XX. THESIS ON TUBERCULOSIS

Merle Voss

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Preface.

The purpose of this paper is threefold. The first, a desire to have more knowledge and understanding of tuberculosis; the second, to try to set down the story and facts of tuberculosis in such a form as to be interesting to nurses in particular; the third, a natural interest in the present, "five year follow-up campaign."

As I read the many chapters on tuberculosis by famous physicians and research men, I realize the great ignorance of the majority of people to this disease. This statement includes more since the discovery of the tubercle bacillus, doctors have made great progress in the battle against tuberculosis. They have gained the coöperation of many organizations through education and establishing the importance of destroying tuberculosis. This is going to be a long and tedious battle against a disease that is eating into the life blood of every population.

I have found very few books in the event of reading that would not take intensive study to master the principles of tuberculosis. If this material is difficult for an individual in the medical profession to grasp, then it must be even more confusing to the lay

person with no medical background. The lay person must be educated to tuberculosis and its prevention if we can ever hope to succeed in conquering it. The second purpose then in my thesis is to collect and condense facts to be readable at least to those in the nursing profession.

The recognition of the importance of the importance of follow-up work has long been recognized but only in the last few years has anyone been able to perform this with a percentage. It will be interesting to see the results of the five year follow up campaign on tuberculosis

Chapter One

History

TUBERCULOSIS
(Chapt. 1--History)

From the time of its rudest conception to the present, advancement of the knowledge of tuberculosis has been a parallel movement. The development in its most precise sense of the conception of tuberculosis as a pathological extity is one of comparatively recent origin. Indeed, so long ago as the 4th and 5th centruies before the Christian Era this malady attracted attention.

The expression "phthisis" was primarly applicable to all disorders which brought on wasting; later its limitations were narrowed to cachectic disturbances of the respiratory system. In the works of Aristotle, Diascairedes, the phepician of Cleopatra and Celsus while the clinical picture of phthisis given by Aretoäus has remained classic from the elegance of its descriptions under the name "phthoe"--the latter recognizes consumtion in its most genuine form and to him most likely belongs the credit of having first clearly described the disease as a special pathological manifestation.

The clinical comprehension of pulmonary tuberculosis made an imperfect and vacillating progression through the labors of Galen, Caeleus, Aurelianus, Orebasius, Aelus, Alexander Trallian, Paulus Aegeneta, Maimonides and the Arabian physician Phazes. Farrestus in 1653 published his "Observationium et Curatiomune Opera Omnia" at Rouen, in which he distinguishes himself by giving a detailed recital of a variety of cases founded on extensive practical observations.

The earliest serious indications of this is found in the writings of Platirus Bennet, Bonetus and Sylvius. According to Hoffman the works of Aretaeus showed evidences of a recognition of tuberculosis

nodoseties in the lungs. The compelation of Bonetus contains the clinical histories of several cases of pulmonary phthisis with notes of the autopsy findings, describing cavities and the "sebaceous" contents of "granulations".

It was not until the close of the seventeenth century that attention was directed to the close similarity between serofulous lymph nodes and the caseous nodules in phthisical lungs. This early association of scrofula and tuberculosis, the result of the investigations of Sylvius posesses considerable historical interest in that changes in phthisical lungs were the manifestations of scrofula in assumed pulmonary lymphatic glands.

So long ago as 1646 and fully twenty-five years before the observations of Sylvius, there appeared a work by Fabricius Heldonis in which are recorded a number of dissections in consumption cases in which a mesenteric and pulmonary affection were combined in a revised edition of Bonitus. He made a post mortem examination and found "grandines" in the lung, liver, spleen, kidneys, mesenteric glands and intestines, likening these bodies to millet seed.

The notion of tuberculosis as a general disease characterized by multiple localizations in various organs is really of relatively recent date. The physicians of antiquity developed the symptomatology of phthisis. Boyle in 1810 and his wealth of cases enabled him to develop the conception of tuberculosis as a process affecting different parts of the body.

Wholly absorbed in the doctrine of chronic phlegmasia of the lungs Broussais recognized two orders; one entertained by inflammation of the

blood vessels, capillaries and the other, the resultant of that of the lymphatic vessels.

The concurrence of true tubercle with caseous pneumonia must have been so frequent that it could not have escaped observation or been considered a mere coincidence. Explanation of such an instance was readily found in the views of Dettrich and Buhl, who believed that tubercle were secondary and consecutive to the absorption of the caseous material.

The first effort to produce tuberculosis experimentally dates back in all probability to the year 1789. Kortum innoculated a boy in the region of the neck with "scrofulouspus". Fortunately the result was negative. Experiments of a similar nature were made on dogs three years later by Hebriard; also by Lepellier on guinea pigs in 1830. In no instance were the animal innoculations successful.

In the year 1843 Klenche announced that after the innoculation of rabbits with miliary tubercle and other tuberculous matter the animals became tuberculous.

The distribution of tuberculosis is in all parts of the world. There are records in India of the 13th century. As early as 600 B. C. in China records were made of the symptoms. Arabic physicians taught tuberculosis as a communicable disease and had a crude form of isolations.

Galen, a Greek, in 200 A. D. instigated sanitorium, bath-houses, sunlight, air, diet and use of milk from well cared-for animals. In the 17th century in Italy and Spain the laws prescribed a rigid quarantine. The infected room must be replastered and old plaster burned.

Villemins, 1865, announced the results of his experiments on the innoculability of tuberculosis. The far reaching significance of introduction into an organism susceptible to its impressions, this agent

reproduce and reproduce itself during the same time as the principle and determining cause.

With the investigations of Chauvean Gerluch, and Bollinger upon ingestion of tuberculosis, the inoculability of the disease was firmly established.

Klebs announced in 1877 that the innoculation of the animal with cultures from tuberculous products upon the whites of eggs produced lessons similar to those following the injection of tuberculosis tissues themselves. The announcement of the discovery of the bacillus tuberculosis was made by Kock before the Physeological Society of Berlin on the 24th of March, 1882.

In the Harz Mountains in the province of Hannover, Germany on December 11, 1843 a boy by the name of Robert Kock was born. There were thirteen children in the family of whom Robert was the third. There were eleven boys in the Kock family. Robert liked best to wonder over the Harz Mountains with a natural history book, a bottle of alcohol, pins and boxes in search of plants, insects and stones for the museum he had set up in his room. His desire to explore the secrets of nature was favored one day by the discovery of an old pocket-lens in a little chest belonging to his father. The lens was scratched, but Robert begged it from his father and set out to name new discoveries.

At first the family was so poor that there was little thought of sending the boy to a university. Just as Robert was about to graduate from high school at the head of his class, the family fortunes were improved so Kock went to university at Gottingen.

He was interested in natural science more than in mathematics or Latin and Greek. He decided to study medicine, so that he might become a ship's doctor. One of Kock's teachers was the famous scientist, Jacob Henle.

Twenty years before this time Henle had written a book about contagions. In this book he said, "Before microscopic forms can be regarded as the cause of contagion in man, they must be found constantly in the contagious material, they must be isolated from it and their strength tested." In January, 1866, Kock finished his medical course in Gottengen and passed the state examinations, which entitled him to practice medicine. He secured a post in a hospital in Hamburg.

In 1869 he settled in Rakwitz with his wife. His work here was interrupted by the outbreak of the Franco-Prussian war in 1870. Kock took part as an army surgeon.

On his twenty-eighth birthday his wife gave him a microscope. Kock hung a curtain across his consultation room and set up a little laboratory. Over in Paris, Pasteur was stirring up medical men with his experiments to prove that communicable diseases are caused by particular living organisms. As a result of Pasteur's work, Hester in Scotland was proving that a surgical wound can heal without becoming infected, if the dressings used and the hands and instruments of the operating surgeon are completely freed from living microbes.

Kock experimented with mice in obtaining confirmation of his suspicion of the anthrax bacilli being present in anthrax in sheep. He devised his famous "hanging-drop" method of study. He found the organisms in every experiment, proving his assertion.

