always present. Very often the attacks are provoked by easily discoverable psychical causes."

In a disease of such manifold manifestations as hysteria it is difficult to give a satisfactory description that will cover all cases. Any ordinary individual may be profoundly upset by some sudden emotional shock and be "hysterical" temporarily. That is far different from the disease "hysteria" which is very real to the patient and frequently a calamity to the patient's family.

The subject of hysteria is often extremely capable and clever in the ordinary affairs of life, and when things go right is bright and cheerful. The patient craves affection and attention, but treasures up slights and grievances. Small things disturb the equanimity beyond all reason, whereas big things may be overlooked and accepted with surprising resignation.

She is easily influenced and is apt to mirror the ideas of others.

The patient is apt to exaggerate aches and pains and the small affairs of everyday life.

There is often a sort of vacant expression to the face that is more or less characteristic. This is especially true if an hysterical explosion is imminent.

The disease is marked by paroxysms or explosive outbreaks which vary in duration and intensity, and may simulate any disorder of the nervous system, (1) mental; (2) motor, or (3) sensory.

(1) *Mental disturbances:* The attack may begin with crying over some emotional disturbance. The crying may alternate with laughing, this may be followed by delirium. In the delirium some word or phrase may be constantly repeated, or the patient may carry on imaginary conversations. This condition usually ends in unconsciousness of longer or shorter duration. From this the patient possibly passes into a normal sleep from which he wakens in a normal mental condition.

Sometimes actual hysterical insanity supervenes, the patient being insane for weeks or months.

Or an emotional shock may change completely the patient's character. From being loving and affectionate she may become hard and cruel. She may take an aversion to things she was fond of, or turn completely against those she formerly loved most. The reasoning is usually correct, but the patient starts from wrong premises.

In other cases the patient will faint away, become unconscious and remain in that condition for hours. I have seen such a patient lapse into unconsciousness in the middle of a sentence, during an hysterical outbreak, and remain totally unconscious for twenty-four hours, finishing her sentence when coming to without ever being aware of the intervening time. During the period of unconsciousness the quiet breathing was the only evidence of life, all other functions being in a state of suspended animation. The bowels and bladder remained quiescent.

(2) *Motor disturbances:* Some patients have hysterical convulsions. These may vary from slight twitchings of various

muscles to the most violent movements of the arms and legs. The twitchings may occur during sleep. A favorite form of spasm is for the patient to assume the position of opisthotonos, supported only on the heels and the back of the head, the entire muscular structure rigid. As a rule, the patient remains in this position but a few moments, but the spasm may be frequently repeated over hours or days, the patient being unconscious between times.

A patient during an attack where convulsions are present seems to be in an hyperesthetic condition, and any slight disturbance will start the convulsion. I have seen a patient have one of these convulsions every time any part of the person was touched; even an attempt to feel the pulse. The slamming of a door or other unexpected noise would do to start one.

A soreness of the muscles and a tired feeling afterwards is common. One of the worst cases I ever saw was a man in my service at the Metropolitan who had a convulsion every time his abdomen was palpated. I made the discovery by accident the first time, and exhibited the case at a clinic afterwards. In his case the convulsions were brief and consciousness was complete between times. Sensitive spots in the abdomen are said to be common. Sometimes the spine may be specially sensitive. These are called hysterogenous zones.

In other milder cases the convulsive stage may be simply a rapid shallow breathing with palpitation, that is, the patient can feel the heart beat, though it may not be rapid. I have seen the respirations as high as seventy and eighty and ninety per minute, the pulse about the same, heart regular and sounds clear. One such case was considered at the point of death by the house physician, and the patient felt she was herself; the nurses and internes were all alarmed and used stimulants and what not. The attacks were purely hysterical and passed over in a few minutes without assistance.

Hiccough lasting for hours and days may be another form of hysterical spasm.

Paralysis of various muscles may take place. Hysterical aphonia is common. It may last for several days or a week. We may have hysterical hemiplegia, paraplegia or monoplegia.

In the cases I have seen aphonia has developed abruptly, the voice returning, as a rule, only after some days and then gradually. On other occasions I have known the voice to return suddenly under stress of anger.

I have seen hysterical monoplegia of the leg where the patient was unable to walk at all for weeks, and dragged the foot for a long time thereafter.

Closely allied to the above are hysterical contractions. The limbs may become fixed in all sorts of odd positions. Strümpell says: "All hysterical contractures disappear completely in chloroform narcosis, after the use of an Esmarch bandage, and in deep sleep."

3. *Sensory disturbance.* Anesthesia of various parts of the surface of the body are noted in hysteria. Sometimes a dulling in sight, hearing, taste and smell.

Conversely, hyperesthesia may be a prominent symptom.

Indigestion with flatulence is frequently found. The patient may be constipated, have diarrhea, or may alternate between the two conditions.

There is usually polyuria, rarely ischuria.

Finally cases have been reported of hemoptysis and hematemesis that were said to be manifestations of hysteria. In these cases it is well to be careful to investigate the actual origin of the blood, for simulation is common, and next it is desirable to exclude all other possible causes of the hemorrhage.

Cases of very high fever have been reported. Such cases should be viewed with suspicion. One of my patients at the Metropolitan had the most remarkably variegated temperature, going as high as 107° F. at times, but without regularity. She was finally caught placing the clinical thermometer on a hot water bag.

Complications and Sequelæ: No permanent changes.

Diagnosis: On first seeing a case of hysteria, if seen in a convulsion, it may be somewhat difficult to differentiate it from epilepsy. The history of the case will be of help. The convulsion itself lasts much longer in hysteria and may be repeated with slight provocation. It usually starts from some emotional

disturbance which may itself seem very unimportant to the onlooker.

I have seen hysterical attacks which consisted of excessively rapid respirations mistaken for a dangerous condition, alarming patient and attendants alike. The heart and pulse remained normal.

Some patients will have temporary attacks of loss of power in the hands with the fingers drawn into distorted positions, or there will be temporary loss of power in the legs, with rapid respirations, attacks which pass over in a few moments. These are easy of diagnosis.

Hysterical paralysis lasting weeks or months are not so easy of diagnosis unless the history of the patient is known.

Prognosis: Is favorable, as regards life, but the disease may last indefinitely.

Treatment: Hysteria is a real disease entity and not to be treated lightly. It requires skill and patience on the part of all concerned. Most cases have their basis in an unstable nervous system—a something they start out in life with. The inherited faulty nervous system means that the parents, one or both, have faulty nervous systems, and that probably means faulty upbringing for the child.

It is essential that hysterical patients have some occupation. If obliged to care for themselves they can often do it. They cannot afford to let themselves go and don't.

Mitchell's rest cure is good for some. It gets them away from disturbing surroundings.

Morphine must never be used.

The homœopathic remedy is of value. *Ignatia, moschus, chamomilla, belladonna, cannabis indica, nux vomica* are the most often indicated.

CATALEPSY.

(Greek, Κατα λαμβανω, to seize.)

Definition: A spasmodic disease marked by suspension of consciousness and sensation with rigidity of muscles.

Historical Note: Formerly catalepsy was considered to be

a separate disease entity; it is now thought to be symptomatic only. It occurs most often in hysteria.

Etiology: Not known. Occurs most often as a symptom of hysteria. It may occur in melancholia, katatonia, general paresis, brain lesion.

Symptoms: In catalepsy the patient is unconscious, or, at any rate, oblivious of his surroundings, and the arms and legs will remain indefinitely in whatever position they are placed no matter how absurd or uncomfortable.

Diagnosis: Is self-evident.

Prognosis: Depends on the disease with which it is associated.

Treatment: Also depends on underlying condition.

NEURASTHENIA AND PSYCHASTHENIA.

Synonyms: Nervous Weakness. Nervous Debility. Nervous Prostration.

Definition: This is a weakness or irritability of the nervous system. The mental condition is abnormal, due, Strümpell says, "to a disturbed ideational life."

Historical Note: Strümpell says neurasthenia is not a modern disease, but "is as old as our definite knowledge of the diseases of mankind." Beard, in 1880, gave the modern impulse to its study.

Etiology: As in hysteria, neurasthenia is apt to have a nervous heredity. Excesses of work or dissipation, worries of business, etc., cause it. Prolonged anxiety and worry over love affairs.

Symptoms: A leading symptom of neurasthenia and psychasthenia is an inability to concentrate one's mind. It may be that the brain is tired, or it may be due to the constant intrusion of some worrying thought about one's health, or business, or other affairs. The patient is unable to decide on what to do. He suffers from insomnia.

Because of these things the patient is depressed and morose. He may even think of suicide. Or the patient may alternate between depression and exaltation. He is always more or less nervous and irritable.

The patient may be hypochondriacal and be in constant dread of some particular kind of disease. A dread of heart disease is one of the most common. Sometimes the heart feels tired and the patient has to move around to start it up. The anxiety produces attacks of palpitation which make the patient certain of his bad condition. With the nervous palpitation there is often nervous dyspepsia. Sexual neurasthenia is common.

The various phobias sometimes take possession of the patient; he is afraid to be alone; or he suffers from agoraphobia, a fear of open places, etc.

Osler lays special stress on a feeling of pressure in the head. There are vaso-motor sweatings and flushes.

There is often muscular weakness, the patient tiring very easily.

There may be hyperesthesia to pain. Sometimes the reflexes are exaggerated.

Occasionally the special nerves may be affected.

Complications and Sequelæ: Sometimes neurasthenia is the result of organic disease.

Diagnosis: Neurasthenia is usually easily diagnosed. Sometimes it is on the borderland of hysteria.

Examine the patient carefully to exclude organic disease of any kind.

Prognosis: Good with proper care. It may take a long time to bring about a cure.

Treatment: Change of habit and rest are necessary. Sympathy for the patient may or may not be a good thing.

The rest cure is indicated in some cases.

Hydrotherapy and massage are of help in toning up the system.

Aconite, the patient is apprehensive and afraid to be alone.

Gelsemium, the patient must move about to keep the heart going. The heart feels tired. There is a feeling of weight at the base of the brain.

Cactus grandiflorus, nervous dyspepsia and palpitation.

Nux vomica, feels as though she would drop if she moved. Feels tired and faint. Has sinking spells.

Carbo vegetabilis and *nux vomica* in combination for much gas in the stomach.

Ignatia, irritable, contrary, everything wrong.
Phosphoric acid, homesickness.
Arnica tires easily and aches as though from exertion.
I have verified all of the above.

TRAUMATIC NEUROSES.

Synonyms: Accident Neuroses. Railway Brain and Spine. Traumatic Hysteria.

Definition: The name given to the nervous and mental symptoms following the shock of accident.

Historical Note: According to Osler, Erichsen called the condition railway spine. Walton and Putnam pointed out the hysterical character.

Etiology: This neurosis may follow a slight blow or fall or it may follow serious accident in a railway train, automobile or what not, with loss of consciousness and severe physical injury at the time. There may be no actual physical injury, simply the shock of being in or responsible for, or seeing some shocking accident.

Morbid Anatomy and Pathology: It has been thought that there was inflammation of the meninges or spine. In some cases minute injuries are found, in others no gross pathological change can be found. It seems to be purely psychical.

Symptoms: Most of us have had personal experience, more or less severe. I once saw a woman killed by a train in the subway. For more than a year afterwards I could not bring myself to use that station again. The condition is largely a mental one. Strümpell says: "The material trauma is, as we express it, associated with a 'psychical trauma.' Among these we may reckon not merely the fright associated with the accident, but the whole series of new ideas occasioned by the accident and entering most vividly into the consciousness of the injured person—anxiety about the further course of the injury, about the restoration of the capacity to work and earn a living, and also about obtaining any probable damages, etc. Sordid mental motives also are sometimes mixed with the others in consciousness, the desire for indemnification in money, for a better way of living," etc.

Whatever physical injury may have been sustained may be entirely recovered from. But the mental condition of the patient may be altogether different from what it was before the accident. These symptoms may not develop for some time afterwards.

The patient is easily tired out. He is nervous and morose, sometimes even despondent to the point of melancholia. He is unable to get his mind off his troubles and to concentrate it on anything useful. More or less incapacitated for work, the affair becomes an obsession, and the patient may go from physician to physician trying to get sympathy and aid for threatened law suits.

Headache is a very common symptom of these cases. So is sleeplessness. Nervous so that every little noise startles him is another.

Weeping on slight provocation and loss of nerve and indecision are other symptoms. The eyesight is not as good as usual. The hearing may be impaired. Sensitive spots on the spine or various other places may develop. The patient thinks he is unable to use his arms or legs as formerly.

Complications and Sequelæ: Occasionally these cases go down hill till the mind is completely lost.

Diagnosis: Follows accident after a time. Eliminate simulation if possible, and exclude organic disease.

Prognosis: In mild cases is good. In severe cases is bad.

Treatment: This is psychical to a large extent.

Arnica is probably a valuable remedy.

HEAT AFFECTIONS.

Synonyms: Sunstroke. Heat Stroke. Heat Apoplexy. Insolation (*sol,* the sun). Thermic Fever. Heat Exhaustion. Heat Prostration.

Definition: Disease produced by excessive heat.

Historical Note: It has been recognized since ancient times that the direct rays of the sun, or that excessive heat, could cause sickness, possibly death. One of the most complete studies

of the subject is the monograph of H. C. Wood, published in 1872.

Etiology: Anything that tends to lower the general condition increases the susceptibility to heat. For example, excesses of all kinds, either of work or dissipation, worry, anxiety. A most potent cause is the use of alcohol. I have frequently seen men fall out of the ranks at the State Camp at Peekskill on account of the heat, but they were usually men suffering from loss of sleep who had been drinking. Persons suffering from disease are also specially susceptible. Excessive heat always increases the general death rate.

A warm, moist atmosphere conduces to heatstroke. In high altitudes with a dry atmosphere it is almost unknown. According to Lambert, in New York City, in one week in 1896, during a hot spell, with the thermometer in the nineties, 648 of the 1,710 deaths were due to sunstroke. During that same week I was in Albuquerque, New Mexico, at an altitude of 5,000 feet, in a dry atmosphere. The thermometer passed the 100 mark every day, yet no case of sunstroke occurred. Various unknown factors must contribute to heat stroke besides moisture. In Galveston, Texas, where the summer heat is great and the moisture excessive, I was told that sunstroke was very rare. This may be because negroes do nearly all the manual labor, and negroes can stand more heat than whites.

Natives are less prone to suffer from the heat than those unacclimated. This is well shown in the governmental figures of the English army in India. (Sir Joseph Fayrer, in Allbutt's *System of Medicine.*) In 1891 among the European soldiers, there were 0.97 per thousand cases of heat stroke; among the native soldiers, 0.20 per thousand.