It was Kock who hit on a method to grow pure cultures. One of the favorite culture mediums of that time was liquid gelatine. Now Kock changed the proportions of jelatine and water so the the mixture would harden when cool. From Kock's day to this, the use of solid culture media remains one of the best ways to obtain microbes in pure culture.

We now come to the year 1882, that wonderful year in which the world

was startled by the announcement that a German by the name of Robert Kock had discovered the bacillus which causes tuberculosis. At the beginning of his search he set down as his guide the laws which have become famous as fundamental in bacteriology. First, in order to prove that a certain microbe is the cause of a certain disease, we must find it present in all the cases of the disease. Second, we must completely seperate this microbe from the diseased organism and grow it outside the body in pure culture. Third, with this pure culture we must be able to give it to healthy animals by innoculating them with it. Fourth, the microbes should be obtained again from animals so innoculated and grown in pure culture outside the body.

Kock was sure that the bacilli must be in tubercles or tiny lumps which he found in the tissues of those who had died of tuberculosis. Even with the best microscope, however, he could not see bacilli in these tubercle. He tried dyeing his specimens. He used a solution of methyline blue to color the tubercle bacilli and resurin solution to color the tissue. Kock completed two hundred seventy-one experiments and in every one he saw the deadly little bacilli which each year caused one death out of every seven in Europe and America.

He devised a medium to culture the bacilli in which was the clear serum taken from the blood fluid of cattle. He innoculated well animals and symptoms of tuberculosis appeared. After they were dead, the bodies were examined and tubercle bacilli were found. The chain of evidence was complete. In 1884 a complete report on the cause of tuberculosis was given to the world.

In 1890 Kock announced that he had discovered a remedy for tuberculosis in tuberculin, a substance made of boiled glycerine extract of the tubercle bacillis. Within a few months, however, tuberculin had failed

to bring about the cures which a hopeful world expected of it. On the twenty-seventh of May, 1910, the world received the news of Robert Kock's death.

In February of the year 1873 a young American doctor had just received a blow which shattered all his dreams of success in his profession and of happiness in the new home he had made for his wife and baby. His name was Edward Livingston Trudeau. He had gone to Dr. Janeway because for some time he had felt tired and feverish. This physician discovered that the young man had tuberculosis in an active and advanced form.

Yet Trudeau was to turn defeat into victory, not only for himself but for thousands of others. He thought he had but a short time to live. He was becoming steadily worse and longed for the peace and quiet of the Adirondack Mountains where he had previously spent happy vacations. He went there early in the spring in a very weakened condition. Yet this apparently dying young man was to outlive most of the stalwart guides who welcomed him.

No one then knew the importance of rest in treating tuberculosis, but as Trudeau spent long hours in a boat fishing or lying in wait to shoot deer he kept at rest without intending to. He gained in weight and then decided to set up his medical practice in Saranac Lake. He gained steadily on this life for ten years. In 1883 he decided to extend his benefits to others. In February 1885 the first cottage was finished and two girls from New York moved in. Thus sanatorium treatment for tuberculosis began in America.

Gradually through Dr. Trudeau's enthusiasm and with the generous help of his friends, the sanatorium and laboratory grew. Before he died in 1915, at the age of sixty-seven, Trudeau saw his sanatorium and laboratory ranked

among the most famous medical institutes in the world.

Today, at Saranac Lake, the Trudeau Sanatorium has space for nearly 200 patients, and the Edward L. Trudeau Foundation has research laboratories to which students come from all parts of the world. Both the cure and prevention of tuberculosis depends on healthy living and good food, on fresh air, sunlight and particularly on rest.

In 1894, Biggs began the first administrative campaign for the control of tuberculosis. He arranged for the laboratory examinations of spectum to diagnose the disease for reporting of cases by physicians to the Board of Health and for isolation and disenfiction. Reporting was first only requested. When it was made compulsory in 1897, a storm of protest burst from the more conservative physicians. The more conservative physicians went to the capitol at Albany and tried to persuade the legislature to take away from the Board of Health the power to deal with tuberculosis. The Board of Health and the public supported Biggs. Gradually the opposition died down and our modern program for the control of tuberculosis was established on a firm foundation.

The health department has divisions of tuberculosis and child hygiene to direct concerted community health programs along each of these lines. Furthermore, alongside of, and cooperating with the health department, stand a great group of voluntary agencies, anti-tuberculosis associations, child health associations, and visiting nurse organizations which play an invaluable role in the common task.

Another interesting historical point in tuberculosis is the story of the Christmas Seal. This renouned project was started by a postal clerk in Copenhagen, Denmark in 1903. His name was Einar Holbolt. He wanted to do something for tuberculous children and accidently hit on the idea of the Christmas stamp. He aroused interest in the local citizens, then obtained

permission from the King Christian XX. The first stamps were adorned with the picture of Queen Louise.

The first sale was from December 6th to January 6th, started in 1904. The sum of 68,000 kroner was raised. In 1905 the site for a sanatorium at Kolding was purchased and so Holbolts desire to help tuberculous children came true. The building was completed in 1910. The proceeds from the sale increased from year to year and was popular alike with rich and poor.

Holbolt's method to raise money to fight tuberculosis was soon adopted by various other foreign countries; Sweeden in 1904; Norway in 1906; and more than forty other countries.

Many honors came to the "Father of the Christmas Seal". His death February 23, 1927 from a heart attack was mourned by many.

In 1904 Jacob A. Reis, imigrant to America and friend of Holbolt, brought the idea to America. Theodore Roosevelt called him "America's most useful citizen". Miss Emily P. Bessel launched America's first Christmas sale December 9, 1907. In Wilmington, Deleware was a little open air shack on the Brandywine where poor victims of tuberculosis were brought back to health. Miss Emily Bissel, secretary of the Deleware branch of American Red Cross, knew what closing the shack would mean to those poor unfortunates, thus the seal sale idea was championed. She hoped to make $300.00 but much to her surprise and gratification $3,000.00 was raised. Today the sale of seals practically supports the entire tuberculosis campaign.

In the year 1872 Dr. Carson advised treatment of tuberculosis by collapsing the lung. In 1884 Fordinini actually carried out this proceedure and performed the first pneumothoraz. In the United States Dr.

Murphy performed the first pneumothorax and in the State of Oregon Dr. Watson in 1912 was the first.

Descriptions of treatments will be discussed in later chapter

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Chapter Two

General Statistics

Chapter II General Statistics

Tuberculosis is certainly a long way from being conquered. The tubercle bacillus kills 70,000 Americans yearly and puts over 500,000 in danger of health from tuberculosis. It is curable in nearly 100% of all cases when it is in its early stages. With the present power of science that could be made available to all, there is no reason why all cases of tuberculosis should not be discovered when they are early and 100% curable.

Tuberculosis can never be wiped out so long as the bulk of its victims are discovered after the symptoms are far advanced.

Tuberculosis occurs in very uneven proportion with concentration in certain spots because of infection and close contact. In racial distribution the Negro, Indian, Mexican respectively have the highest infection rate. Tuberculosis is more prevalent in urban communities than in rural. There is no difference in prevelance in sex. In age groups, the mortality rate is high in children under three years of age. The following chart gives a clear comparison of age groups.

Japan and some of the countries of the south are suffering increase in tuberculosis. Thirty years ago the death rate in Japan was 65 and has increased to 135 per thousand population. A comparison of the mortality rate can be found in the following figures;

Japan----------135

Austria----------95-100

France----------85

Scandanavia and England--70

United States----------55

Australia and New Zealand35

The human type of tuberculosis is the most frequently found. It does not affect domesticated animals but wild animals in captivity are quite susceptible. The guinea pig is very susceptible while the rabbit is not. The bovine type of tuberculosis is prevalent in 40% of the dairy cattle in England and from ½ to 1% here in the United States. The rabbit is very susceptible to bovine tuberculosis. The avian type of tuberculosis is found in poultry.

There are three methods of invasion of the tubercle bacillus into the body. The most common one is inhalation. Ingestion is caused from the neglect of bovine tuberculosis. Inoculation has proved very interesting to science as this is very uncommon.

The most common sources of infection are from sputum discharges, food and pus from infected areas. The bacilli differ in virulence to a great extent. True too, the bacilli may not always be virulent but have a remarkable ability to grow.

We may consider the process an infection, so long as it does not develop to interfere with structure or function. If it does then it is classed as disease. There is a reaction as long as the tubercle is present, in the tuberculin test. The test only ceases to cause a reaction when the process is entirely healed. Tuberculin can only be a diagnosis of infection, not of disease. Infection develops immune bodies to some degree but is considered best to remain free from infection. Pregnancy depresses a Tuberculin reaction.

Tuberculosis can develop and spread to other parts of the body in different ways. The infection center may grow,

and spread and become a compound, irregular mass. Through the the lymphatics and blood as the capilaries are all tangled with the lymphatics and entanged in the pleura, the lymph empties into the blood. In periphera the lymph is carried to the edge of the pluera thus causing pluritic infection and inflamation. The lymphatic centre does not catch all of the bacilli letting some spill over. In the direct system, the most common, there is involvement of the walls of the bronchius which breaks through. The bacilli come up in sputum because they are carried by cilliary action to the throat and thus may be carried to other parts of the lung. They may lodge in the last part of the small intestine or large intestine from pulmonary tuberculosis.

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Chapter Three

Types

(Chapter III--Types)

Tuberculosis may infect any organ of the body. The infection may be primary tuberculosis or secondary reinfection tuberculosis.

By primary tuberculosis is meant the totality of the morbid processes following directly and uninterruptedly the first implantation of tubercle bacilli. It is obvious in many instances no sharp line of demarcation can be drawn between primary and reinfection tuberculosis. The first infection of tuberculosis is in the vast majority of cases in the United States caused by inhalation of tubercle bacilli. In the vast majority of cases the primary complex heals by fibrous encapsulation and calcification. When this occurs, two practically indelible marks are left: calcified parenchymal and regional lymph--node foci have been formed which may contain viable tubercle bacilli; and the body has become allergic, which means that its reaction to tubercle bacilli or their metabolic products has been altered, a fact that can be demonstrated by the tuberculin reaction.

Of all persons who acquire a primary infection at some time, only a small per centage ever develop clinical disease. Following the period of the primary infection and its more or less direct consequences, a latent period usually intervenes before the common type of chronic pulmonary tuberculosis develops. The latter is due to a reinfection with tubercle bacilli. The reinfecting bacilli are derived from five potential sources. The first is termed exogenous reinfection which may be inhaled from the outside. The other four are endogenous; the primary pulmonary focus may break down and discharge bacilli directly into other parts of the lungs; a caseated primary regional lymph-node focus may rupture into a bronchus, causing a tuberculous aspiration bronchopneumonia of various extent and severity; bacilli from an active primary regional

lymph-node focus may reach the lung by way of the lymphatics and the blood stream; the samll apical foci which, after having been stationary or undetected for a prolonged period of time, become progressive, ulcerate, and discharge bacilli into the bronchial tree.

Pulmonary tuberculosis, a specific proliferative and inflammative disease of the lungs caused by the bacillus tuberculosis. It may be active chronic or remain latent indefinitely.

The bacillus tuberculosis is a minute, nonmotile rod, often slightly bent and is usually recognized by special staining methods. It is very resistant to acids but readily destroyed by sunlight. There are several types but only two of importance in human pathology, human and bovine. The human bacillus which is distributed chiefly by means of milk from tuberculous cattle, is not an uncommon cause of gland, bone and joint tuberculosis in children, but it is almost a neglible factor in adults.

Infection takes place chiefly by inhalation, the bacilli being conveyed to the respiratory tract in droplets of sputum or mucus ejected in coughing, sneezing or in the dust of dried sputum; by ingestion of the bacilli which are in milk from tuberculous cows or in food contaminated with tuberculous sputum or other ejecta.

The original lesion produced by the tubercle bacillus is a minute node miliary tubercle which is at first gray and translucent but later yellow and ppaque. Microscopically, fresh tubercles consist of a collection of epithelioid cells and usually one or more giant cells surrounded by a layer of small round lymphaic cells.

It is characteristic of tubercle to fuse and form larger masses. Owing to the lack of blood vessels and the action of products of the bacilli, tubercles soon undergo degeneration and become caseous or cheesy.

In all but the most acute cases connective tissue soon forms at the margin of the caserus mass and tends to limit it and isolate it.

In unfavorable cases of tuberculosis the infection spreads beyond the connective tissue barrier, tubercles develop in new territory and the cheesy masses soften and discharge into adjacent channels. In the case of pulmonary tuberculosis the discharge usually occurs into a bronchus, leaving a cavity in the place of the cheesy mass and setting up areas of bronchopneumonia. Unfavorable cases, fibrous tissue, completely encapsulate the tuberculous lesion, the tubercle bacilli eventually die, and the necrotic area is gradually replaced by a scar or is converted into a stony(calcareous) concretion.

There are two forms, chronic and acute. Types of acute tuberculosis are acute pneumonic, bronchopneumonic and acute miliary type.

Chronic ulcerative tuberculosis begins with the development of tubercle, usually near the apex of the lung, and tends to spread downward. Various forms of lesions-cavity caseation, discrete fresh tubercles often being found at autopsy in the same lung. In many cases, however, the infection is arrested at an early stage and in this event the only lesion at death may be a small cheesy or calcareous mass surrounded by fibrous connective tissue. In some instances the term chronic fibroid tuberculosis is applicable as the progress of the disease is held in obeyance for years by an enormous overgrowth of connective tissue surrounding and isolating the

tuberculous areas.

The symptoms of acute pneumonic tuberculosis are similar to those of pneumonia, but the patient has had a cough and loss of weight for sometime before the onset. The temperature rises gradually and cough and expectoration which is frequently bloody develop. Chills and sweats occur. Signs of softening and excavation develop in the affected lung. Anemia and emaciation become pronounced, the patient gradually gets worse, and may die in a few weeks.

In chronic pulmonary tuberculosis the specific cause is the tubercle bacillus, while the predisposing causes are poor hygiene, fatigue and malnutrition. The disease begins in the lungs, the pluera or the bronchial tubes. The apices are usually the first involved because this part of the lung is poorly inflated in the act of breathing and does not get enough oxygen. If the patients defensive mechanism is weakened, the bacilli gain a foothold in the lungs. They grow and multiply and form a tiny lump which is called a tubercle. The tubercle bacilli increase in number and destroy the tubercle changing it to a thick yellow liquid resembling cheese in appearance and is called caseation. Neighboring tubercles fuse, forming large tubercles called conglomerate tubercles. The constant absorption of toxins of the tubercle bacilli undermines the patients general health and produces all the various general symptoms of the disease.

The minimal or incipient tuberculosis is usually mild and the process affects only a very small area in one or both lungs and sometimes very tiny cavities develop but there are

no complications. Moderately advanced cases have one or both lungs involved but more extensively than the minimal cases, but there is very little or no cavity formation. For advanced cases have more extensive involvement than the previous group; there are usually numerous cavities in the lungs and serious complications.

There is an increase of symptoms, constant fatigue, loss of weight, cough and expectoration, night sweats, fever, rapid pulse, pain in the chest, nervous instability and hoarseness. These may develop into dreaded complications. The secondary infections are infections of the tuberculous areas in the lung by other bacteria such as pneumococcus, streptococcus, and others. Hemorrhage may occur in all stages of tuberculosis usually due to ulceration into a large blood vessel. Perforation of the lung results when the tuberculous process is near the pleura and causes pneumothorax. This also occurs when pus enters the chest. The symptoms of pneumothorax are a sudden attack of pain in the chest with severe shortness of breath cyanosis and cough.