Men are more often attacked than women. Probably on account of greater exposure. Children are quite as susceptible as adults.

Cases due to sun exposure are more frequent in the afternoon. Eighty per cent. of my recorded cases occurred after midday, the other twenty per cent. between 10 A. M. and noon.

Heat stroke also occurs at night among those who work in

hot places. The big bakeries and sugar refineries furnished many such cases during my service at the Brooklyn Homœopathic Hospital. These cases usually occurred before midnight. In India night cases are frequent.

Morbid Anatomy and Pathology: Rigor mortis sets in almost immediately. Putrefactive changes begin very early. The coagulability of the blood is impaired. The red corpuscles are frequently crenated, and are slow in forming rouleaux. The alkalinity of the blood is lessened, and it may even be acid. Venous engorgement is marked. Wood states that immediately after death the left ventricle is firmly contracted, whereas the right heart and pulmonary arteries are filled with dark fluid blood. Delafield and Prudden have not found the left ventricle always contracted. Parenchymatous changes have been reported in the heart, liver, and kidneys. Von Gieson has found acute parenchymatous degeneration in the neurons throughout the central nervous system. He has suggested that these changes are due to an autointoxication induced by the heat. Sambron suggests that heat stroke is an infectious disease. He bases his idea on the fact that heat affections occur only in certain places and are apt to be epidemic in character.

Symptoms: There are two types of heat-stroke, (1) ***sunstroke proper,*** the sthenic type, and (2) ***heat exhaustion,*** the asthenic type. In both there may be prodromal symptoms of dizziness, headache, a distress in the epigastrium, with nausea, perhaps vomiting, dryness and heat of the skin, frequent urination and great discomfort from the hot atmosphere. Colored vision is common. If taken in time the symptoms may go no further.

Sunstroke or *insolation* may be thus preceded, or it may set in at once with loss of consciousness. This form is usually due to the direct rays of the sun. The patient falls unconscious. The pupils are usually contracted. The face is flushed. The pulse is rapid, full and bounding. Breathing is stertorous. The temperature rises rapidly to a great height; it is frequently as high as 110° F., it may be much higher. I have seen it as high as 112° F. in the axilla. The skin is dry and hot to the touch. This condition may cause death in a very short time, in one or two hours;

or the patient may go on for a day or more. If recovery takes place the symptoms may ameliorate in a very few hours, or the patient may get better more slowly, taking several days for all bad symptoms to disappear.

In ***heat exhaustion*** or ***heat prostration*** the patient has usually been in a hot, close atmosphere, but not exposed to the direct rays of the sun. The symptoms range all the way from simple syncope to the most profound coma. Prodromal symptoms may or may not have been present. In these cases, the asthenic type, the face is pale, the pulse rapid, but weak. The skin is cool and may be wet with perspiration. The temperature is not high and may even be subnormal. The patient presents all the symptoms of collapse. Cheyne-Stokes respiration may occur and the patient promptly die. Or these symptoms may gradually abate and reaction set in, with the development of a thermic fever running for several days and ending in recovery or death. Or the symptoms of collapse may be relieved without a strong reaction in the other direction, and the patient make a speedy recovery.

In profound cases of either type convulsions may occur. There may also be paralysis of the sphincters with involuntary loss of urine and feces.

Complications and Sequelæ: In certain rare cases true cerebral apoplexy or uremia may develop. The disease may be associated with acute alcoholism. Occasional cases of mania have followed heat stroke. A very frequent sequence is an inability to stand high temperatures. More or less persistent headache, or headache always on exposure to heat is quite common. It is supposed to be due to chronic meningitis.

Diagnosis: As a rule, diagnosis is not difficult. The circumstances of the case preclude error. Occasionally the diagnosis is in doubt. In cerebral apoplexy the temperature is low, in sunstroke very high. In apoplexy there is usually a difference in the size of the pupils, in sunstroke they are alike. In apoplexy the pulse at first is slow, in heat affections rapid. In apoplexy we have symptoms of paralysis supervening, in heat stroke simply a general weakness without paralysis. If apoplectic convulsions occur they are unilateral, if heat convulsions occur they are symmetrical.

The diagnosis between acute alcoholism and heat stroke is sometimes difficult. In alcoholism the temperature is low or subnormal, in heat affections it is not, as a rule. The acute effects of alcohol wear off after a short time, whereas a heat stroke patient will usually have severe headache on recovering consciousness.

Uremia may come on in hot weather. It is more apt to be associated with convulsions, Cheyne-Stokes respiration and suppression of urine, than heat affections. The sthenic form of heat stroke can be differentiated by the high temperature. The asthenic form by the moist skin of heat exhaustion, and dry skin of uremia. The unconsciousness of heat affections becomes either better or ends in death in a few hours. The unconsciousness of uremia may last for several days.

Prognosis: In mild cases, with prodromal symptoms only, the prognosis is good. In severe cases going on to unconsciousness the prognosis is bad—nearly half the cases die.

Treatment: When the initial symptoms of headache, dizziness, frequent micturition, or discomfort set in, the patient should rest in a quiet cool place. Clothing should be loosened and cooling applications made. By this means further trouble may often be averted.

In sunstroke, with unconsciousness, the patient should be placed in the shade, and all constricting clothing loosened. Plentiful quantities of water should be applied to reduce the temperature. Water at an ordinary temperature is to be preferred to ice water. Raue calls attention to the fact that a frozen member is rubbed with snow, not hot water. Similarly, in sunstroke, ordinary water is cool enough to reduce the hyperpyrexia of the patient. When the temperature begins to approach 102° F. the application of water should be stopped.

In heat exhaustion, with low temperature, the treatment consists in the application of external heat and in stimulation. If the temperature is subnormal hot baths are indicated.

Alcohol is useful at times in heat exhaustion; it is worse than useless in sunstroke, it is dangerous.

During convalescence a liquid diet is indicated—milk, buttermilk, albumin water, and so on.

In case convulsions occur Osler recommends inhalations of chloroform. In heat exhaustion, strychnine in 1/30 grain doses may tide over a crisis.

Homœopathic remedies are extremely valuable. As a preventive, on the appearance of prodromal symptoms, I can fully endorse Raue's use of *gelsemium;* the lower dilutions every one or two hours are preferable. This is especially useful in damp stifling weather.

Glonoin is the remedy in sthenic cases, with great congestion, full pulse, high fever, loss of consciousness. Cowperthwaite says the glonoin face is pale. The third to sixth dilution. It may be given hypodermically. The old school recommend it in 1/100 grain doses.

Belladonna has flushed face, congested eyes, full, bounding pulse, unconsciousness, high fever, hot, dry skin.

Amyl nitrite has the congestion of sunstroke. There is also a special distress about the heart with more or less irregularity of rhythm. The ordinary crystals may be used.

In heat exhaustion *camphor* may be useful. The patient is collapsed and there are indications of heart failure. Drop doses of ordinary spirits of camphor may be given as often as five or ten minutes until reaction sets in.

In the susceptibility to heat, chronic headaches, and other sequelæ of heat stroke, *glonoin* is one of the most useful remedies.

Raue also mentions *stramonium,* "headache from being exposed to the sun." A patient at the Flower Hospital jumped out of the window in his heat delirium. *Stramonium* restored consciousness after a few doses.

Other remedies may be indicated, but the above are the most generally useful.

MULTIPLE SCLEROSIS OF BRAIN AND SPINAL CORD.

Synonym: Disseminated Sclerosis.

Definition: A chronic disease of the central nervous system, characterized by the development of numerous sclerotic nodules in the brain and spinal cord.

Historical Note: Charcot formulated a definite type.

Etiology: Unknown. The condition is rare. It occurs from twenty to thirty-five.

The sexes are equally affected. It occurs in neurotic families, usually. It may follow the infections. Cold, traumatism, over-exertion, mental shock, may cause it.

Morbid Anatomy and Pathology: The sclerosed spots can be seen and felt scattered through the brain and cord. They may be found in any part of the brain, but in the cord seem to have a predilection for the white substance. They consist of neuroglia tissue. The nerve fibers are not wholly destroyed, they are merely lessened in number.

Symptoms: There is a muscular weakness and more or less ataxia. There is intention tremor. This is found in the arms as well as the legs. It also affects the tongue, causing scanning speech, that is, the bringing out of each syllable separately. Nystagmus is frequently seen, an ataxia of the ocular muscles. The gait is peculiar and stiff. Romberg's sign is present—inability to stand with the eyes closed. The reflexes are exaggerated. There is persistent ankle clonus. The pupils are often irregular and react slowly, both to light and to accommodation.

There may be optic atrophy.

Paralysis rarely appears and then very late in the disease.

There are sometimes vertigo and headache. There is often mental deterioration, and, finally, apoplectiform attacks with hemiplegia, which rapidly disappears. Also emotional laughing and crying. There may be bulbar symptoms as dysphagia, glycosuria, polyuria. There is no disturbance of the bladder.

Complications and Sequelæ: Apoplexy, paralysis, bed sores, death.

Diagnosis: In typical cases this is easy.

Atypical cases may simulate myelitis, spastic paraplegia, apoplexy, tabes, or Friedrich's ataxia. In the last two the reflexes are lost. In sclerosis they are increased.

Prognosis: Generally bad, although it may run for twenty years. Sometimes remissions occur.

Treatment: Absolute rest in bed is the first requisite.

Warm baths may help.

Sometimes the use of electricity is valuable.

Sulphur, calcarea carbonica, silicea, nux vomica, argentum nitricum, plumbum are some of the remedies that may be thought of.

APOPLEXY.

(Greek, *ἀπό*, from *πλήσσειν*, to strike.)

Definition: Apoplexy is the popular name given to sudden loss of sensation, motion, and consciousness, whether due to cerebral hemorrhage, embolism, or thrombosis, or to hemorrhage into the dura mater of the brain.

Pathologically, the causes are somewhat different; clinically, the symptoms are nearly the same, and absolute certainty in differential diagnosis during life is often impossible. For this reason I propose to consider the symptom-complex produced by these varying conditions under one heading, namely, *apoplexy*.

Historical Note: Cases of apoplexy have undoubtedly occurred at all times throughout the world's history. Ancient writers spoke of such cases. Virchow, by his studies of diseases of the arteries, did much to crystallize modern conceptions of the pathology of various forms of apoplexy. Durand-Fardel, in 1854, gave a list of the most frequent sites of hemorrhage into the brain. The development of brain localization by Broca, Meynert, Fritsch, Wernicke, Jackson, Charcot, Nothnagel and others during the last half of the nineteenth century have made it possible, in many cases, to locate the exact site of the brain lesion during life from the symptoms presented.

Etiology: Traumatism is the direct cause in many cases. A fall or blow on any part of the head, even a fall where the head itself is not directly injured, may cause cerebral or dural hemorrhage.

All of the conditions that cause arteriosclerosis, as syphilis and Bright's disease, may result in rupture of a cerebral artery or in embolus. Minute aneurisms in the cerebral vessels may rupture causing hemorrhage.

Age is an important factor. Old people are more subject to apoplexy than young. Strain is often an exciting cause.

Syphilis is the most frequent cause in young people.

Middle ear disease and valvular heart disease may cause embolus or thrombus. Also suppurations in other parts of the head, as the scalp, nose, orbit or face, or even in other parts of the body.

Morbid Anatomy and Pathology: The most frequent site of cerebral hemorrhage is about the fissure of Sylvius, although it may occur in other parts as well. The resulting symptoms may be due simply to pressure, or they may be due to actual destruction of brain tissue. The blood clot is at first dark colored, and later becomes brownish. If the patient recovers the clot gradually becomes absorbed. Sometimes it results in a larger or smaller serous cyst, at other times it disappears altogether. The size of the original clot varies; it may be small or large, or there may be a number of varying size. In many cases that come to autopsy the clot is hidden, and only careful dissection of the brain will reveal it. In others again the clot is quite superficial. I have seen a clot as large as the closed fist. This was a case of fracture of the base. The accident happened on Sunday. For an hour or two the patient was unconscious. The next day he was up and around, and presented no symptoms. For several days this continued, until Friday when the man suddenly died. I made the autopsy and found the clot as above. In another case the clot in the right ventricle weighed an ounce.

Thrombus and embolus usually result from some disease causing arterial change, or from some disease causing mitral disease of the heart, as gout, alcoholism, or the acute infections.

Meningeal hemorrhages may also be found in all degrees, and due to the same causes as cerebral hemorrhage.

Symptoms: There are certain premonitory symptoms that should always arouse suspicion in plethoric subjects, or in those known to have arteriosclerosis, Bright's disease, or valvular heart lesions.

They are persistent headache, especially a very full feeling, or a very severe frontal or more often occipital headache. There may be vertigo, sometimes slight nausea. The pulse may be slow but full and strong. I have had such patients where I felt a cerebral hemorrhage was impending, but it did not materialize

then. Several such patients have eventually died of apoplexy. One woman lived ten years after I made note of what I feared, but she finally died of apoplexy.

The symptoms of apoplexy may vary all the way from a slight disturbance of speech, irritation or diminished control of one-half the body, without unconsciousness at any time, to complete coma with paralysis or convulsions.

It has been my fortune to see a number of cases during or immediately after the initial symptoms of the attack. Two of them, one an old man and the other an old woman, fell to the floor unconscious. This was followed by profuse vomiting and involuntary stools.

The man I saw but once. The woman was paralyzed on the left side. Her unconsciousness lasted but a short time. The paralysis continued without improvement until her death seven years later. The mentality and speech were not affected.

Another patient, a youth of nineteen, had syphilitic endarteritis and hemorrhage. At noon, on a certain date, the patient noticed an inability to pronounce words distinctly. This was temporary, and the patient assigned it to trouble with his throat. Later in the evening the patient began to have difficulty in using his right hand while playing tenpins. He was unable to hold the ball. This was followed by an inability to speak. He made inarticulate sounds which meant nothing. When asked to write what he had to say he wrote a meaningless jumble of letters. On being questioned further he blurted out, "Read it." In a few minutes the inability to articulate passed off in a measure. He acted as one stuttering, speaking slowly and carefully but distinctly. A game of whist was proposed, but the patient was unable to control his right hand and could not hold the cards. Next morning the power of speech was gone. The right side of the body was almost completely paralyzed. He could move the fingers of the right hand a little but could not grasp things. He could walk, but the right leg dragged and he could not lift the foot from the floor. Sensibility on the right side was also dulled.

In this case gradual improvement began almost at once, and after three months the patient was in fair condition. There was

still slight loss of power on the affected side, but he could walk and could use his hand. Speech returned and articulation was distinct; the only defect was that occasionally he would use words associated in some way in place of each other. For instance, he might say bread for butter, wedding for funeral, and so on. Mentality was clear, and he told us it had never been otherwise. He had been conscious at all times while his aphasia lasted of what was going on about him. Finally, about four months and a half after his first attack, the patient had a second to which he succumbed in forty-eight hours. At the second hemorrhage the left side of the body, the previously paralyzed side, had frequent convulsions, whereas the right side became paralyzed.

The above cases are typical. Others again are very mild, there is slight loss of power. If speech is affected that may be slight also. Occasionally a slight attack of apoplexy will cause a mental confusion, and the patient will lose his identity temporarily.

Some cases of mysterious disappearance are undoubtedly of that character. One of my patients, a man aged fifty-six, suddenly dropped out of sight one afternoon. About two weeks later one of his friends got an incoherent letter from him from Boston. When brought home there was some mental confusion still left, and there was decided difficulty in articulation with very slow and deliberate speech. The patient could only recall that on the day he disappeared he had felt well except for a severe headache. He had gone from his office to mail some letters and get a glass of soda water. From then on his ideas were hazy. How he got to Boston he did not know except that he thought he went in a boat. Who he was or why he was there he did not know. He adopted a fictitious name and began looking for work. He finally recalled the name of a friend and wrote him signing his adopted name. When found he was living in a squalid lodging house. The patient finally made a complete recovery, and to-day, five years later, is holding a very responsible position. Sooner or later he will probably have another attack of apoplexy, but it is to be hoped he will not lose himself again.

The course of apoplexy varies in different cases as seen above. The attack may be overwhelming; the patient suddenly loses consciousness, there is stertorous breathing, later Cheyne-Stokes respiration; the pulse is full and hard, slow at first; temperature subnormal at the beginning, rising to 104° F. or even higher just before death; twitchings or convulsions of one-half the body; unequal pupils which may or may not slowly react to light, the eyes converged or turned to one side; evidence of complete paralysis of half the body. Such cases may die very promptly or they may linger for a few hours or even several days without regaining consciousness.

In other cases the onset is slower, the symptoms becoming more and more pronounced for several hours until, like the case cited above, there is aphasia and complete paralysis of one-half the body. Occasionally a case of this kind will go on to death from respiratory paralysis. I had one such case at the Metropolitan Hospital, a woman who gradually became paralyzed in one-half, the left, of the body. There was no loss of consciousness and no aphasia. She died in about thirty-six hours after the onset of symptoms from respiratory paralysis.

By far the most frequent type is where loss of consciousness is more or less abrupt and lasts for a few hours to a day or two. Then consciousness returns, but one-half of the body is kept paralyzed. The affected side of the face loses its normal expression because its natural lines are obliterated. The tongue deviates to the affected side when protruded. There may be difficulty in swallowing because of the paralysis of the tongue and throat. The arm and leg of one side, usually the side opposite to the paralyzed side of the face, are paralyzed. The muscles are flaccid and show no resistance to passive motion. The deep reflexes are exaggerated on the affected side. There may or may not be loss of sensibility on that side. If the lesion is in the cortex convulsions of half the body may occur.

If the hemorrhage occurs on the left side of the brain there is some form of aphasia, depending on the exact location of the lesion. The right side of the body is paralyzed. If the hemorrhage occurs in the right side of the brain there is no aphasia.

In these cases the symptoms reach their maximum in a few hours or a day or two. They remain stationary for a time, then in the majority they begin to improve and, after several months, there may be a return to an almost normal condition. The leg shows signs of improvement first, then the arm. If improvement does not set in within six to eight weeks, the paralysis remains permanent.

The emotional nature is practically always affected. The patient becomes a little hysterical at times, and cries frequently without obvious cause. I believe with Marie that the mentality is always affected more or less where there is aphasia, and that it never quite returns to normal. There is sometimes a complete change of character—one case of mine became suspicious.

Strümpell says although no one is exempt from apoplexy, yet there is a type of individual of "apoplectic habit," they are short and stout with thick necks and fond of eating and drinking. Sometimes prodromal symptoms occur, headache, flushed face, vertigo, tinnitus aurium, spots before the eyes and muscular weakness.

Complications and Sequelæ: In cases that do not improve, or that improve only in part, the paralyzed muscles eventually atrophy to some extent. If patients are bed-ridden, bed sores may result if the nursing is not of the best. Contractures of permanently paralyzed muscles may result. Mentality may eventually fail.

Diagnosis: In the unconscious stage apoplexy must be differentiated from sunstroke, acute alcoholism, uremic coma. The last is the one most frequently mistaken. Uremia usually has a distinct decrease in the secretion of urine. There is no difference in the condition of the two sides of the body. The pupils are even, the eyes do not deviate to one side. In uremia the pulse may be rapid, in apoplexy it is apt to be full, hard and slow at the beginning.

It must not be forgotten that apoplexy is sometimes the end of Bright's disease. It may also occur in association with sunstroke or with alcoholism.

Prognosis: This must always be guarded. If the patient re-

covers consciousness after a time, he may go on and recover a certain amount of use of the paralyzed limbs, or even recover completely. There is always a probability that a patient who has suffered one attack of apoplexy will suffer another.

Treatment: Absolute rest in bed is the first essential. Mental quiet is also of importance. Care must be taken that no bed sores develop. Baths not too hot—90° to 95° F.—and medicated with salt, may be given every other day.

Electricity may be tried for the paralysis. After the acute symptoms have subsided, at the end of a month, massage may be used to prevent contractures.

Aconite may be given in the beginning if the patient is restless and apprehensive. I have seen marked benefit from its use. *Aconite* 200 relieved the following symptoms in one case, expects to die before morning; very restless, expects to die before night.

Belladonna is indicated in congestion of the face with full, bounding pulse.

Arnica is of service, especially in traumatic cases.

Opium in potency may be of service in the stupor.

Ferrum phosphoricum. If an attack seems to be impending the head feels full, there is severe headache.

Glonoin. Vertigo even on turning in bed; frontal and occipital headache as though it would kill her. Light aggravates. Nausea.

Strychnia phosphorica is valuable in the various paralyses.

PACHYMENINGITIS.

Definition: Inflammation of the dura mater of the brain and cord.

Historical Note: First described by Charcot in 1871.

Etiology: Exposure, traumatism, anemia, alcoholism, syphilis, disease of the skull or vertebræ.

Morbid Anatomy and Pathology: The dura is much thickened. There is an increase in the connective tissue. There may be indications of past hemorrhages. In the cervical region the thickening may be so great as to cause pressure enough to produce secondary degeneration of the cord.

Symptoms: There is pain in the neck and arms. There is headache with excitement and restlessness. There may be apoplectiform convulsions with transient aphasia. There may be choked discs. Often there is numbness, paresthesia or anesthesia, especially of the arms and hands. There may be paralysis after some weeks.

Complications and Sequelæ: The condition may cause bed sores. It may end in convulsions.

Diagnosis: This is difficult. The symptoms may come on suddenly.

Prognosis: Bad.

Treatment: Rest in bed is essential.

Medicines must be prescribed symptomatically.

Aconite, apis, belladonna, bryonia, gelsemium, each should be studied.

LEPTOMENINGITIS.

Synonym: Purulent Meningitis.

Definition: An acute inflammation of the pia mater of the brain and cord extending sometimes to the dura and cord itself.

It is rarely a primary affection, but most often secondary to the acute infections or to inflammatory processes in the neighborhood.

Cerebro-spinal meningitis is the most common form and has been described elsewhere.

A chronic form occurs in other nerve diseases.

Etiology: Most often occurs in the very young. Other forms occur as the sequelæ of typhoid fever, pneumonia, influenza, gonorrhea, or other infections.

Morbid Anatomy and Pathology: First there is an infiltration of the coats of the arteries, then an exudation and thickening of the pia. This is followed by serous effusion causing pressure.

Symptoms: The symptoms resemble those of cerebro-spinal meningitis. There is stiffness of the neck and back with severe pain. Headache is a prominent symptom. The vertebræ are sensitive. There is hyperesthesia. All the senses are acute.

There may be delirium, convulsions, or spasms. There is often strabismus. There is some rise in temperature. Respiration is disturbed.

Diagnosis: Headache is always a prominent symptom. Other diseases must be eliminated.

Prognosis: The prognosis is bad. But few cases recover. The disease may run its course in a few hours, more often in a few days.

Treatment: Warm baths may give some relief. Lumbar puncture may be done to relieve pressure.

Aconite is useful at the beginning.

Apis is valuable to control the exudate.

Belladonna may be given if the patient is flushed and has a full, bounding pulse.

Gelsemium is more apathetic.

Hellebore has an unconsciousness with boring the head in the pillow. There is the "cri encephalique."

Veratrum viride, face flushed, head retracted, pupils dilated, pain in nape of neck.

TUBERCULAR MENINGITIS.

Synonyms: Basilar Meningitis. Acute Hydrocephalus.

Definition: Meningitis caused by tubercular infection. Usually the base of the brain is the seat of the lesion, hence the term "basilar" meningitis.

Historical Note: In 1827 Guersant noted what he called granular meningitis. In 1830 Papavoine described tubercles found in two of his meningeal cases, and called attention to the fact that tuberculosis existed in other parts of the body as well. Gerhard, of Philadelphia, in 1833, was the first to call attention to it in this country.

Etiology: Tubercular meningitis is always secondary to a tubercular focus in some other part of the body. It most commonly occurs in subjects of pulmonary tuberculosis. It is a part of all cases of miliary tuberculosis.

Children are more often affected than adults. In children the

original trouble may be tuberculosis of the glands, bones or joints.

Bacteriology: As in other forms of meningitis, the membranes of the cord are likewise affected. Lumbar puncture will give a fluid that may be normal in appearance. Cultures are best made from the sediment thrown down by the centrifuge. In tubercular meningitis the lymphocytes are in excess. Tubercle bacilli will be present, though they may be few and hard to find.

Morbid Anatomy and Pathology: Tubercles may be found in great number over the entire surface of the brain, as in the case of a young girl, a patient of mine at the Metropolitan Hospital. There may also be an inflammatory condition with considerable exudation, especially in the ventricles.

Symptoms: There may be an initial period of restlessness, headache varying in severity from time to time, loss of appetite, constipation, and sometimes vomiting. There may even be a certain amount of delirium early in the disease. The patient is pale, peevish, and tends to grind the teeth during sleep. Emaciation is the rule. Young children often complain of pain in the abdomen.

As the disease progresses the symptoms become more pronounced. The headache becomes more severe, intelligence becomes more and more clouded until there is either wild delirium or complete coma. There is tenderness at the nape of the neck, the neck and spinal column become stiff. Light and noise may disturb the patient. Sometimes he emits short, sharp cries, the hydrocephalic cry. Emaciation also becomes more pronounced.

Ptosis, strabismus, and lateral movements of the eyes appear. The pupils may be uneven, or they may contract and expand. The ophthalmoscope may reveal choked disc. There appear all sorts of combinations of twitchings of muscles or groups of muscles, even convulsions, local or general; in other cases there may be a rigidity of the extremities, in others again there is paresis. The reflexes are at first exaggerated, later diminished.

The pulse is apt to be slow, sometimes variable. It becomes very rapid at the close. Respirations are usually rapid and irregular. The temperature rarely gets above 102° F., sometimes it is subnormal, especially near the end.

Strümpell reports having seen a temperature of 87.8° F. just before death.

Diagnosis: Diagnosis is sometimes difficult or impossible in the beginning, and only to be made after further progress of the case. Gastric symptoms with slow pulse should excite suspicion. The fully developed case is easily recognized as meningitis. The exact type depends on the finding of other tubercular lesions in the body. Finally, lumbar puncture and examination of the spinal fluid may be necessary.

Prognosis: Tubercular meningitis is practically always fatal and rarely lasts longer than three or four weeks. Some patients die within a few days.

Treatment: General hygienic care should be instituted.

Shaving the scalp and giving inunctions of iodoform ointment is the only procedure for which cure has been claimed.

Homœopathic medication may relieve some symptoms temporarily.

CHRONIC HYDROCEPHALUS.

Synonym: Water on the Brain.

Definition: An abnormal accumulation of fluid in the cranium. It may be congenital, the form described below, or it may come on in adult life when the symptoms simulate those of brain tumor.

Etiology: Unknown exactly. Syphilis or alcoholism in the parents have been supposed to cause it. It seems to run in families.

Morbid Anatomy and Pathology: The skull enlarges in circumference. The frontal and parietal eminences are most prominent. The bony structure becomes thin, the sutures and fontanelles open. The ventricles of the brain are distended, thinning the brain tissue. The fluid is colorless and has a specific gravity of 1.004 to 1.006. There may be as much as a quart.

Symptoms: The most evident symptom is the enlargement of the cranium. The child's head may be so large at birth as to interfere with labor. Or it may be of normal size at first. Then it begins to enlarge, sometimes very slowly, sometimes more rapidly. Nearly always the enlargement is symmetrical. The

forehead becomes very prominent and then overhangs. The face does not enlarge. The head looks and is out of proportion.

With this enlargement of the cranium there is nearly always a lack of mental development. The child is backward in learning to walk and to use its arms and hands. Sensation is rarely disturbed.

There may be choked disk or optic atrophy. Sometimes the patients have convulsions.

Diagnosis: A typical case can be easily diagnosed. A rachitic skull may be enlarged, but in that case the mentality is not interfered with.

Prognosis: Some cases become arrested and the patient reaches adult life. They may or may not have an impaired mentality. The majority die during childhood.

Treatment: Mainly hygienic. Some favorable results have been obtained by lumbar puncture.

Homœopathic remedies are invaluable.

Calcarea carbonica for the typical *calcarea* baby.

Calcarea phosphorica for the lack of ossification.

Helleborus and *sulphur.*

ABSCESS OF THE BRAIN.

Synonym: Suppurative Encephalitis.

Definition: Literally, this is an inflammation and breaking down of the brain substance, usually secondary to pus elsewhere in the body.

Historical Note: As knowledge of suppuration and the organisms causing it has become clearer, cases of true inflammation of the brain have become fewer. It is recognized now that abscess occurs secondary to pus infection elsewhere.

Etiology: Traumatism of the head is the most frequent cause of brain abscess, infection occurring probably through an open wound. Foreign bodies introduced by direct injury may cause abscess. Extension from neighboring parts, as middle ear disease, less rarely from the nose. Occasionally by metastasis from infective endocarditis, or from the lungs.

Bacteriology: The streptococci, staphylococci or pneumococci are the organisms most frequently found.

Morbid Anatomy and Pathology: Brain abscesses are like abscesses elsewhere. They may be single or multiple. If large the surrounding brain substance suffers softening. Sometimes long standing abscesses are found encapsulated.