Acute miliary tuberculosis is really a tuberculous septicemia or bacteremia. Occurs usually when a tuberculous nodule in the lung of lymph node degenerates into the blood stream. The tubercle bacilli then lodge in the lungs, the meninges or coverings of the brain or in the peritoneal cavity. These become covered with fine miliary tubercles. As a result symptoms of irritation of the brain, of the lungs or of the abdominal cavity develop.

The typhoid form of symptoms develop gradually, the temperature steadily rises, the pulse is rapid, the abdomen

distended and the spleen enlarged. The widal test is negative and the illness may continue for weeks and the patient finally dies.

The pulmonary form frequently follows whooping cough or measles and begins gradually with the symptoms of an ordinary bronchitis. The condition gets steadily worse and finally a bronchopneumonia develops which may be fatal.

In Acute miliary tuberculosis the onset is gradual. The patient has a general malaise headache, increasing prostration, anorexia, fever with profuse sweating. As the disease progresses a dry brown tongue, muttering, delerium and stupor develop. There is a low leucocyte count. The temperature curve is very irregular, pulse rapid and cyanosis is often marked. In moningeal infection there is intense headache, vomiting, rigidety of the neck, muscular twitching, delerium, occular and facial pulsies, stupor, coma, with Cheyne-Stokes breathing. The cerebrospinal fluid shows a slight increase of cells (lymphocytes) and sometimes the tubercle bacillus. The prognosis is almost always a resulting fatality. The severe illness usually lasts three or four weeks.

Tuberculosis affects many other organs of the body. In tuberculosis of the skin although uncommon, maybe contracted by innoculation but is usually through the blood stream. It is almost never fatal. It can be treated by general care and ultra violet light using surgery dressings for protection.

Tuberculous infection of the eye is more common or the surface structures. It is contracted through the blood stream and is rarely fatal.

Infection of the spleen, liver, muscle is a terminal

condition from the blood stream carrier.

Tuberculosis of the Larynx is secondary to pulmonary. It is very common as 5% of pulmonary infections have lyarngeal tuberculosis. It is treated by local and general ultra violet. The results depend upon the management of the treatment of the pulmonary infection. This phase will be discussed in a later chapter.

Tuberculosis of the kidney is rare in young people. It usually develops between the ages of 20 and 40. It is almost impossible to discover the portal of infection. The symptoms are pain, bladder irritation, hematuria. Bladder symptoms are progressive characterized by great urgency and tenesmus which may become so great that the patient is passing urine almost constantly. A physical examination will show tenderness over the kidney and in the suprapubic region. A cystoscopic examination is necessary to determine the affected kidney. Xrays in the late stage are characteristic. The only effective treatment is nephrectomy and then only after the soundness of the other kidney has been established. This operation should be followed by prolonged constitutional treatment.

Tuberculosis of the prostate is usually associated with tuberculosis of other parts of genito urinary tract. The treatment consists of radical removal of the prostate gland, the seminal vesicles and the epedidymis if it is involved.

Tuberculosis may invade any of the bones of the body. The infection is carried through the blood and usually attacks the ends of the bones in the epiphysis. The infected area presents a moth eaten appearance which is characteristic in Xray pictures. The bone abscesses are slow in forming, often

taking years. To distinguish them from acute inflammatory abscesses they are called cold abscesses. These often burrow, forming long sinuses far from the original site of bony infection. Sometimes under the psoas muscle forming a psoas abscess. The most common sites of infection are the knee, hips, elbow and the vertebrae. When the vertebrae are infected it is called Pott's disease. There is severe pain when infection reaches the periosteum. There is a rigidity of the joints due to muscle spasm about the joint. This muscle rigidity also causes a limp and in some cases deformity. The treatment of orthopedic tuberculosis will be discussed in a later chapter.

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Chapter Four
Treatment

(Chapter IV--Treatment)

The most important treatment of tuberculosis is prophylactic. The dissimination of information concerning the prevention of the disease; the supervision of schools, tenemint houses, factories and other public building is of utmost importance. The systematic inspection by skilled veternarians of dairies and slaughter houses with the view of declaring unmarketable the milk and meat of tuberculous animals is another step in preventing the spread of tuberculosis. The suppression of promiscous expectorating in public places and the establishment of special hospitals and dispensaries for the indigent suffering from tuberculosis, furthers the campaign. Tuberculous patients should be taught to expectorate only into proper receptacles and burned. They should sleep alone.

Medical treatment of tuberculosis has developed considerably throughout the ages. The essential treatment for any tuberculous infection is rest.

The treatment of acute miliary tuberculosis, usually fatal, is according to the predominant symptoms. The typhoid form is treated like typhoid fever; the pulmonary form is treated like pneumonia and the miningeal form is treated like other forms of miningitis principally by repeated lumbar puncture to remove the infected spinal fluid.

The treatment of acute tuberculous pneumonia is like any other form of pneumonia but the usual precautions should be taken to prevent spreading the infection. When the acute stage is over, fresh air and overfeeding are the principal methods employed.

The main object of treatment in tuberculosis is to increase the resistance of the patient which is accomplished by overfeeding, by rest, by living in the open and by removing all foci of infection. These principals were proven by Trudeau. The disease is treated best in special sanatariums, but if finance and social conditions make this impossible, effective treatment can be carried out at home.

For a period of time climate was considered of great importance in treatment however with the new methods of treatment climate plays little importance with the exception of sunlight. The diet should be high calorie and rich in calcium.

Cod liver oil is frequently used as a tonic. Calcium preparations are sometimes used to stimulate healing in the tubercles.

Symptomatic treatment is of great importance. The excessive cough is treated by codeine and sometimes by inhalations of steam with benzoine, creasote, or oil of pine needles. It is sometimes necessary to give tonics to stimulate the appetite. Cold sponges are given for high fever. Fever denotes bed rest.

Hemorrhage is often a serious complication and when it occurs the patient should be put to bed immediately and the foot of the bed elevated. No food is given by mouth, only ice pills. Morphine is indicated and a blood transfusion to increase blood clotting. Ergot is sometimes ordered to contract the bleeding vessels in the lung. Pneumothorax is treated by bed rest until the air is completely absorbed.

Artificial pneumothorax is a method frequently used in pulmonary tuberculosis when other measures fail. It is

performed by inserting a needle into the chest and allowing 100 to 300 c.c. of air nitrogen gas to enter gradually by means of a special apparatus. This causes the collapse of the lung and promotes healing.

Tuberculosis of the glands of the neck, often called scrafula, is very common in children of tuberculous parents. The glands become extensively involved and form what is known as a cold abscess. This often breaks through the skin and causes marked disfigurement. It was formerly an operation to remove tuberculous glands by extensive dissections but these disfiguring operations are no longer necessary. In the early recognition of glandular tuberculosis, hygienic treatment aided by tuberculin, Xray and radium will cure most cases without serious disfigurement. Occasionally it is necessary to puncture a gland which has broken down. The treatment of tuberculous glands is the treatment of early tuberculosis in children.

Tuberculous infection of the mediastinal glands is common in children, particularly in infants under two years of age. It is often the only demonstrable tuberculous lesion and from it later, general tuberculosis may result. The tuberculous glands can be recognized by the Xray.

Empyema due to tubercle bacillus is secondary to pulmonary tuberculosis. Tubercle bacilli escape from the lung into the pleural cavity and lead to an exudate and finally other germs invade the pleura and develop to empyema. There are two methods of treatment; the first, the closed method where a catheter is inserted in puncture wound made by a trocar between the ribs. At regular intervals pus is withdrawn

and Dakins two ounces solution, is instilled until drainage is free of germs.

When closed drainage fails, open drainage is used. A rib resection is done and a drain is inserted. It is necessary for frequent sterile dressing applications to be made.

In tuberculosis of the lung, rest has been a long standing treatment. Only in recent years has the value of lung compression by either fluid or air been recognized. Dr. Fordanini an Italian surgeon, first recognized this method of treatment. When adhesions form, this treatment is impossible. Surgical treatment is used in these cases. The phrenic nerve, one of the branches of the cervical plexus supplies the diaphram, one of the most important resperatory muscles, and controls its upward and downward movements. When it is divided , the paralyzed diaphragm rises in the chest and remains fixed. It compresses the lung, reducing its capacity about one third and so puts it at rest. Many cases of tuberculosis, especially, those with cavities in the lower lobes of the lung are cured by this operation.