Symptoms: The symptoms vary with the location of the abscess. In some cases there may be no symptoms for a long time.

The general symptoms are rather vague. There may be headache, vertigo, and occasional vomiting. Sometimes irregular fever with slow pulse. There may be choked disc. There may be loss of weight, slight mental depression, and the patient does not feel well. If the abscess grows larger progressive paretic symptoms may develop, sometimes with convulsions. There may be more or less stupor. Hemianopsia, aphasia, staggering gait (in cerebellar cases) are all symptoms.

Diagnosis: May be very difficult. Chronic middle ear disease may simulate it. Unless localized symptoms are produced the general symptoms may resemble purulent meningitis.

Prognosis: Is always grave. If due to middle ear disease, operation may relieve.

Treatment: If sure of an abscess, and it can be located by the symptoms, operative interference is the only cure.

TUMORS OF THE BRAIN.

Definition: Tumors of various kinds may occur in the brain. The main ones are: (1) Glioma—a form of tumor originating in the neuroglia. (2) Sarcoma. (3) Gumma—syphilis. (4) Tubercles. (5) Carcinoma. (6) Cysts.

Etiology: Most brain tumors are secondary to similar tumors elsewhere in the body. There may be some unknown congenital predisposition to their formation. In many cases they seem to start from trauma. They are more frequent in early adult or middle life. Men are more often affected than women.

Morbid Anatomy and Pathology: This depends on the character of the tumor. If of large size pressure causes a flattening of the convolutions. The veins are compressed, causing effusion

into the ventricles. There is softening of the brain tissue about the tumor.

Symptoms: Headache is the most pronounced symptom. This is constant and persistent, but may be worse sometimes than at others. It is dull and deep-seated, as a rule. Occipital headache sometimes indicates tumor in the posterior fossa.

Optic neuritis or choked disk is a very common symptom occurring in four-fifths of the cases. Later on vision may become affected, due to atrophy of the optic nerve.

Vertigo may occur. If marked it usually means that the tumor is in the cerebellum. The pulse is apt to be slow on account of the cerebral pressure, sometimes irregular. Vomiting may occur.

Convulsions, either unilateral or general, are liable to happen if the tumor is near the cortex.

The mental condition becomes changed. The patient looks dull and apathetic. Speech is slow. The patient loses interest in things and people and becomes untidy.

With these general symptoms the patient loses flesh and strength.

There are other more particular symptoms varying with the location of the tumor in the brain. If the hemispheres are the seat of the growth, we get localized convulsions, later monoplegia. If the lesion is on the left side there may be aphasia.

The following summary of focal symptoms of brain tumors, according to location, is taken from Strümpell's Text-Book on Medicine. It was prepared by Knapp:

Prefrontal Region:—Marked mental impairment (abnormal attempts at wit); symptoms of invasion of the central region (Jacksonian epilepsy, aphasia); staggering gait; disturbances of smell.

Central Region:—Jacksonian epilepsy; monoplegia; tactile hyperesthesia and loss of stereognostic sense; motor aphasia.

Posterior Parietal Region:—Word blindness; disturbances of muscular sense; homonymous hemianopsia.

Occipital Region:—Homonymous hemianopsia; soul-blindness.

Temporo-Sphenoidal Region:—Often a latent region. Word deafness if the left side be affected; disturbances of taste, smell and hearing.

Corpus Callosum:—Often a latent region. Progressive hemiplegia, often bilateral from invasion; mental disturbances.

Optico-Striate Region:—Hemiplegia; contracture. In posterior part, hemianesthesia, homonymous hemianopsia, post-hemiplegic chorea, athetosis.

Crus-Cerebri:—Crossed paralyses of the oculo-motor nerve and limbs.

Corpora Quadrigemina:—Oculo-motor paralyses; reeling gait; blindness; deafness.

Pons and Medulla:—Crossed paralyses of face and limbs, or tongue and limbs. Other cranial nerve lesions.

Cerebellum:—Marked cerebellar ataxia; marked vertigo and vomiting.

Base, Anterior Fossa:—Mental impairment; disturbances of smell and vision; exophthalmos.

Base, Middle Fossa: Disturbance of vision; oculo-motor disturbances; hemiplegia.

Base, Posterior Fossa:—Trigeminal neuralgia; neuro-paralytic ophthalmia; paralyses of face and tongue; disturbance of hearing; crossed paralyses.

Hypophysis:—Disturbance of vision, especially bitemporal hemianopsia; oculo-motor disturbances; acromegaly.

Complications and Sequelæ: The development of brain tumors is slow. As they increase in size the various spasms and paralyses develop, as noted above. The mentality becomes more and more impaired. Sometimes a case terminates suddenly from hemorrhage.

Diagnosis: The slow development of the symptoms, especially persistent headache, vomiting, vertigo, etc., should suggest cerebral tumor.

Abscess, as a rule, does not present choked disk. There is usually a definite cause for abscess.

Syphilis will, of course, lead one to suspect the symptoms to be caused by syphilitic gumma.

Large tubercles may simulate tumor.

Prognosis: Except for syphilitic gumma, tumors of the brain are fatal after a longer or shorter time. Sometimes gumma may be cured by active antisyphilitic treatment.

Treatment: The patient should be put under the best general hygienic measures possible.

In case of syphilis anti-syphilitic treatment must be instituted. It must be radical. Mercurial inunctions up to seventy-five grains daily may be tried. Also large doses of *potassium iodide*. *Salvarsan* may also be tried.

If the tumor is decided to be near the surface surgical interference may be tried. Opening the skull simply to relieve pressure is justifiable in desperate cases.

In all cases symptomatic prescribing may ameliorate the discomfort.

PARANOIA.

(Greek, παρα, opposite of; νοεω, to think.)

Synonyms: Monomania. Systematized Delusional Insanity.

Definition: A mental disease characterized by delusions arising without apparent cause, which become fixed ideas.

Etiology: Usually occurs in members of neurotic families. Worry, dissipation, or mental shock may act as exciting causes.

Symptoms: Paranoia is of insidious onset though some sudden foolish act may first call attention to it. The patient has often usually been known, for a longer or shorter time, as somewhat peculiar, or neurotic. He is self-centered and touchy.

Then he develops some delusion. Most frequently he suffers from delusions of persecution. He begins by being insanely suspicious. He imagines he is being talked about, or that people look at him with suspicion, and so on. Then he thinks people malign him, or are trying to poison him. Later the supposed conspiracy against him assumes large proportions in the patient's mind, everybody is against him.

Hallucinations, mostly of hearing, occur. Usually the voices have something disagreable to say. The patient is usually defiant towards his supposed adversaries. He may put his troubles up to some particular person and attempt to punish him by killing or in some other way.

There may be delusions of exaltation, when the patient thinks himself God or some great personage. Or the patient may become excessively pious. Or he may develop erotomania.

Masturbation and sexual perversion are common.

Diagnosis: The diagnosis is based on the fixed idea which has been reasoned out, together with his egotism and defiant attitude.

The melancholic's fixed idea is one of depression and a feeling that he is being properly punished for some past delinquency.

Prognosis: Usually results in dementia.

Treatment: Should be institutional.

DEMENTIA.

Definition: A more or less complete enfeeblement of the mental faculties of a mind that was once normal.

Etiology: May be due to disease of the brain, as syphilis, tumor, etc., to old age; as the end of other forms of insanity, due to epilepsy, etc.

Symptoms: Loss of mental power. The judgment is impaired. The patient becomes forgetful, then the memory goes altogether. They are emotional, grave or gay without reason. They stray away and get lost. They become disoriented. They become careless and filthy in their habits. They may become obscene, masturbate, etc.

They have delusions and hallucinations.

Diagnosis: Easy.

Prognosis: Bad.

Treatment: Institutional.

SECTION XIV.

Diseases of the Nervous System.

NEURALGIA.

Definition: Nerve pain. All pain is nerve pain, but neuralgia, as a disease entity, is a pain that follows some particular nerve trunk, is usually acute, shifts from one part to another, and occurs in paroxysms.

Historical Note: Valleix described sensitive spots in 1841.

Etiology: Predisposing causes are middle life, the more frequent occurrence in neuropathic families, and a general run-down condition, due to illness, overwork, worry, or improper living.

Certain forms of neuralgia, as sciatica, occur more frequently in men. Other forms, especially facial neuralgia, occur more often in women.

Exciting causes are exposure to cold and dampness. Traumatism. Pressure from growths or foreign bodies. The infectious diseases are sometimes associated with or followed by neuralgia. Constitutional diseases, as gout and diabetes, may cause it. Certain intoxications, as alcoholism, lead or arsenic poisoning. Autointoxications. Reflex neuralgias, that is, neuralgia due to some distant organ. Eye strain, or carious teeth may cause attacks of neuralgia.

Morbid Anatomy and Pathology: Not known.

Symptoms: Neuralgia may be preceded by some indefinite sensations of aching or prickling. More often it comes on quite suddenly, is acute in character, and lasts from a few moments to several days, with intermissions. The pain may be an ache, or it may be boring, lancinating, or burning in character. It usually follows the course of some nerve so definitely that the nerve can be outlined by the patient. There are definite spots sensitive to touch where the nerves come near the surface of the body.

During the paroxysms cold air, mental excitement, or movement of the affected part may increase the pain. There may be anesthesia or hyperesthesia of the skin. There is frequently motor irritation. Sometimes vaso-motor disturbance. For example, there may be sweating. One of my patients, during an attack of facial neuralgia, will sometimes have the face "set" and immovable, giving a curious expression to the countenance. In severe and protracted cases there may be a whitening or falling out of the hair, or a change in the character of the skin. In some cases there will be an urticaria or a herpes. Herpes zoster, for example, most common with intercostal neuralgia may appear on other nerve trunks. I have seen it in sciatica.

Certain of the more common forms of neuralgia may be mentioned specifically.

1. *Facial Neuralgia. Neuralgia of the Trigeminus. Tic Douloureux. Prosopalgia.*

This form may affect one or more branches of the trifacial nerve, usually on one side only.

During the attack the teeth may be very sensitive.

It may be caused by disease of the ear, nose, or teeth, or possibly by eye strain, and all these must be carefully considered when treating a case.

The tender points to be sought are over the supraorbital and mental foramina.

2. *Occipital Neuralgia.* Involves the upper cervical nerves and usually both sides. Aneurysm, growths or spinal caries may cause it. The tender point is midway between the mastoid process and the upper cervical vertebræ.

3. *Neuralgia of the Brachial Plexus.* Is not limited so closely to one nerve trunk, but may be more or less general in the cervico-brachial region, or it may extend down the arm or forearm. The most frequent causes are tumors, aneurysm, spinal caries, sometimes arterio-sclerosis and angina pectoris. Strümpell says the amputation neuralgias belong to this class. It may be due to occupation, as piano playing, constant writing, etc.

Tender spots may be found over the brachial plexus, over the radial nerve on the external surface of the upper arm, over

the ulnar nerve at the inner condyle, or over the median nerve at the inner border of the biceps.

4. *Intercostal Neuralgia.* The fifth to the ninth intercostal nerves are most often affected, and more often in women, and on the left side.

Growths or caries of the spine may be at the bottom of it. Sometimes tabes, syphilis or aneurysm. Herpes zoster is an occasional accompaniment. The pain is severe, as a rule, so that the patient restricts the motion of the chest as much as possible.

Tender points near the spine, in the middle and near the sternum.

5. *Mastodynia. Neuralgia of the Mammary Gland.* Occurs almost exclusively among women. The whole breast is tender, and sometimes extremely sensitive nodules are found in it.

6. *Neuralgias in Region of Lumbar Plexus.* These neuralgias are rare. The pain may be in the lumbar region, about the buttocks or genitals, or down the thighs and legs.

7. *Sciatica. Ischialgia.* This is neuralgia of the sciatic nerve and is a very common form.

It may be caused by excessive work, by exposure, by pelvic tumors, sometimes by the pregnant uterus, and occasionally by constipation.

As a rule, sciatica is a prolonged condition; it is apt to recur. The nerve is sensitive to direct pressure. Motion may be practically impossible and the patient bedridden. Atrophy of the leg may occur.

Tender spots are at the middle or lower border of the gluteus muscle, in the bend of the knee, at the head of the fibula.

By flexing the thigh with the leg extended a gluteal pain is caused.

8. Other *neuralgias,* not so common or so definitely localized as the above, may occur in any part of the body. The feet seem to be prone to neuralgic affections and belong to this grouping.

Complications and Sequelæ: Have been mentioned.

Diagnosis: Is usually easy, as the pains are quite characteristic. Care must be exercised in locating the exact cause, if possible and removing it.

Prognosis: Depends to a large extent on the cause.

Treatment: A patient subject to neuralgia should avoid the things that produce it, that is, exposure, etc.

Next the cause should carefully be looked into that any possible disease of neighboring organs, tumors, etc., may be treated or removed.

Sometimes local applications, blisters, etc., are useful.

Electricity in various forms may be of service.

Strümpell says *quinine* is sometimes helpful.

Morphine may sometimes be necessary.

Homœopathic remedies are *belladonna, aconite, cimicifuga, rhus toxicodendron,* etc.

Belladonna—facial neuralgia in paroxysms—could not touch teeth during—not sensitive between—one dose relieved almost instantly. I have verified this in several cases.

Aconite is the remedy in cases due to exposure to a strong wind.

Cimicifuga is useful in neuralgic and rheumatic pains.

Rhus toxicodendron is especially useful in sciatica.

HABITUAL HEADACHE.

Synonym: Cephalalgia.

Definition: Headache, as its name indicates, is pain of the head, or more correctly, of the cranium.

Etiology: Many patients have habitual or frequent headaches, the cause of which cannot be found. In other cases it can be. One of the most frequent causes is eye strain. Other headaches seem to originate in disease of the nose or ear. Frequent headaches occur in nervous people. Headaches occur in anemia and in full blooded persons. They may occur from emotional excitement, worry, or overwork. In the overtired mentally or physically.

The headaches associated with other definite diseases are not considered here. Only those headaches which are habitual and without other definite symptoms.

Symptoms: The pain in habitual headache is usually limited to some particular part of the head. It may be frontal, occi-

pital, parietal, temporal, on the vertex, bilateral or unilateral. More rarely it is general. The pain may vary all the way from a slight discomfort to the most agonizing pain. The patient may walk the floor, or again, may not be able to get off the bed. Light and noise may aggravate. There may be nausea and vomiting.

The pain usually remits, although the subjects have headaches lasting over many years. Some patients will have them every day, recurring at the same hour. Others will have them less often, once a week, or once a month. Sometimes the headache will last for several days at a time.

Patients who suffer from very severe headaches, after the acute pain has subsided, will often have a feeling of soreness in the head.

The scalp may be sensitive to the touch. Indurative headache.

While the pain lasts the patient may be cross and irritable. Little things annoy. Mental effort is impossible. If the headaches are severe and very frequent the whole disposition of the patient may be changed and he may become a nervous wreck.

Complications and Sequelæ: May complicate various conditions. Neurasthenia may follow.

Diagnosis: Must eliminate general diseases, eye strain, etc.

Prognosis: If an underlying cause can be found it may be good. If not, the condition lasts a long time.

Treatment: First, the patient must be carefully studied to find a possible cause for the headaches.

The habits must be regulated. A simple nutritious diet must be prescribed.

"Headache cures" should be taboo; many of them are dangerous of themselves, others may lead to the formation of drug habits.

The homœopathic remedy is invaluable. I will mention a few drugs which I have verified:

Calcarea carbonica. Large blond women who have cold hands and feet with their headaches. Sometimes the patient is hysterical, thinks she will go crazy. Wants to scream and throw things.

Pulsatilla. Fullness cerebellum to shoulders—feels as though she could not turn her head, but does want to make nervous little motions all the time. Bowels irregular, tongue moist, teeth imprint on tongue, face flushed, pupils enlarged. Headache affects vision—glasses do not relieve. Extremities always warm; nervous and irritable.

Cimicifuga. Occipital headache, neck and shoulders.

Veratrum album. Headache brought on by excitement—back of head—aggravated by talking—relieved by pressure—gas in stomach at such times and no appetite. Palpitation.

Gelsemium. In eyeballs—chilly sensation, vomit relieves—hysterical—cries with pain.

Belladonna. Headache at vertex, throbbing, hot. Back chilly. Palpitation.

Bryonia. Headache severe over eyes. Vertigo. Cold cloths tied tight relieve.

Nux vomica. A frontal headache from indiscretions in diet or from catarrhal conditions of the nose.

Glonoin. Headache at base of brain. Suspected to be of uremic origin.

Sanguinaria. Right side of head from right eye to occiput, with nausea and vomiting.

Other remedies that may be thought of are *ferrum phosphoricum, ignatia, lycopodium, mercurius.*

MIGRAINE.

Synonyms: Megrim. Hemicrania. Sick Headache.

Definition: A peculiar form of headache, usually unilateral, and frequently associated with gastric and sensory disturbances.

Historical Note: Migraine has been thoroughly studied by Bois, Raymond and Müllendorff, who all suffered from it themselves.

Etiology: Strümpell says the condition is probably due to congenital predisposition. It most often occurs in neurotic families. Women are more often affected than men. It usually begins at puberty. The patients may be anemic. Frequently there are disturbances of menstruation.

Morbid Anatomy and Pathology: Is unknown.

Symptoms: Migraine returns with more or less regularity. Often it seems to bear some relation to the menstrual periods. There are usually certain prodromal symptoms of general discomfort. There is pressure and vertigo. The patient is chilly and yawns.

During the attack the scalp is hypersensitive. There is loss of appetite, nausea and vomiting. The pain in the head is most often left sided, in the temporal region. Sometimes it occurs in both sides, sometimes it alternates. The patient is dull, mentally. He is sensitive to light and noise. The pupils may be dilated or contracted. Frequently there is ptosis. There are flashes before the eyes; there may be scotoma, sometimes hemianopsia. The face may be pale or flushed. Sweatings occur. Occasionally there may be temporary aphasia, hemiparesis, or hemianesthesia.

As a rule, patients wish to shut themselves up in the dark during an attack. At the end they have a vomiting spell, and sometimes pass a large amount of urine.

Diagnosis: The diagnosis is made from the severe headaches with nausea and vomiting that begin with puberty, and recur with more or less regularity.

Prognosis: Migraine is not dangerous to life. The prognosis is always doubtful as to permanent cure.

Treatment: The patient must be placed under the best possible living conditions, physical and mental.

Narcotics are bad and should be forbidden.

Antipyrine is recommended by the old school.

Strümpell suggests salicylate of soda, 20 to 30 grains in strong black coffee.

The various remedies mentioned under "Habitual Headache" may be tried as indicated.

PARALYSIS.

Definition: Paralysis is loss of voluntary motion in the muscles of the body controlled by the will. Paresis is a weakening of this power.

Kinds of paralysis. *Monoplegia,* paralysis of a single muscle or group of muscles; this may be of cortical or peripheral origin. *Paraplegia* is usually symmetrical and due to a lesion in the spinal cord. *Hemiplegia* is paralysis of half the body and is of cerebral origin.

Etiology: Paralysis may be due to disease of any part of the nerve tract from the cortex of the brain to the muscles. There may be inflammation, degeneration, new growths, hemorrhage or traumatism somewhere along the line. Alcoholism, plumbism and intoxication by other poisons may cause paralysis. It is sometimes a sequel to herpes zoster and the acute infections.

Sometimes a functional paralysis, that is, a paralysis without known pathological cause, occurs. Hysterical paralysis might be called functional.

Morbid Anatomy and Pathology: Depend entirely on circumstances. The paralysis occurs on the distal side of the seat of trouble, wherever that may be.

Symptoms: Paralysis may affect any single muscle or group of muscles, or it may be more or less general. The affected muscle atrophies. If the muscles are flaccid, passive motion is possible. If the muscles are spastic then passive motion is not possible. The skin over the paralyzed part may be red and shiny. It is smooth and without the lines of normal skin.

The following special paralyses may be mentioned:

1. *Paralysis of the Eye.*

Etiology: Injuries, pressure from injuries or growths, rheumatic and other diseased conditions.

Symptoms: The eye appears prominent. There is ptosis. The pupils are unequal. There is inability to move the affected eye. The healthy eye deviates. As a consequence the patient sees double.

Diagnosis: Sometimes ptosis occurs during severe headache. Sometimes alcoholism is so profound as to cause lack of co-ordination of the eyes and the victim sees double. In extreme weakness near the end of life sometimes lack of co-ordination appears. I have met with each of these conditions.

2. *Paralysis of Muscles of Mastication.*

Symptoms: The patient cannot chew his food. The seat of trouble is near the base of the skull.

3. *Facial Paralysis—Bell's Palsy.*

Etiology: This may be caused by disease of the brain or medulla. It may be caused by cold. It may be caused by middle ear disease.

Symptoms: A queer expression is produced because only one half the face is mobile, the other half is set. The eye on the affected side does not close. Speech is interfered with. The tongue deviates to one side when protruded. The patient cannot whistle. Contractions of varying degree may ensue.

4. *Paralysis of the Shoulder.*

Symptoms: The affected shoulder drops. It is difficult or impossible to move the arm.

5. *Paralysis of the Back and Abdomen.*

This only occurs in extensive general paralysis.

6. *Paralyses of the Upper Extremity.*

Symptoms: These depend somewhat on just where the lesion is located. The patient may not be able to raise the arm. There may be wrist drop. There may be wasting of the thenar muscles. These last two symptoms cause deformity of the hand. I have seen temporary paralysis of the arm due to traumatism. I have seen it caused by herpes zoster.

7. *Paralysis of the Diaphragm.*

This is always serious because of its interference with respiration.

It may be caused by traumatism. It may be caused by rheumatism.

8. *Paralysis of the Lower Extremity.*

Symptoms: The gluteal fold is lost on the affected side. There is foot drop. The patient cannot walk, or, if he does, the affected limb drags.

Treatment: The affected part may be rubbed and massaged. Some form of electrical treatment may help.

Plumbum is a valuable remedy at times.

Strychnia phosphorica in potency, the 2x or 3x, often helps. It acts as a nerve stimulant.

SPASM.

There may be spasm of the different muscles or groups of muscles. The spasm may be tonic, where the muscle affected remains contracted; or it may be clonic, with alternate contraction and relaxation.

The result of the spasm depends, of course, on the location. Probably the most frequent form is facial spasm or *convulsive tic*. The spasms cause the patient to make peculiar grimaces. Sometimes this is associated with spasms of the neck so that the position of the head is abnormal also.

In all cases the cause must be sought and, if found, removed. There may be some local irritation that can be eliminated. The spasm may be the result of occupation, which will have to be suspended or stopped altogether. In other cases hysteria or some other disease is the basis of it.

OCCUPATION NEUROSES.

Synonym: Professional Spasm.

Definition: This is a disturbance of coördination in a muscle or group of muscles that is used constantly. For example, writer's cramp, piano player's cramp, telegrapher's cramp, all of which occur in the hand or arm. Ballet dancers, and sewing machine operators may have a similar condition in the feet and legs. Players on wind instruments sometimes have tongue cramp. The affected part can be used for other purposes than the regular vocation.

Etiology: The professional cramp is caused by the excessive use of certain muscles. It is said to be due to faulty methods of using them, as all persons engaged in similar work do not suffer from occupation spasm. The condition occurs usually in neurotic subjects. Fright or anxiety may be exciting causes. Men are more often affected than women.

Morbid Anatomy and Pathology: The condition is probably due to cortical irritation. No permanent changes are found.

Symptoms: Writer's cramp. The most common form for the trouble to take is the spastic, *i. e.*, the fingers are seized with

tonic or clonic spasms as soon as the patient tries to write. It is almost or wholly impossible for him to write—if he succeeds it is only by great effort, and then the writing is irregular and distorted. Sometimes the pen is held so tightly it cannot be removed.

In other cases a tremor of the hand and forearm may develop making it impossible for the patient to write.

In still others the arm becomes rapidly tired and writing is impossible.

There is sometimes a feeling of numbness or tingling. Occasionally the hand and arm become hot and flushed and the veins prominent.

The hand can be used ordinarily for other things the same as usual. Once in a while piano playing or sewing will produce similar symptoms in the same patient.

Diagnosis: Easy. One must differentiate from chorea, paralysis agitans, etc. Nervous persons sometimes imagine they have cramps of occupation.

Prognosis: Usually chronic, though cured eventually. The patient may have to change occupation.

Treatment: Rest is essential, at least from the usual occupation. Massage and manipulation and systematic exercises may do much.

Galvanism is of service in some cases.

Aconite has seemed to be of help in some cases of writer's cramp, especially when there was an associated neuritis.

Argentum metallicum is useful in cases with marked cramp of the fingers.

Sulphuric acid has tremor of muscles and writer's cramp.

NEURITIS.

Synonym: Inflammation of the Nerves.

Definition: Neuritis, inflammation of the nerves, may be *localized,* it may affect a single nerve; or it may be general, that is, it may affect many nerves, *multiple neuritis.*

Etiology: The great majority of my cases have been in women betwen the ages of 25 and 55.

Local causes are exposure to cold, traumatism, extension from disease of neighboring organs. Other causes are the infectious diseases, particularly tuberculosis, of which I have seen many cases; leprosy. Various poisons, as alcohol, lead, arsenic, etc.

Morbid Anatomy and Pathology: The nerve or its sheath may be red, swollen and inflamed. The axis cylinder and medullary sheath may be destroyed. Atrophy of the muscles may occur late. Complete recovery may take place.

Symptoms: In *localized neuritis* the principal symptom is the localized pain in the affected nerve, and more or less inability to use the part fed by the nerve. Any single nerve or group of nerves may be affected.

The pain develops first. The nerve is also sensitive to pressure. Later motor weakness develops; it may go on to paralysis. This may be followed by atrophy of the muscles. There may be redness and edema of the part. Numbness and tingling are common sensations. Sensibility may be lessened.

In rare cases—following injury—the neuritis may travel toward the spine.

There may be the reaction of degeneration.

Multiple neuritis: Polyneuritis.

The onset is frequently sudden. The patient will suddenly have acute pain and become helpless, sometimes falling, the onset is so abrupt. There is fever, with the usual loss of appetite, etc. There frequently is considerable pain. Sometimes the jonts are somewhat swollen. Loss of voluntary use of the arms and legs soon ensues. The patient cannot walk and has to lie in bed. He cannot use his arms and has to be fed, etc. Several of the cases that I have seen have been attacked while away from home and been taken from the street in an ambulance.

Pain may be general and the patient be hardly able to bear the weight of the bed clothes. There may be only tingling of the affected part.

Wrist drop and foot drop soon develop. Later there may be great atrophy of the muscles and contractions.

The reflexes are diminished.

Electrical excitability is lessened or lost. There is delayed sensation of pain.

In the alcoholic type the patient may have delirium tremens, or he may be disoriented, that is, he may lose track of time and place. I have never met with this, my cases have been mentally clear.

There is steppage gait, the foot is lifted and slapped on the floor.

Complications and Sequelæ: Neuritis is of itself a sequel. The contractions usually disappear as the patient improves.

Diagnosis: Usually easy. The history of the patient—addiction to alcohol, a worker in poisons, a subject recently of acute infectious disease, together with wrist drop and foot drop, ought to make the diagnosis clear.

Prognosis: The books say patients may die in a few days or two weeks in severe acute cases—because of extension to the respiratory muscles. I have never seen a case die—and I have seen many cases at the Metropolitan, especially in the tuberculosis wards. All got well eventually. They lasted from a few months to two years.

Treatment: Rest in bed is necessary. In fact, subjects of multiple neuritis are helpless and cannot be out of bed. Plenty of good nourishing food must be given.

Old school books recommend *arsenic* and *strychnia.*

Aconite is by far the most useful homœopathic remedy in this condition and must be continued for a long time.

Belladonna may be indicated if there is much fever and the patient very sensitive to touch.

Nux vomica is useful in alcoholic cases.

Cimicifuga may be given if the pains are rheumatic in character.

NEW GROWTHS.

Synonyms: Neuroma. Neuro-fibroma.

Definition: Growths on nerves or their sheaths. Various forms of new growths, especially sarcoma, may attack the nerves. They cause few symptoms in some cases, severe neuralgic and paralytic symptoms in others.

Surgical treatment is the only one that gives relief.

ERYTHROMELALGIA.

Synonym: Red Neuralgia.

Definition: "A chronic disease in which a part or parts, usually one or more extremities, suffer with pain, flushing, and local fever, made far worse if the parts hang down."

Historical Note: First described by Weir Mitchell in 1872.

Etiology: More men probably affected.

Morbid Anatomy and Pathology: Unknown. Mitchell found neuritis in some cases, in others arterio-sclerosis.

Symptoms: The condition is found most often in the feet. It may be in the hands. There is pain, swelling, redness and pulsation. There is excessive sweating of the part.

Occasionally there are other symptoms, as headache, vertigo and redness.

Prognosis: This is doubtful.

Treatment: Stretching of the affected nerves is recommended.

Homœopathic remedies might cover the case. *Belladonna, mercurius, silica,* or other drugs may be indicated.

REYNAUD'S DISEASE.

Synonym: Spontaneous Symmetrical Gangrene.