Thoracoplasty consists of resecting portions of the ribs from the first to the eleventh. The ribs swing in thus reducing the capacity of the chest cavity and putting the lung at rest. This is permanent.

Nursing care of this operation is very important. A comfortable position for the patient is very necessary. Shock must be watched for closely. This sometimes appears as late as the second day and must be treated as shock.

Coughing must be prevented if possible and the patient

must be supported in any case with pressure over the incision. Narcotics may be necessary but as this is a long convalescence care should be taken not to form a habit. The drainage will be profuse because of extent of incision therefore necessitates frequent sterile dressing.

Tuberculosis of the bone and lymphatic glands responds well to ultraviolet administered as heliotherapy or air cooled quartz light. If care is used chronic pulmonary tuberculosis responds well. Treatments are not usually given directly over the chest in these cases.

Tuberculosis of the bone is a very prominent and common cause of many malformations of bones in children. Tuberculosis of the spine, commonly called Pott's disease, is a chronic destructive ostetis of the bodies of the vertibrae. These form the anterior or weight supporting portion of the spinal column. Tuberculosis starts as a focus in the body of the vertebra and the disease may spread very rapidly and cause deviations laterally or a Kyphosis or lordosis. In the cervical region, an abscess may develop due to the destructive process in the vertebrae. They generally appear as retro-pharyngeal abscess and cause difficulty in swallowing. These abscesses under adequate fixation and recumbent treatment will generally absorb. Generally the more vertebral destruction present, the more likely is there to be an abscess. Calcification in an abscess is usually a sign of healing and a sign of age.

If the disease is extensive and involve many vertebrae, one or more abscesses may occur. Distruction of one or several vertebrae may occur as well as involvement of the ribs, and

articular and muscular processes, such as the laminae and transverse processes. Such extensive bone destruction has led to spinal cord pressure, and even complete section resulting in paraplegia.

The whole object of treatment is to protect the vertebral bodies from jar and superincumbent pressure until a cure is established. Therefore the superimposed weight must be removed from the part affected, and the spinal column so fixed as to secure rest and protection from motion to the vertebral bodies.

Recumbency is the safest and best method of treatment applied in one if the following ways;

1. Bradford frame in dorsal and lumbar Pott's disease.
2. Head traction in cervical Pott's disease - Thomas collar.
3. Plaster shell.
4. Plaster jacket.

The Bradford is a flat bed frame and gives rest and support to the diseased back which cannot be obtained on a soft mattress. The Whitman is an arched frame with the same principal, made of iron piping the length of the patient plus one inch, thus covered with two canvases laced tight and in such a manner as to leave an open space under the buttocks and to prevent any sagging. This facilitates the use of the bed pan. The patient is fastened securely by means of straps of cross webbing. The patient is moved and turned only with assistance.

The clothing should be made for convenience and comfort. The child should be able to feed itself and drink through a

tube of some sort. The daily bath is important and the patient must be kept supported at all times. Pressure areas must be watched for and prevented and remedied.

Tuberculosis of the hip is usually first noted by a limp. Motion in the hip joint in the acute stage is impossible and attempts to move the leg causes a great deal of pain.

The object of treatment is to remove all bruising of the joint by jar or by motion and give the part entire rest. This is done by fixation, traction and protection. Fixation is accompolished by putting the child in a recumbent position on a Bradford frame. Traction is accompolished by pulling an adhesive retension on the leg on the affected side. The skin should be watched closely for excoriations, discolorations and edema.

Tuberculosis of the knee joint in children occurs next in frequency to tuberculosis of the spine and hip, and is followed in frequency of occurrence by disease of the ankle joint. It is characterized by intermittent lameness, general enlargement, stiffness and pain when part is in use. There is limitation of motion and heat over the infected part are universal symptoms. The treatment must be thorough and efficient. Fixation as long as there is any activity of the disease and protection until the epiphysis is normal in strength is important. In the acute stage the patient must be kept in bed with traction. For ambulatory treatment the leg must be in plaster from the pelvis to and including the foot. Thomas knee splint may be used with a thick sole on the shoe of the well limb in order that the affected limb may swing clear. Crutches are very necessary. Excision of

the knee joint is used only when conservative treatment fails.

Tuberculosis of any joints are treated with these same principals in view. There must be support, traction, fixation and rest for the diseased member. Nutrition, sunlight, and fresh air are important factors. Excision of the part is the part is the last resort.

Heliotherapy is used more as a prophylaxis and is recognized as such. This must be used cautiously and only for a short time exposure until the patient has become used to the treatment. It is very easy to burn a patient and so precautions must be taken.

Chronic tuberculosis is often affected the worse by pregnancy. Tuberculous patients frequently become pregnant but spontaneous abortion rarely occurs. The child is rarely affected at birth although this has been known to occur. In the early portion of pregnancy the patient often becomes markedly anemic and malnourished but later her general health improves and she gains weight and seems much better. After delivery however, the picture again changes and the disease often makes rapid progress. The fever cough, and other symptoms become rapidly worse. Incipient cases are often lighted up during the post partum period.

The tuberculous mother should never be allowed to nurse her child for two reasons. Lactation is a drain on her strength and may start her on the downward grade and secondly the close contact with the child may cause it to become infected from the mother even though the breasts are free from the disease.

There should be an early examination of her chest for

any sign of this disease. If it is discovered, the question is whether the pregnancy should be terminated or not. If so the patient must be put on a rigid anti-tuberculous treatment. This is best done in a sanatarium.

In terminating pregnancy the religious scruples of some patients must be considered. Most physicians agree if the disease is active and pregnancy is less than three months it should be terminated. If it is after the fourth month the pregnancy should be allowed to terminate.

In labor the patient should not be allowed to become exhausted; hemorrhage is especially harmful. Rectal ether with as little inhalation ether as possible should be used.

Potentially active cases of pulmonary tuberculosis, often in apparently healthy persons are found by a simple skin test. Countless thousands of former tuberculous patients owe their present health and in many cases, their very lives to a tuberculin test. The National Tuberculosis Association stresses the fact that, "The Tuberculin Test should be used routinely by every general practitioner". It is nearly fifty years since Kock devised the formula of his famous Old Tuberculin, and over thirty years since von Pirquet and Mantoux demonstrated the value of the tuberculin test as a means of diagnosing tuberculosis. Still greater numbers owe their escape from the white plague to the fact that "open cases" of tuberculosis were found and removed from their home, school, or place of business.

In spite of the great record of Old Tuberculen, students of tuberculosis have long recognized its shortcomings. Many

attempts have been made to improve it or to separate from it the active principle responsible for the diagnostic skin reaction in persons having acute or latent tuberculosis. The National Tuberculosis Association through its committee on Medical Research began a series of experiments several years ago which led to isolation of the pure protein fraction of the tubercle bacillus responsible for the tuberculin reaction. This product known as Purified Protein Derivative or more commonly, Tuberculin P.P.D. is now available for general use.

Tuberculin P.P.D. is supplied to the physician in tablet form. The tuberculin incorporated in these tablets is obtained from the filtrate of cultures of three human type strains grown on synthetic medium first adopted by the Bureau of Animal Industry of the U.S. Department of Agriculture.

In this tablet form tuberculin keepsfor a comparatively long period. In use these tablets are conveniently dissolved in a measured amount of sterile buffered diluent, furnished with the tablets, to provide .00002 mg. or .005 mg. (first and second strength test doses respectively) in .1 c.c. of the solution. The test doses must be made fresh as needed.

The technic of testing with P.P.D. is the same as the intradermal Mantaux testing with Old Tuberculin. The test is read in 48 hours. If the patient does not react to the first test strength of P.P.D., the second test strength is administered; if this is negative, the patient is not a reactor.