Definition: There is vascular change, ischemic or passive congestion, which progresses to loss of vitality and necrosis.

Historical Note: The condition was first described by Maurice Reynaud in 1862.

Etiology: It may appear at any age, in women more often than in men, and in neurotic persons with a neurotic family history.

Morbid Anatomy and Pathology: There is local syncope of the part. Then local asphyxia. Then death of the part.

Symptoms: The local syncope may last a few minutes to a few hours. It may attack one finger or toe or several. Or it may attack the ears, buttocks or chin. It is usually symmetrical. There is tingling to severe pain. The color is lost. The temperature of the part is lowered.

The local asphyxia causes the part to become darker than nor-

mal. This may last a short or a long time. It may subside and recur. It may go on to necrosis.

It may last years. The patient may lose a little at each attack.

Complications and Sequelæ: Amblyopia. Aphasia. Hemiplegia. Epilepsy. Hysteria.

Diagnosis: Exclude gangrene from other causes, as diabetes, etc.

Prognosis: Doubtful.

Treatment: Wrap the parts to keep them warm, cold is inimical.

An elastic bandage applied a few minutes and suddenly released may assist a flow of blood to the part.

Secale causes gangrene, it may cure it.

Some of the deep-acting snake poisons, as *crotalus* and *lachesis,* may be of service.

ACUTE ANGIONEUROTIC EDEMA.

Synonyms: Quincke's Disease. Acute Circumscribed Edema.

Definition: Circumscribed edematous swellings, usually in tissues readily distended, as the eyelids, lips, etc.

Historical Note: First described by Quincke.

Etiology: Family predisposition. Patients subject to urticaria. Stellwagon says it occurs most often in those with weak digestion and easily disturbed nervous system. Articles which disturb digestion seem to bring it on in many.

Morbid Anatomy and Pathology: It is a vaso-motor neurosis.

Symptoms: There may be indefinite prodromal symptoms, malaise, nausea, etc. More frequently the attack comes on suddenly without warning. The eyelids, ear lobes, and lips are favorite places for the swelling to appear. Sometimes the genitalia or other parts are affected. Attacks recur. One of my patients has frequent attacks, sometimes several times in a month. The eyes will be almost closed at one time, at another the upper lip will be swollen and stiff. The swellings come on suddenly and usually last but a few hours to a day.

Diagnosis: The diagnosis is made from the sudden appearance and disappearance of the swelling. Edema associated with other diseases is more or less permanent. Angio-neurotic edema usually occurs in nervous subjects.

Prognosis: Rare cases have been reported where the edema suddenly attacked the larynx, causing death. Ordinarily the prognosis for given attacks is good, but the tendency is to recur at more or less frequent intervals.

Treatment: Must be general, building up the patient.

Nux vomica to tone up the digestion has served me best.

Apis is sometimes useful, it is a valuable remedy in edema of any sort.

PROGRESSIVE FACIAL HEMIATROPHY.

Definition: Progressive wasting of one side of the face, including the soft tissues and even the bones.

Etiology: Unknown. It usually begins early in life, and women are more often affected than men. It may start with some trifling injury.

Morbid Anatomy and Pathology: Mendel found a neuritis of the facial nerve.

Symptoms: The disease occurs more often on the left side of the face than on the right. There is a sharp division between the two sides at the median line. There is a change in the color of the skin. Then the fat begins to disappear, the bones and the muscles to atrophy. The teeth and hair on the affected side fall out. The eye becomes sunken. The tongue and palate become affected also. Occasionally the condition extends to the shoulder and arm. Sensibility, as a rule, is not affected.

Complications and Sequelæ: The disease causes a permanent deformity.

Diagnosis: The condition must be distinguished from other atrophies.

Treatment: The condition is incurable. Electricity may be tried to stop its progress.

PRESSURE PARALYSIS OF CORD.

Definition: Disease of the spinal cord, due to pressure.

Etiology: There may be pressure on the cord from many causes:

1. Hemorrhage—spontaneous or traumatic—great exertion.
2. Injuries to back—displaced or broken vertebræ cause the trouble.
3. Caries of the vertebræ—tuberculosis.
4. Tumors—carcinoma, sarcoma, etc.

Morbid Anatomy and Pathology: Varies, of course, with the disease or injury.

Hemorrhage occurs most often in the gray matter of the cord in the cervical or lumbar region. If hemorrhage is due to disease the pathology will be that of the underlying condition.

In case of fracture or dislocation due to traumatism, or in case of spondylitis, the condition is obvious.

Symptoms: The symptoms vary with the location of the lesion. In case of hemorrhage or severe accident they come on suddenly. When caused by new growths or by disease of the vertebræ the symptoms are of slower development.

In acute cases there is pain. In all, paralysis eventually develops below the seat of pressure. It may be immediate in case of accident. The bladder and rectum may be paralyzed.

In cervical injuries the temperature is very high. In dorsal injuries it is subnormal.

Complications and Sequelæ: If the case progresses there may be bed sores and cystitis.

Diagnosis: Except in case of accident exact diagnosis may be difficult. All symptoms must be carefully weighed.

Prognosis: Varies with the cause. In case of hemorrhage death may occur quickly. If not there may be improvement, possibly complete recovery.

Treatment: In case of accident treatment is surgical.

In hemorrhage the cord may be aspirated.

If due to disease this must be treated to get relief from the spinal symptoms.

COMPRESSED AIR ILLNESS.

Synonyms: Caisson Disease. The Bends.

Definition: Disease due to stay in excessive atmospheric pressure.

Historical Note: Triger, an engineer who built the first caisson in 1839, noted that some of the workmen complained of pains in the extremities. In 1878 Paul Bert promulgated his theory as to what takes place.

Etiology: Paul Bert's theory—(Keays):—The blood of a man or of an animal, when in compressed air, takes into solution an increased quantity of oxygen and nitrogen from the air, the quantity of the gases absorbed being in direct proportion to the increase of pressure. The gases taken up by the blood are gradually distributed to the fluids of the various tissues. With rapid decompression the nitrogen gas bubbles off in the blood. These bubbles act as emboli, block up the capillaries in one or another part of the body and, by cutting off the blood supply or by direct mechanical violence, cause the symptoms of compressed air illness. Symptoms of illness may be prevented by making decompression slow enough to allow the absorbed nitrogen to escape from the lungs. Further experimentation by von Schiotter, Hill and his associates, and others, have confirmed this theory. Post-mortem findings, moreover, in many fatal cases of compressed air illness, both in men and in animals, give conclusive proof that this theory is correct.

(1) The higher the pressure, the greater the chances of illness; (2) the longer the time of pressure, the greater the chance of illness; (3) the greater the decompression, the greater the chance of illness.

Fatal cases are rare below 30 years. The symptoms may be hastened by exercise. Decompression should be slow.

Boys and those past 40 years of age are not safe in the caisson. Fat people and those with organic disease, especially of the heart, should keep out. Alcoholism, fatigue, green men, and occasional idiosyncrasy are all important predisposing causes.

Morbid Anatomy and Pathology: After a long illness, myelitis, hemorrhage, pneumonia, etc., may be found at autopsy.

After a short illness, free gas is found in the blood.

Symptoms: The symptoms come on a half hour or more after the workman has returned to normal atmospheric pressure. There is dizziness, sometimes headache, nausea and vomiting. The patient feels faint. There are various pains in the arms, legs and abdomen. The legs, less often the arms, may become paralyzed. The bladder is affected with retention of urine. Bad cases may rapidly become comatose and die.

Diagnosis: Is self evident.

Prognosis: Severe cases may die promptly, mild ones may recover entirely after a varying length of time.

Treatment: Prophylactic: Those about to enter a caisson should be thoroughly examined. If they have organic disease they should be kept out. New men should, after examination, have a preliminary short test followed by re-examination.

Decompression should be slow, not more than three pounds in two minutes. The men should move about during decompression.

Four hours a day should be the limit of time in the caisson, but in shifts of two hours each.

If symptoms appear after coming out the patient should immediately go under pressure again, the second time decompression should be more gradual.

Heat, vibration, massage, electricity, may all be tried.

Fluid extract of *ergot* in dram doses every hour is said to give relief.

SPINAL IRRITATION.

Synonym: Spinal Neurasthenia.

Definition: A form of functional disturbance of the spinal cord.

Etiology: Spinal irritation may be caused by emotional excitement; physical or mental overwork; alcoholic or sexual excesses; hypochondriasis.

Symptoms: The symptoms are of gradual onset. There is fatigue in walking. There is pain in the back and loins. There may be formication, numbness and paresthesia. The bladder may be irritable. There may be sexual weakness, even impotency and pollutions. Tender spots may be found over the vertebræ.

Sometimes there are flushing and sweating alternating with coldness. The patient is hypochondriacal.

Diagnosis: The diagnosis is made from the evident spinal symptoms without physical cause, together with the mental state of the patient.

Prognosis: Good.

Treatment: Bad habits must be corrected. Cold baths and spinal douches are of service.

Nux vomica is probably the best remedy, long continued.

MYELITIS.

Definition: Inflammation of the spinal cord. It may be transverse or diffuse, acute or chronic.

Etiology: Traumatism. Exposure. Toxic diseases. Compression by new growths. It occurs mostly in men from 25 to 45.

Morbid Anatomy and Pathology: The cord may look normal, but be soft. Capillary hemorrhages may be found on section.

Leucocytosis and micro-organisms may be found microscopically.

Various parts of the cord may be affected.

Symptoms: Myelitis following the acute infections may be of sudden onset and develop in a few days. There may be motor disturbances, sensory disturbances, reflex disturbances, or trophic disturbances. The disease may limit itself to certain parts of the cord. That being so the symptoms of a given case will, of course, vary with the part affected.

In cervical myelitis the arms and legs show marked disturbances in motility. There may be, at first, a weakness of the legs, the patient has difficulty in walking. There may be twitchings or even spasms of the leg muscles. There is sometimes ataxia. Finally, there may be complete paralysis. The same set of symptoms will follow in the arms.

There may be changes in the pupils.

With the motor disturbances, later in the course of the disease, there are disturbances of sensibility. Rarely there is hyperesthesia below the affected part of the cord. More often

there is anesthesia to a greater or lesser degree. First there is an irritability, formication, itching and prickling of the skin. The skin is less sensitive than usual, especially to heat and cold. Often there is delayed sensibility to pain.

If the myelitis is high up in the cord the reflexes may at first be exaggerated. In extensive disease they are finally lost.

Myelitis occurring at any level may affect the bladder. There is first delayed micturition. Then an irritability causing frequent urination. Finally, paralysis, with involuntary micturition. This last is of importance because cystitis so frequently follows.

The bowel is affected in a similar manner. Constipation is usual at first, finally involuntary stools.

The sexual function is nearly always lost.

Trophic disturbances show themselves in dryness and roughness of the skin, and sometimes in true muscular atrophy. Bed sores are common.

In dorsal myelitis the upper extremities are free. The reflexes are increased below.

In lumbar myelitis reflexes are lost. Disturbed sensation is found on the backs of the thighs, the buttocks and genitals.

Complications and Sequelæ: Cystitis. Pyelitis. Bed sores. Pyemia.

Diagnosis: Multiple sclerosis is the condition most often mistaken for myelitis. Staccato speech and nystagmus will differentiate it.

Pressure as from a displaced vertebræ or traumatism must be eliminated.

The site of the lesion is best differentiated by the height of the sensory disturbances.

Prognosis: Recovery may take place, but it is very rare. Death may take place in a few days or a few weeks. In some cases the disease process stops, leaving a permanent paralysis.

General weakness, cystitis, bed sores or infection from them, respiratory failure or some intercurrent disease may kill.

Treatment: The patient should be cautioned against overexertion. He should be placed under the best possible hygienic

surroundings. A simple nourishing diet should be instituted. The digestive tract must be cared for.

Ordinary tub baths, not too warm, seem to help some cases. Electricity may be tried.

Aconite is of service if tingling sensations in the nerves are pronounced.

Arsenicum album, twitchings, numbness, exhaustion from slightest exertion.

Hypericum, myelitis due to traumatism.

Mercurius, legs paralyzed.

Phosphorus, hands and feet numb, tremble from every exertion.

Picric acid if sexual disturbance is marked.

Silica, paralysis of limbs, parts feel cold.

LOCOMOTOR ATAXIA.

(Greek, αταξια, want of order.)

Synonyms: Tabes Dorsalis. (Latin, Tabes, to waste away; Dorsalis, of back.) Posterior Spinal Sclerosis.

(Greek, σκληρος, hard.)

Definition: A disease of the spinal cord, characterized by changes in its posterior columns. There is a consequent loss of coördination in the voluntary muscles, which gives it its name.

Historical Note: Early writers described symptoms that can now be recognized as belonging to this disease. It was first described, though imperfectly, as a disease entity by W. Horn in 1827. The first good description was given by Romberg in 1846. Duchenne called it locomotor ataxia in 1858. Many writers have given good descriptions since then. Fournier and Erb showed its relation to syphilis.

Etiology: About ninety per cent. of the cases give a history of syphilis. Some authorities claim that all the cases occur in syphilitics, although all syphilitics do not, of course, develop locomotor ataxia. It is more common in men and develops from ten to fifteen years after syphilitic infection. One of my patients contracted syphilis in 1858 and developed tabes in 1880.

Excesses are sometimes thought to be exciting causes. Likewise exposure to cold. Strümpell says he has never seen a case where syphilis could be absolutely excluded. He thinks it due to a toxine. Present day tests with the Wasserman reaction frequently show active syphilis to be present.

Morbid Anatomy and Pathology: The pia mater of the cord is thickened. Section reveals shrinking of the posterior columns. They are grayish in color and harder than common. The posterior nerve roots are shrunken. These changes usually take place first in the lumbar region and gradually extend up as the disease progresses. The posterior cornua are also occasionally affected and the degeneration may extend to a considerable part of the gray matter and to the lateral columns. The changes are parenchymatous and interstitial. The nerve filaments are destroyed and replaced by new connective tissue. Atrophy of the optic nerve is quite often associated with the atrophy of the posterior columns.

Within the last few years the cerebro-spinal fluid in cases of locomotor ataxia has been studied. There is found, usually, an excess of lymphocytes. It often reacts to the Wassermann test. It contains a form of globulin.

Symptoms: The characteristic symptoms of locomotor ataxia are loss of knee jerks, incoordination of the legs, the Argyll-Robertson pupil, and gastric crises. Any case presenting all of these symptoms is locomotor ataxia. The disease is of slow development and all of these symptoms do not become marked for several years.

The symptoms that first annoy the patient are neuralgic or "rheumatic" pains in various parts of the body, but mostly in the legs. These may last for several years, coming and going without apparent reason. One patient of mine reported that he had had rheumatic pains for nearly twenty years.

Next the patient notices disturbances of sensation. One patient said he felt as though he had billiard balls in his shoes. This sensation made his gait somewhat unsteady, especially in the dark. He was accustomed to spending an occasional evening at his club, and in walking to the nearest street car he found that his gait was unsteady, as though he had been drinking.

There is also decreased sexual desire and decreased sexual capacity at this time. Urination may be slow and unsatisfactory. It may become involuntary.

As the case progresses the difficulty in walking in the dark becomes more pronounced. Indeed, that may be the first symptom specially noticed. The patient finds if he has to get up in the night that he cannot get about the room no matter how familiar it is. Careful examination will show Westphal's sign —absence of one or both knee jerks. It is also apt to show Romberg's sign, inability to stand steady with the eyes closed. Ankle clonus is absent. The triceps reflex is lost. The Argyll-Robertson pupil is also present; that is, the pupil reacts to accommodation, but not to light.

With the loss of coördination there is no loss of muscular power. It is simply an inability to direct the muscles in the desired movements. This inability progresses until the patient becomes perfectly helpless.

Hypotonia—loss of muscular tonus, is another symptom. There is loss of resistance to passive motion.

Besides the disturbance of sensation already noted the patient may have paresthesia. Very characteristic symptoms are the lightning or lancinating pains. These occur usually in paroxysms. Girdle pains, or gastric crises, are common. Severe pain in the rectum, a feeling of constriction, with loss of power to expel feces.

There are other disturbances of sensation. For instance, delayed sensation. Sometimes sensation of pain is lost. There may be anesthesia, sometimes areas of hyperesthesia.

Optic atrophy may occur. Wilson says if this occurs during the ataxic stage it seems to halt the incoördination.

Trophic disturbances sometimes occur. This may take the form of perforating ulcer of the foot. There are sometimes arthropathys—especially of the knee. The joints may become deformed. Fractures occur easily and without pain.

There is a general muscular emaciation.

Hysteria may be associated with tabes.

Complications and Sequelæ: May have infection of bladder, a

cystitis from incontinence, or from inability to urinate voluntarily. Paralysis is a frequent sequel.

Diagnosis: A typical case is easily diagnosed. Hysteria may simulate it for a time and sometimes is associated with it. Multiple neuritis may have loss of power and wasting, but Argyll-Robertson pupil is wanting. Knee jerks are not lost.

Tumors of cerebellum, the reflexes not abolished. Vertigo and vomiting may occur.

Prognosis: Lasts for years. Usually the patient dies of some intercurrent disease.

One case was under observation eleven years, and then died of pneumonia.

Treatment: The ataxic patient will need to lead a quiet life, physically. Mentally he may be quite active. The patient referred to above lived eleven years after the diagnosis was made, and during all that time was a lawyer in active practice.

Alcohol and tobacco should be forbidden. Patients need plenty of good food and fresh air.

Some patients can be benefited very much by being taught systematic exercises, which, after a time, seem to develop new conduction tracts so that they regain considerable control over the affected limbs.

Electricity in various forms seems to help some patients, especially the static spark, and faradic brush.

It may be necessary to wash out the bladder in cases with cystitis.

Iodide of copper, suggested by the late T. F. Allen, benefited some of my cases.

Aluminum was recommended by Bœnninghausen.

Angustura vera was recommended by J. T. O'Connor.

Argentum metallicum has some of the ataxic symptoms and the gastric crises.

Belladonna, nux vomica and *strychnine* may also be indicated.

A combination tablet of *nux vomica* and *carbo vegetabilis* controls the gastric symptoms of one patient, and has for years.

FRIEDREICH'S ATAXIA.

Synonyms: Hereditary Ataxia. Family Ataxia. Congenital Ataxia.

Definition: A form of ataxia occurring in certain families. A chronic degenerative disease, the result of defective development of the spinal cord.

Historical Note: The condition was first described by Friedreich in 1861.

Etiology: There is a family tendency to degeneration among brothers and sisters. It is rare between parents and children. It is possible that syphilis or intemperance in the parents may predispose to it in the children. Near relatives as well as brothers and sisters may present cases of the disease. Half the patients are under eleven years of age. It is rare for it to develop after puberty. The sexes are equally affected.

Morbid Anatomy and Pathology: The spinal cord is thin and small. It shows arrest of development of certain parts. There is congenital degeneration of the motor neurons. There is thickening of the pia mater, especially of the posterior side. There are sclerotic changes of the posterior and sometimes of the lateral columns. In advanced cases there is sclerosis of the medulla.

Symptoms: The leading symptom is ataxia. As a rule, the legs are affected first, later the upper extremities. The disease develops gradually, showing first an unsteadiness or uncertainty of motion, later a decided ataxia, and in some cases a final paralysis. The loss of coördination affects the trunk as well as the extremities.

Nystagmus is a common symptom. There is no change in the pupil reflex. The patella reflex is usually absent. In rare cases it is exaggerated. Romberg's symptom is present. Speech is slurring owing to incoördination of the muscles involved. Deglutition is not interfered with.

There is rarely any anesthesia, sensation remains intact. There are no gastric crises, no bladder symptoms. Pain is rare. There is what Strümpell calls "muscular unrest"—a tremor al-

most choreic in some cases. Occasionally the patient has cramps. In some cases contractures result, and there may be curvature of the spine, more often equinovarus.

The intellect remains clear.

Complications and Sequelæ: After running many years paralysis may result. Tuberculosis is said to be a common complication.

Diagnosis: The early age, usually before puberty, rarely after, at which Friedreich's ataxia develops, together with normal pupil reflexes, serves to distinguish it from tabes dorsalis.

Prognosis: The disease is very slow, but progressive, lasting on the average over twenty years. Dana reports one case as lasting forty-six years. The patient may die of exhaustion, more frequently from some intercurrent disease.

Treatment: The disease is incurable. The patient should be placed under good hygienic conditions.

Baryta carbonica, causticum, and the remedies mentioned under tabes may be indicated.

MUSCULAR ATROPHIES.

Definition: A slow wasting of the voluntary muscles, due to various lesions in the motor tracts.

Historical Note: Charcot was the first to describe amyotrophic lateral sclerosis, Duchenne and Aran the spinal progressive muscular atrophy.

Etiology: Not known. Probably congenital weakness. May occur in different members of the same family.

Morbid Anatomy and Pathology: There is degeneration of the entire motor tract. The muscles are shrunken and pale.

Symptoms: Atrophy usually begins in the thumb and hand muscles. Thence it travels up the arms to the shoulders. The legs are affected next. Finally the face and tongue may be affected—bulbar symptoms. The atrophy of the muscles may be slow or rapid. There is spasticity—the hand becomes "ape-like." There is a spastic gait. When the face and tongue are affected there is difficulty in speech and swallowing. The face becomes expressionless.

Fibrillary contractions may be seen. Reaction of degeneration is present.

The affected parts are cold and lifeless.

The reflexes may be increased or decreased very gradually. There is no sensory disturbance.

Complications and Sequelæ: Paralysis.

Diagnosis: Usually clear. There are no sensory or trophic changes. The condition must be distinguished from other diseases with atrophy by the history.

Prognosis: Bad. It may last for years.

Treatment: Hygienic. Electricity. *Strychnine* 1/100 t.i.d. by injection.

Remedies, *cuprum, phosphorus, plumbum, zinc, arsenic, argentum nitricum, nux vomica,* etc.

MUSCULAR DYSTROPHIES AND MYOPATHIES.

Definition: Muscular atrophy, due to disease of the muscles themselves, instead of the central nervous system.

Historical Note: Greisinger, Erb and Duchenne were the first to clearly describe these conditions.

Etiology: Not known. Different members of the same family are often affected.

Morbid Anatomy and Pathology: Sometimes there is first a hypertrophy of the muscle—then atrophy and waxy degeneration follow. The nerves are intact.

Symptoms: This group of atrophies occurs in early life. The condition is apt to appear in several members of the same family, although sporadic cases occur.

There is a pseudo-hypertrophy that appears to be a forerunner in some cases. This begins in the legs. The single muscles are enlarged, but they are weak. Walking becomes difficult and patients are unable to walk upstairs. Finally paralysis may result, or more commonly atrophy appears.

In those attacked very early, infantile myopathy, the atrophy usually begins in the facial muscles. The eyes do not close properly because of atrophy of the palpebral muscles. The patient cannot whistle. Later the atrophy extends to the muscles

of the lower extremities and to the muscles of the arms. The patient cannot walk.

In juvenile myopathy, which appears about the time of puberty, the muscles of the shoulder girdle, the trapezius, latissimus dorsei, and the pectorals, are affected first. Then the arm muscles. Later the pelvic girdle. The deltoid and the muscles of the forearm and lower leg escape. The condition may later attack the face. Bulbar symptoms do not appear.

Fibrillation does not occur. Reaction of degeneration does not result.

Diagnosis: Muscular myopathy appears very early in life, there is absence of fibrillary contraction. There is absence of the reaction of degeneration. The muscles of the face, shoulder girdle, upper arm, pelvic girdle, and thigh are the ones affected. Bulbar symptoms are not present.

Muscular atrophies due to disease of the nervous system may appear at any time. There is extensive fibrillary motion. There is reaction of degeneration. The muscles of the thumb and hand and of the foot and leg are first affected. As the disease progresses it extends towards the trunk. Bulbar symptoms appear after a time.

Prognosis: The prognosis is bad. The disease is of slow development, but is constantly progressive. Patients may die of some intercurrent disease.

Treatment: Systematic exercises, long continued, may do some good.

Homœopathic remedies suggested are *arsenicum album, cuprum, phosphorus, plumbum, sulphur, zinc.*

SPASTIC PARAPLEGIA.

Synonyms: Primary Lateral Sclerosis. Spastic Spinal Paralysis.

Definition: Chronic progressive spinal degeneration with stiffness of the legs, but without sensory disturbance. Children and adults are equally liable.

Historical Note: Erb and Charcot were the first to differentiate it.

Etiology: Not fully understood.

Morbid Anatomy and Pathology: Degeneration of the motor fibres of the pyramidal tracts.

Symptoms: The characteristic symptoms are, motor paralysis, hypertonicity and rigidity, and increase of tendon reflexes.

The legs are affected first. The arms may be later. Rarely the face is implicated. There is first a muscular weakness. This is followed by rigidity. The tendon reflexes are greatly exaggerated. When once a reflex is started the limb keeps jerking till forcibly stopped. When at rest there is a rigidity of the muscles. This may be overcome by slow handling. Attempts at walking produce the peculiar spastic gait, a shuffling. Permanent contractions of the affected muscles may finally occur.

There is a family form of spastic paraplegia where several members of the same family may be attacked.

Complications and Sequelæ: Paralysis may result.

Diagnosis: We must exclude multiple sclerosis, myelitis and pressure on the cord.

Prognosis: This is unfavorable.

Treatment: Massage may help some.

Cicuta, lathyrus, zinc, cuprum may be tried.

ATAXIC PARAPLEGIA.

Synonym: Posterior Lateral Sclerosis.

Definition: A degeneration of the posterior and lateral tracts of the cord, with sometimes the anterior tract slightly affected. This causes paralysis with symptoms of spasticity and ataxia.

Historical Note: Gowers was the first to describe this condition.

Etiology: Common to men in middle life. The cause may be unknown or it may be secondary to myelitis or to intoxications.

Morbid Anatomy and Pathology: There is a degeneration of the posterior and lateral tracts of the cord. Occasionally the anterior tract is implicated.

Symptoms: The symptoms are those of spastic paraplegia plus ataxia. There is an unsteadiness in the gait. Romberg's

symptom is present. There may be pains in the legs. The reflexes are exaggerated, nystagmus is seen.

There may be areas of anesthesia and paresthesia. The sexual power is lost. There may be cystitis.

The mind remains normal.

Diagnosis: This is made from the combination of spastic and sensory symptoms.

Prognosis: The disease is slowly progressive with long quiescent periods.

Treatment: Rest is of most importance.

Besides the remedies suggested under spastic paraplegia, there is one other, mentioned by Wilson, *ustilago.*

CHRONIC ANTERIOR POLIOMYELITIS.

Synonym: Chronic Atrophic Spinal Paralysis.

Definition: An atrophy of a late and rare form, originating in the degeneration of the cells of the anterior horns.

Historical Note: Oppenheim and others have written of it.

Etiology: Unknown. It occurs late in life.

Morbid Anatomy and Pathology: Disease of the anterior horns of the cord.

Symptoms: There is first a paralysis of the legs, later of the arms. This begins as a weakness. There is no pain, no sensory disturbance. In the course of a few days or weeks the paralysis develops. There is fibrillation and atrophy of the muscles. In rare cases the disease extends to the muscles of the lips, tongue and pharynx. Reaction of degeneration is present. The reflexes are diminished or lost. The sweat is lessened. The bladder and rectum are not involved. After a time a few cases get better.

Complications and Sequelæ: There may be involvement of the respiratory musoles. Inhalation pneumonia may occur.

Diagnosis: Neuritis is of sudden onset and pain is a marked symptom. There is no pain in this form of poliomyelitis. In progressive muscular atrophy the atrophy and weakness progress together. In chronic poliomyelitis the paralysis comes first, atrophy afterwards.

Prognosis: Usually bad.

Treatment: Must be purely symptomatic.

ACUTE ASCENDING SPINAL PARALYSIS.

Synonym: Landry's Paralysis.

Definition: A paralysis beginning with the lower extremities, then the upper, then other muscles. Sensibility remains normal.

Historical Note: First described by Landry in 1859.

Etiology: Unknown. Young and strong men are most often attacked.

Morbid Anatomy and Pathology: None known unless a very acute multiple neuritis.

Symptoms: There is general malaise, fever up to 104° F., and pain in the back and legs. After a few days or weeks paralysis suddenly appears, first in one leg, then in the other, causing paraplegia. Later this extends to the arms, then the trunk. There may be bulbar symptoms. There is no passive resistance to motion.

Sensibility remains intact, the reflexes are lost. The bladder and rectum are not affected.

Diagnosis: The disease must be differentiated from neuritis.

Prognosis: Always doubtful at best. Death may occur in a few days or weeks. A few cases recover.

Treatment: Baths and electricity may be tried.

Secale is sometimes of benefit.

NEW GROWTHS OF THE CORD.

New growths may appear in the cord or in the meninges.

Etiology: The cause may be unknown. Traumatism seems to be the exciting cause in some cases.

Morbid Anatomy and Pathology: *Varieties:* Glioma starts in the neuroglia of the posterior half of the cord. It may be very extensive. There may be hemorrhages. There may be tuberculosis or syphilis of the cord. Sarcoma may attack the dura. Cancer of the vertebræ may extend inwards.