It is advised that tuberculin testing be done on the flexor surface of either forearm at approximately the junction between its upper and middle thirds. After cleansing the

selected area with alcohol, injection should be made with a 1 cc tuberculin syringe and a sharp, short-beveled tuberculin needle of 26 gage. All injections are to be made intradermally in such a manner as to form a small rounded elevation over the needle point.

Reactions to both test-strength doses should be read in 48 hours, at which time the reaction is most likely to be at its height. Positive reactions may be classified arbitrarily on the basis of size as follows; an area of swelling measuring from 5 to 10 mm. in diameter is read as one plus (+), those of 10 to 20 mm. in diameter as two plus (++), areas of swelling exceeding 20 mm. in diameter are read 3 plus (+++), the designation four plus (++++) being reserved for those areas exceeding 20 mm. in diameter and associated with definite skin necrosis.

A zone of redness less than 5 mm. in diameter and without perceptible swelling is read as doubtful, and cases falling into this group should be watched and retested at appropriate intervals.

A positive tuberculin test always means the presence of tuberculin infection. Failure to get a positive reaction, however, does not always exclude tuberculosis. Sensitiveness to tuberculin may be absent in acute miliary or generalized tuberculosis and during some infectious diseases. It is found occasionally that a child once a reactor may become negative to later tests. Rarely, a calcified pulmonary nodule may be seen in a child who fails to react to tuberculin. In such a case, it is probable that the focus of disease is obsolete

and that no living tubercle bacilli are present.

(Tuberculous Meningitis) Case Study

Master G.B. was admitted to the Good Samaritan Hospital, August 21, 1937 at 4:00 P.M. He was brought in on a stretcher and put to bed in a private room on the medical floor. Master G.B. was eleven years old. He was dark with a clear skin burned brown from the waist up by the sun. He was very fond of swimming and had acquired the sun burn while swimming. His mother and father were both alive and in good health. He had one brother who was alive and in good health. He was 15 years old.

The patient had been in contact with the mother's sister who had had an active pulmonary tuberculosis but at this time was under control. The patient had lost his right eye when three years old due to an acute iritis. The mother said that Master G.B. had had temper fits since he was a baby.

The patient was in apparently good health until August 7. The boy took sick suddenly with a chill and upset stomach and headache. He had a headache for two or three days. The mother thought he hadn't looked right all week. He had some temperature during the week, not over 102°. He had quite a bit of stomach pain and gas and had taken castor oil. As they lived out in eastern Oregon it took sometime to get to Portland to the family doctor.

However August 16th he was brought in to doctor M. On August 21, 1937, the patient grew steadily worse and so was admitted to the hospital complaining of head pain and vomiting. A tentative diagnosis was made of tuberculous meningitis.

The patient was in a stuporous condition all during the

time he was in the hospital. He responded very little and questions had to be asked several times before he apparently heard them.

The patient was in the hospital from August 21, 1937 to September 11, 1937 when he passed away. During that time, ten spinal punctures were performed. The first spinal puncture was done by Dr. M. assisted by Dr. W. and Miss V. Fluid was withdrawn under markedly increased pressure at this time. The boy was beginning to have a convulsion. This lasted several minutes with head and eyes turned strongly left. All the extremeties were spastic and jerking and the jaws were clamping. The pupil of the left eye was dilated widely and failed to respond to light. During the evening the boy was irrational and very excitable and complained of headache and stomach ache. This was August 21.

The second spinal puncture was performed by Dr. M. on August 23. The first fluid was bloody and the pressure 19 m.m. of nercury. About 10 c.c. were withdrawn.

The third spinal puncture was done by Dr. M. The fluid was slightly hazy. There was no marked increase in pressure on the 31 of August.

The fourth lumbar puncture was done by Dr. M. and the fluid was quite clear. This was the morning of September 2nd.

On September 2nd Dr. R. was called in. He performed a lumbar puncture. The initial pressure was 29 c.m. of water. About 25 c.c. of fluid drained. At the same time 5 c.c. of 2½% prontosil was diluted to 20 c.c. and injected by gravity into the spinal canal. The spinal fluid was grossly clear.

On September 3, another spinal puncture was done by Dr. R.

The pressure was 14 c.m. of water. The fluid as clear but tinged pink from prontosil. About 15 c.c. of fluid was withdrawn and 5 c.c. of prontosil were diluted to 20 c.c. and injected intraspinaly by gravity. A guinea pig innoculation was ordered by Dr. R.

On September 4 the seventh puncture was performed by Dr. R. and 5 c.c. of prontosil diluted to 20 c.c. were injected by gravity intraspinally.

On September 6, a spinal puncture by Dr. R. was done. Initial pressure was 15 c.m. of water and 5 c.c. of prontosil diluted to 20 c.c. were injected intraspinally. A guinea pig innoculation was ordered.

On September 7 and 8 spinal punctures were done by Dr. R. and sulfanilamide 20 c.c. of 8% was injected. On September 8, the pressure was 7 c.m. of water.

September 1st an Xray of the head was ordered by Dr. M. The report returned. An unusually large orbital fissure was seen in the right side and the right orbit was rather oblong in comparison with the left. The difference in density probably due to a slight rotation of the head and possibly to the fact that the patient had had an inflammatory process in the right eye.

On September 11, the patient deceased. An autopsy was performed by Dr. G.B.I. The external examination showed a body of a slightly emaciated white male, 15 years of age. The only structural change was noted in the right eye which had been mentioned before.

In the gastroentestinal tract, the mucosa of the lower ileum presented noticeable enlargement of the solitary

lymph follicles which bulged above the surface giving the mucosa a finely nodular appearance.

The left kidney presented; a cavity in the upper pole 1½ c.m. in diameter; a rough gray granular wall, and was filled with white caseous material. The major calex to the upper pole was dilated and the lining was rough and granular.

Over the basilor surfaces of the temporal lobe of the brain were found a few scattered surface nodules. Many were present over the dorsum of the cerebellum. The nodules were about 1½m.m. in diameter, pale gray in color and quite firm on palpation.

The final anatomic diagnosis was tuberculous meningitis and caseous tuberculosis of the upper pole of the left kidney.

Tuberculous meningitis is a form of acute miliary tuberculosis which occurs especially in children between the second and fifth year. There are two stages of symptoms. The stage of irritation where the symptoms begin gradually. There is a steady loss of weight. The person becomes restless and irritable and the disposition changes. Suddenly there is a rise in temperature and the child complains of intense headache and vomits. The vomiting occurs suddenly without warning, even when the stomach is apparently empty. Sometimes the rise in temperature is accompanied by convulsions. The child has night terrors and cries out, probably due to the headache.

In the paralysis stage, the temperature persists, the pulse becomes slow, the child begins to be drowsy and develops rigidity and retraction of the neck. Sometimes twitch- of the muscles or convulsions occur. Finally paralysis of

various muscles sets in. The paralysis usually involves the eye muscles. The pupils become dilated and the child may become blind. As the disease progresses the child gradually goes into coma and dies.

This is usually a fatal disease. It is treated principally by repeated lumbar puncture to remove the infected spinal fluid. The fluid is usually clear or slightly cloudy and comes out with considerable pressure.

The test for tubercle bacilli in the spinal fluid is made by inoculating some of the sediment from a centrifuged sample of the spinal fluid into a guinea pig. After three to four weeks the animal is killed and the organs are examined for the presence of tubercles.

From spinal fluid obtained from Master G.B. on the 4th of September an inoculation of a guinea pig was made; also another innoculation from spinal fluid September 6th was made. Both pigs were examined October 27th, the first was negative for tubercle bacilli and the second was positive.

The laboratory report on urine was normal. The blood report with counts taken August 21, 22, 26, and 30 was normal in regard to red count. There was a marked increase in leucocytes, polymorphoneuclear designating an acute suppurative condition such as meningitis.