Symptoms: Depend on the part of cord affected. May cause some symptoms of compression.

When on the dura meningeal symptoms are present.

Diagnosis: Unilateral symptoms appear first. Changeable symptoms are due to vascular changes.

Prognosis: Depends entirely on the cause.

Treatment: Surgical.

SYRINGOMYELIA AND HYDROMYELUS.

(Greek, συριγξ, tube; ὑδωρ, water; μυελος, marrow.)

Definition: Syringomyelia. Disease caused by an abnormal dilatation of the central canal of the spinal cord.

Hydromyelus. Disease caused by an effusion into the central canal.

Etiology: Congenital defect. Probably started by injury.

It occurs more often in men.

Morbid Anatomy and Pathology: There is probably a congenital condition. There is gliosis, formation of fibrous tissue, which breaks down leaving cavities. It affects the anterior or posterior horns. It most often occurs in the spinal region of the cord and may extend up or down.

Symptoms: The disease is of gradual onset. It usually begins in one arm. There is motor weakness and muscular atrophy of the smaller muscles first. Fibrillation occurs. Reaction of degeneration finally results. The reflexes may be lost or exaggerated. There is disturbance of sensation, first for pain and temperature, later for pressure and touch. On account of the loss of sensibility trophic changes may occur, and injuries may happen unknown to the patient. There is thickening and shortening of the fingers. The disease of the cord may extend downward causing symptoms in the lower extremities similar to those in the upper. It may extend upward causing bulbar symptoms.

Complications and Sequelæ: Kyphosis may result.

Diagnosis: The loss of sensibility distinguishes syringomyelia from muscular atrophy. Leprosy may present similar nerve symptoms, but the skin will eventually differentiate it.

Prognosis: This is always bad. Death may occur from some other intercurrent condition.

Treatment: There is no known treatment of any value. Careful homœopathic prescribing may do some good.

UNILATERAL LESIONS OF THE CORD.

Synonym: Brown-Sequard's Spinal Paralysis.

Definition: The group of symptoms caused by a lesion cutting off a lateral half of the cord.

Historical Note: The condition was first described by Brown-Sequard.

Etiology: Traumatism may cut off half of the cord; caries of the vertebræ may do it; tumor or syphilis may be unilateral and cut off half.

Morbid Anatomy and Pathology: Depends on the cause.

Symptoms: Because of the crossing of the sensory fibres the symptoms of a solution of continuity of one-half the cord are somewhat peculiar and characteristic. There are motor symptoms on the affected side and sensory symptoms on the opposite side of the body below the lesion.

There is usually paralysis, more rarely ataxia, on the side of the lesion. The ankle and knee reflexes are exaggerated. Some cases present points of hyperesthesia. There may be local rise of temperature.

On the side opposite the lesion there is cutaneous anesthesia to pain and temperature. There may be no disturbance of touch sensations.

Diagnosis: The peculiar combination of symptoms is characteristic of unilateral lesion of the cord. The exact cause may not be clear.

Prognosis: This depends entirely on the cause.

Treatment: This also depends on the cause.

PROGRESSIVE BULBAR PARALYSIS.

Synonym: Glosso-labio-laryngeal Palsy.

Definition: A slow developing paralysis of the muscles of speech, expression, phonation, deglutition and finally of respiration.

Historical Note: The symptoms were first described by Duchenne in 1860. The condition was named by Wachsmuth.

Charcot, in France, and Leyden, in Germany, discovered the pathological lesion in 1870.

Etiology: It occurs most often between the ages of 30 and 60 in neuropathic families. Probably a congenitally defective nervous system is the basis.

Morbid Anatomy and Pathology: The disease is located in the nuclei of the medulla. Later the degeneration extends to the nerve fibres of the cells affected. There is atrophy of the muscles supplied by the affected nerve centres.

Symptoms: The disease is of slow development. Defects in speech occur, first in sounds in which the tongue is used—d, l, n, r, s, t; next in sounds where the lips are called into play—b, p, v, w, y. We have alalia and anarthria.

The pharyngeal muscles become involved. Later the tongue begins to atrophy. There is fibrillation, or trembling of the organ. Loss of control of the tongue and pharynx makes it difficult to swallow. It also makes it difficult to dislodge particles of food and to push them back into the pharynx. Mastication is feeble and unsatisfactory. Food may regurgitate through the nose.

The lips become stiff, then atrophy. It becomes impossible to close them. The saliva constantly dribbles. The lower part of the face loses its expression.

The larynx becomes paralyzed. The patient becomes hoarse, then loses his voice. There is choking and dyspnea because food lodges in the trachea.

There is probably reaction of degeneration.

The knee jerk may be increased, local reflexes absent.

Salivation is a constant symptom late in the disease. Finally tachycardia may result from paralysis of the vagus.

Sensibility remains unimpaired. The mentality remains clear to the end.

Complications and Sequelæ: The progress of the disease is very slow, three to five years. Death usually occurs from inanition or from respiratory or cardiac paralysis.

Diagnosis: This is usually easy on account of the characteristic history and expression. Other diseases of the medulla may produce similar symptoms.

Prognosis: This is bad.

Treatment: Massage and electricity may do something for the patient.

Feeding by the stomach tube or by nutrient enemata may become necessary.

Homœopathic remedies that may be of service are: *Anacardium, belladonna, mercurius, nux vomica, nux moschata, plumbum, hydrophobinum.*

MYASTHENIA GRAVIS.

Synonym: Asthenic Bulbar Paralysis.

Definition: Myasthenia with predilection for the muscles innervated by the bulb; results in general paralysis.

Historical Note: Erb and Oppenheim first described the condition.

Etiology: Under 30, as a rule. May be caused by toxines.

Morbid Anatomy and Pathology: Not known.

Symptoms: There is undue rapid fatigue of the muscles. Begins as a ptosis, then affects the facial muscles, then the extremities, finally the chest. Movements of the affected parts are strong at first, but fatigue rapidly ensues. For example, the patient can talk a little while, then fatigue sets in and the ability is lost. Or the patient may walk up one flight of stairs unassisted, a second with help, and be unable to go up a third flight at all. The condition appears to be functional.

There may be remissions, the condition appearing and disappearing at longer or shorter intervals.

Diagnosis: The symptoms are characteristic.

Prognosis: This is uncertain.

Treatment: Rest, of course, is indicated. Electricity may be of help.

Suggested remedies are: *Nux vomica, phosphorus, stannum.*

DISEASES OF THE MEDULLA.

We may have: 1. Hemorrhage. 2. Embolism. 3. Compression. 4. Acute inflammation.

The above various conditions produce similar symptoms.

Etiology: Disease of the medulla or its blood vessels may be at fault. Exertion may act as an exciting cause.

Morbid Anatomy and Pathology: Varies with the cause.

Symptoms: The symptoms may be of rapid or of slow development, depending on the cause. In hemorrhage or embolism they are rapid. They vary somewhat according to the exact location of the lesion.

Bulbar symptoms may be the most pronounced, differing from progressive bulbar paralysis only in the rapidity of development. In other cases there may be a crossed paralysis of the face on one side and the extremities on the other.

Prognosis: Is bad. Death may result in a few hours or days from respiratory or cardiac paralysis.

Treatment: Rest is imperative.

After a time, if the patient does not die and there are no additional symptoms, electricity and massage may be tried.

If hemorrhage is the cause the same remedies are applicable here as in cerebral hemorrhage.

MENIERE'S DISEASE.

Synonyms: Auditory Vertigo. Disease of the Vestibular Nerve.

Definition: A disease characterized by attacks of vertigo and tinnitus aurium.

Historical Note: This disease was first described by Ménière in 1861.

Etiology: The trouble is caused by disease of the internal ear—the labyrinth, semicircular canals, and the vestibular nerve. Any disease or accident that affects the internal ear may be the starting point of Ménière's syndrome.

Morbid Anatomy and Pathology: Not clearly understood.

Symptoms: The attacks occur in paroxysms. There is a loud tinnitus aurium, usually in one ear. This is followed by severe vertigo in which either the patient feels himself reeling or going round, or in which the surrounding objects appear to be going round. Sometimes he falls, but usually he has time to grasp a support. Associated with or immediately following the

vertigo the patient feels sick, is pale and clammy, and vomits. Nystagmus frequently occurs. Consciousness is usually maintained.

Examination usually shows disease of the ear on one or both sides. Partial deafness is usual; when totally deaf the attacks cease.

Diagnosis: The disease must be differentiated from other forms of vertigo.

Prognosis: Doubtful. Some cases get well, some become bedridden.

Treatment: Sometimes proper glasses will cure. Charcot recommends quinine.

Carboneum sulphuratum, chenopodium anthelminticum, natrum salicylicum (*salicylate of soda*), and *salicylic acid* are mentioned by Boericke.

CHOREA.

Synonyms: St. Vitus' Dance. Sydenham's Chorea. Chorea Minor.

Definition: A nervous disorder, characterized by involuntary movements of the voluntary muscles, and frequently complicated by acute endocarditis.

Historical Note: Chorea means dance. In the middle ages the dancing mania was supposed to be cured by a pilgrimage to the shrine of St. Vitus. Sydenham described chorea in 1686.

Etiology: Chorea is found most often in childhood, from the 5th to the 15th year. Girls are more often affected than boys. It seems to run in certain families. Nervous, emotional children seem to be more prone to it. Sometimes fright, sudden grief, or some other profound emotional disturbance will bring it on. Imitation sometimes causes it. I had one such case.

In a large percentage of cases chorea is associated in some way with acute articular rheumatism or endocarditis. In fact, chorea has been classified as a rheumatic condition.

Chorea sometimes develops during pregnancy, especially in young primipara. It is very fatal then.

Morbid Anatomy and Pathology: There are no pathological conditions common to every case. The idea that it is an in-

fectious disease has been advanced on theoretical grounds only

Endocarditis is the most common lesion found.

Some have thought it due to emboli.

It is usually considered a functional nervous disease.

Symptoms: Prodromal symptoms rarely occur. When they do they are very indefinite and consist mostly of a depression of spirits and an irritability of temper.

Usually the involuntary motions are the first things noted. These come on gradually. There is a twitching here and there. There is lack of co-ordination. The patient drops things from the hands or stumbles in his walk. The arms and legs are thrown about or hang limp.

The muscles of the face may be involved. The muscles of articulation may be involved and speech stammering, jerky or impossible.

One side of the body may be worse than the other. One of my cases let her right arm hang limp, and dragged her right foot so as to wear off the toe of her shoe in a very few days. She was naturally right-handed, but for several weeks had no control over that hand at all.

Cases vary in severity, may be slight, or very severe. One case, a man at the Metropolitan Hospital, had extremely severe and erratic motions.

There may be some psychic disturbance. The patient may be irritable, capricious, weepy or unreliable. She is apt to be worse, more erratic and uncertain in her motions, when watched. The movements cease during sleep.

In rare cases there are mental symptoms, hallucinations and so on. (Chorea insaniens.)

Complications and Sequelæ: Endocarditis must always be watched out for. Arthritis may develop.

Diagnosis: This is usually easy. There may be choreiform motions in some other conditions. Hysteria may simulate it.

Prognosis: Is usually good. The patient may have one or more relapses. A few cases of chorea insaniens die.

Treatment: Mental and physical quiet must be insisted on. The child should be taken out of school. It should be shielded from ridicule.

My cases have cleared up under *zinc, calcarea carbonica* and *causticum*. Other remedies may be indicated.

HUNTINGTON'S CHOREA.

This is a type of the disease that develops usually after thirty years of age. It seems to be a family disease. The voluntary motions are very erratic and severe. The entire body seems to partake of the choreic movements. I have seen but one case, a man, at the Metropolitan Hospital.

PARALYSIS AGITANS.

Synonyms: Shaking Palsy. Parkinson's Disease.

Definition: A chronic nervous disease, characterized by tremor, muscular weakness and rigidity.

Historical Note: First described by Parkinson in 1817.

Etiology: Unknown. Occurs slightly oftener in men than in women. Appears at middle age or later. Catching cold, emotions, etc., seem sometimes to act as exciting causes.

Morbid Anatomy and Pathology: This is not known.

Symptoms: The tremor is the characteristic symptom. Strümpell says the trouble usually begins in the right hand, then in the arm and leg of the same side. Sometimes the head is affected. The tremor is quite rapid, according to Osler, about five times per second. The tremor is worse when the emotions are aroused or when the patient is conscious of being watched. Voluntary motion may control the tremor somewhat. A dentist patient can control his hand when resting the arm on a support.

There may be a certain rigidity of the muscles. Facial tremor may make an expressionless countenance. There may be infrequent winking. In advanced cases the head is flexed, the body bent forward, the arms are close to the body and flexed at the elbow. The fingers are flexed.

Movement is slow and deliberate. The patient cannot get up without help.

Leaning forward causes the centre of gravity to be forward; if the patient walks he has to keep going, propulsion; if pushed back, he keeps going back, retropulsion.

The voice may be affected; it becomes a monotone.

Complications and Sequelæ: None.

Diagnosis: On sight.

Prognosis: Incurable, but it does not kill.

Treatment: None of benefit. Hygienic living.

Mercurius and *hyoscyamus* have been recommended.

ATHETOSIS.

(Greek, *αθητος*, unfixed.)

Definition: A disease characterized by continual change of position of the fingers and toes, mostly an inability to keep them still.

Historical Note: First described by William A. Hammond in 1871.

Etiology: Unknown. Sometimes follows epilepsy or hemiplegia. May occur at any age.

Symptoms: The symptoms consist entirely of the continued motions of the parts affected. These are usually the fingers or toes, sometimes the face or head.

Diagnosis: Self-evident.

Prognosis: Goes on indefinitely without change.

Treatment: General hygienic. Remedies recommended in chorea and in paralysis agitans may be tried.

CONGENITAL MYOTONIA.

Synonym: Thomson's disease.

Definition: It affects the voluntary muscles. There is increase in volume and decrease in power, and tonic couvulsions of voluntary muscles.

Historical Note: First described by Thomson in 1876, as tonic convulsions of the voluntary muscles.

Etiology: Childhood. Family disease.

Morbid Anatomy and Pathology: Interstitial muscular tissue hypertrophied and degenerated.

Symptoms: The affected muscle develops a tonic spasm when

put into use. This may last up to thirty seconds. It disappears after use. The patient may take a glass of water and not be able to put it down or drink it because of the spasm.

Diagnosis: Easy, as a rule. In tetanus there is trismus.

Prognosis: Good as for life, bad as to cure. Patient may have remissions.

Treatment: *Strychnine* in potency might help.

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