August 23 at 1:00 P.M. a tuberculin test was given in the right forearm by Dr. W. Tuberculin has been used for the diagnosis of tuberculosis by hypodermic injection to show a reaction at the site of the injection. The reaction consists in a zone of redness, usually with a papule at the point of injection, which reaches its height in 48 hours.

There was no reaction. At 12:00 noon on August 28th, Dr. W. gave 1 c.c. of second strength tuberculin in Master G.B.'s left forearm. This was also negative. However this test is often negative in advanced and acute cases.

Codeine Sulphate grains 1/8 were ordered for restlessness whenever necessary but not more often than every four hours. Codeine is a sedative and is used to lessen nervousness and ally restlessness. The maximum dose is one grain.

August 30th, 40 c.c. of pooled serum was injected is the mixed blood serum from several individuals. It is of questionable value.

As it became increasingly difficult for the patient to take food, glucose 500 c.c. was ordered by Dr. R. to be given intravenously.

Sulfanilamide in varying sized doses was prescribed by Dr. R. Sulfanilimide at present is rather a new drug and has very little definite information concerning it. However it has been used with decided good effects in certain infections. The information that exists at the present time regarding the use of Sulfanilamide in various infections is in particular to hemolytic streptococcus, meningococcic and gonococcic infections. An accurate intake and output as far as possible was kept on Master G.B. Sulfanilamide has a tendency to effect kidney tissue and cause suppression of urine. There was no abnormality in the intake and output balance in this case.

(Nursing Care)

On August 21st at 4:00 P.M. Master G.B. was brought to the Good Samaritan Hospital by Dr. M. Master G. B. was in a state of rigidity. His pupils were dilated, pulse rapid, respirations were sterterous and his face and nails were cynotic. He gave no response. He had three severe convulsions lasting about five minutes. All his extremeties were spastic and jerking and his jaws were clamping. He salivated profusely. It was necessary for three nurses and Dr. M. to hold the patient in bed so that he could do himself no injury. A tongue depressor wrapped in gauze was forced between his teeth so that he would not chew his tongue. When dinally the convulsion wore off he was irrational and complained of headache and stomachache. Towels had to be placed under the patients head and mouth wipes were used to wipe the boy's mouth. It was necessary to change the bed and put on dry linen. The patient was very restless and irritable. At 4:30 P.M. Dr. M. performed a spinal puncture as has been described before. The patients temperature at this time was 103°, his pulse 164 and respirations 44. Codeine grains 1/8 was ordered by Dr. M. for restlessness and was to be administered by hypodermic. The codeine was given and the patient seemed to quiet down and seemed less restless. He seemed to require a large amount of water. He could not be left alone. His lips were rather dry and so vaseline was applied. At this time there had been no diagnosis but was considered a possible meningitis case.

An isolation unit was set up in the room. There was a wash basin and a lavatory in the room which was of great

advantage. A gown and mask were used and equipment for hand scrubbing and discarding of soiled linen and papers was placed in an advantageous position in the room. Isolation technique was observed until it was definitely known Master G.B. did not have epidemic meningitis.

At 7:00 P.M. he was given general evening care. His face and hands were washed, his hair was combed. It was necessary to change the linen that he had soaked when unable to retain urine. He was rubbed with alcohol and seemed a little less irritable. However the patient insisted on his mother staying in the room with him. Yet when he called her, he didn't seem to know when she answered him. His pulse had decreased to 130 and his respirations to 36. He was very difficult to take care of and fought the nurse trying to administer the care. He refused to allow a blood count to be taken at 8:00 P.M. At 8:15 P.M. he was given codeine grains 1/8 by hypodermic. In a few minutes he seemed quieter and ceased fighting the nurse. At 8:30 P.M. the patient finally permitted the laboratory technician to take a blood count. At this time he was given orange juice and seemed to relish it. At 9:00 P.M. his temperature by axilla was 99, his pulse 100 and respiration 30. He seemed very sensitive about having the nurse take a rectal temperature. He was much more quiet and finally went to sleep and slept until 10:30 P.M. When he awoke he seemed rational and asked to urinate. A specimen was saved and sent to the laboratory for routine work. He seemed quite uncomfortable. His back was rubbed with alcohol, the bed straightened and he was given water. He could not go back to sleep and at 11:30 P.M.

without any warning he vomited 500 c.c. of yellow vomitres. He complained of a headache and kept calling for his mother. He refused a hypodermic unless his mother was there.

At 12:00 Midnight his mother returned. This temperature was 100^{6}; pulse 72 and respiration 24. He was given codeine grains1/8 by hypodermic for restlessness. He seemed to be somewhat easier but still complained of pain in his head although not so severe. He was given orange juice and water at intervals throughout the rest of the night. He was restless and unable to sleep. He didn't seem to remember coming to the hospital.

At 4:00 A.M. August 22, his temperature was 100^{2}, pulse 64 and respirations 24. He said the pain in his head was worse and complained of a sore tongue. He was given codeine grains 1/8 by hypodermic for pain. He slept until 7:00 A.M. His temperature at this time was 99, pulse 62, respirations 24. He complained of dizziness and the pain in his head continued. At 8:45 A.M. he was again given codeine grains 1/8. He was given routine morning care of a bath, necessary clean linen, teeth and mouth wash, hair combed. Vaseline was applied to his lips.

The patient was very hard to talk to and very unresponsive. It was necessary to speak to him several times, then rather forcefully before he would answer. He appeared to have difficulty in catching the question because of loss of sense of hearing. Drs' M. and W. called at noon. The patient recognized Dr. M., the family doctor.

Master G.B. was given fluid nourishment at 12:00 noon. At 12:15 without any warning he vomited in projectile manner

a great quantity of yellow vomitus. At 12:45 his temperature was 99^4, pulse 54, and respiration 28. His pulse was much stronger than heretofore. He was given codeine grains 1/8 by hypodermic to quiet him. He seemed to be less restless in a little while and slept at intervals throughout the afternoon. At 4:00 P.M. his temperature was 99^4, pulse 40, and respirations 28.

At 6:00 he was given evening care. It was necessary to change part of the linen. He had liquid nourishment at intervals. At 10:30 he awakened with a start and complained of sharp pain through his head and right side. This lasted but a few minutes. He was turned on his side and seemed relieved. He went to sleep and slept until midnight when he again woke with a start. He seemed quite confused and picked his lips with his fingers. His temperature at this time was 100^6, pulse 50, and respiration 24. He was very drowsy and went back to sleep. He perspired rather freely and so it was necessary to keep his linen dry by changing frequently. At 7:30 August 23 the patient again awakened frightened and bewildered. The nurse talked to him and he seemed relieved and less nervous.

At 8:00 A.M. he was given general morning care. He enjoyed having his back rubbed and the baths seemed to please him. He was still in isolation. His mother and father were both very pleasant people. They seemed to appreciate everything the nurses did for the boy and appeared to have great faith in the nurses.

At 10:00 A.M. a spinal puncture was performed by Dr. M. as has been mentioned before. The patient was awake and

quiet. At noon his temperature was 98^8, pulse 64, and respirations 24. At 1:00 P.M. a tuberculin test was performed by Dr. W. The patient seemed fairly comfortable. He was turned on his side and a pillow was put against his back for support. He drank a little orange juice then went to sleep. At 4:00 his temperature was 101^4, pulse 64, respirations 16. He went back to sleep immediately and slept until 6:00 when he was given general evening care and an alcohol sponge. His temperature at 8:00 P.M. was 100, pulse 56 and respirations 18. He was very drowsy and went immediately to sleep. At 12:00 midnight he awakened, startled by a dream. At this time his temperature was 100^2, pulse 54 and respirations 18. He went to sleep and slept until 6:00 A.M. August 24th. He complained of a sore tongue. He was given general morning care. He ate very little breakfast because of the sore tongue. He seemed drowsy and went to sleep until noon. His temperature at this time was 99^8, pulse 54, respirations 18. He rested until 3:00 P.M. and at this time complained of feeling hot. His temperature was 101 (rectal), pulse 70, respirations 18. He was given an alcohol sponge. He went to sleep and slept until midnight. His temperature was 100^4, pulse 64, respirations 18, following the morning bath. He had another sponge bath which seemed to relieve him. He slept until 10:30 A.M. August 25th. He was then given general morning care. Vaseline was applied to his lips as they were becoming rather dry.

At noon he had a soft diet and ate fairly well, however it was necessary to feed him. He slept most of the afternoon until 5:00 P.M. when he refused his supper. He seemed very

restless and complained of pain and pressure in his head. He was given general evening care and codeine grains1/4 by hypodermic. This seemed to ease him and he slept very well until 8:00 A.M. August 26. He had general care and seemed to have a very good day until 6:30 P.M. when his temperature was 101, pulse 60, respirations 36. He was quite restless. The patient was given an alcohol sponge and this seemed to sooth him. His temperature went down gradually until at 3:30 A.M. August 27th it was 99^6, pulse 60, respirations 18. He slept quite well until 8:00 A.M. when he was given general morning care. He was becoming less and less able to help himself. However by this time it was determined that the patient did not have epidemic meningitis and so he was taken out of isolation. Precautions were maintained however, as the diagnosis was not complete. The patient was eating very little. It was necessary to urge him to eat the small amount he did eat.

His temperature ranged around 100 with little variance in pulse and respirations. At 6:30 P.M. he complained of a slight headache. Cold compresses were applied to the patients head and he seemed more comfortable. He had a fairly good night. Vaseline was applied to his lips at intervals.

He did not awaken until 8:00 A.M. August 28. He ate a small amount of breakfast and immediately vomited it without any warning. He slept most of the morning. He had fluid nourishment at intervals. At 2:00 P.M. he again vomited thick, yellow fluid and then seemed much less restless. He was very difficult to rouse and slept most of the time. His back was rubbed frequently and he had alcohol sponges at in

tervals. At 8:00 P.M. he complained of pain in his left eye. He was very restless and talked in his sleep. At 9:00 P.M. he was given codeine grains 1 by mouth. August 30th, at 8:00 A.M. the patient vomited the small amount of breakfast he had eaten. He was given a one pint soap suds enema but was unable to expel it. It was necessary to syphon it off. Fairly good resutls were obtained.

He drank small amounts of liquid nourishment throughout the day. At 1:00 P.M. he was given 40 c.c. of pooled serum intravenously by Dr. W. This has been described previously. At 3:00 P.M. his temperature was 101 (rectal), pulse 56, respirations 16. He slept practically all the time. August 31st the patient seemed to be in a semi conscious state. He did not seem to recognize anyone, not even his mother. He would call her and not know she had answered him. At 8:00 P.M. his temperature was 101^{8}, pulse 58, respirations 18. He was quiet all that night. He had to be turned and dried frequently. He voided involuntarily and seemed to make no response whatever.

Wednesday, September 1st at 9:00 he went to Xray and had a picture taken of his head, ordered by Dr. M. He was very drowsy all day. It was necessary to keep very close watch to keep the boy dry and to see that he was turned and supported. Towards evening he became quite restless. At 7:30 P.M. he was given codeine grains 1/2 by mouth and at 9:30 P.M. as he still seemed quite restless, codeine grains 1/8 was given by hypodermic. He slept then until 6:00 A.M. September 2nd. His temperature was still up to 100^{8} (rectal), pulse 66, and respirations 20. He seemed quite stuporous

and did not recognize anyone apparently. At 8:30 A.M. a spinal puncture was performed by Dr. M. This has been described previously. At 3:00 P.M. the patients temperature was 102^2, pulse 68, and respirations 9. His breathing was very stuperous and very irregular. Dr. R., a brain specialist was called in on the case. At 8:00 P.M. he performed a spinal puncture and injected 5 c.c. of prontosil into the spine by the means of gravity. The patients temperature at this time was 102^6 (rectal), pulse 76, respirations 22.

At 12:00 midnight the temperature was 104^2 (rectal), pulse 84, respirations 22. The patient was given Sulfanilamide grains V every hour for eight hours. At 12:30 P.M. an intravenous of 500 c.c. of 10% glucose in normal saline was started and discontinued at 1:00A.M. At 4:00 A.M. September 3, the temperature was 101^8 (rectal), pulse 70, respirations 20. At 6:00 A.M. the patient was given magnesium sulphate 30 c.c. as a cathartic. The patients condition seemed very poor.

At 8:00, Sulfanilamide grains X was given at four hour intervals. The patient ate very little. He would sip slowly from a straw but this seemed to be only a reflex. At 4:00 P.M. an enema, 1 pint was given which was effective for flatus and feces. At 6:30 P.M. a spinal puncture was performed by Dr. R. and again 5 c.c. of prontosil was injected by means of gravity. At 8:00 P.M. the patients temperature was 103, pulse 72, and respirations 24. At 8:15 an intravenous of 500 c.c. of 10% glucose in normal saline was started and discontinued at 8:45 P.M. The patients pulse

was 92 and respirations very irregular.

At midnight his temperature was 104^4, pulse 100, respirations 28. The patient was changed and turned often with frequent sponges. There was little change in the patients condition. He moved his arms continuously. At 8:00 A.M. September 4th the Sulfanilamide grains X to be given every four hours was continued. His temperature ranged between 103 and 104^4 all that day and until 4:00 A.M. Sunday September 5th when the temperature dropped to 102^4, pulse 100, respirations 22. September 4th at 8:30 an intravenous of 500 c.c. of 10% glucose was started by Dr. T. and discontinued at 11:30 A.M. September 5th at 2:00 P.M. 500 c.c. of 10% glucose in normal saline, intravenous was started by Dr. T. and discontinued at 3:10 P.M.

Patient seemed absolutely uncomprehensive. There seemed to be no response whatever. September 6th, a spinal puncture was performed by Dr. R. with instillation of 5 c.c. of prontosil. At 4:45 P.M. an intravenous of 500 c.c. of 10% glucose was started by Dr. T. and discontinued at 6:00 P.M. At 9:15 P.M. patient had large soft bowel movement in bed. All clean linen was necessary to keep patient dry. The patients temperature remained up between 102 and 103^2 from September 7 until the patient passed on, September 11. At 4:00 P.M. September 11th the temperature went up to 104^6, pulse 128, respiration 36, and remained up until 11:00 P.M. when the patient became steadily worse. The breathing labored, pulse rapid and cynosis was present. At this time respirations were 34.

From September 7th on, the patient seemed to be slowly

sinking. It was necessary to keep vaseline on his lips and try to put him in as comfortable a position as possible. He kept his eyes closed all the time. At 8:00 P.M. a levine tube was inserted by Dr. W. All nourishment and medicine was given through this tube.

September 8th and 9th his condition was getting slowly poorer. He perspired freely. His body twitched and jerked. He was never left alone.

September 11th his body started to become rigid at 5:00 A.M. By noon the body was held quite rigid. The eyes were washed with boric acid solution. At 4:00 P.M. the temperature rose and the pulse became rapid and weak. At 8:15 P.M. the body was twitching especially the muscles in the arms. At 9:00 P.M. the nasal tube was removed. The face was quite flushed and the pulse was rapid. At 11:00 P.M. respiration became very labored with mucous, frothy white, coming from the nostrils. Cynosis was present. At 11:50 the patient ceased breathing and was pronounced dead by Dr. H.

The body was prepared for autopsy by permission of Master G.B.'s father.

In taking care of this patient I learned the importance of isolation nursing when taking the responsibility of undiagnosed cases. I learned the great importance of keeping patients dry and off pressure areas. And from the gratified parents, who were so appreciative of all the nurses had done for their boy. I learned the imporatace of being able to reassure and allay the fears and nervous strain on relatives and loved ones. His mother and father were both charming

and intelligent people. We tried to make things as easy to bear as possible for them. When a spinal puncture was being performed it was much easier for the boys parents if they left the floor entirely.

I spoke to the mother of having their other son tested for tuberculosis. She was very glad to have my suggestion and seemed to realize the importance of detecting the disease in its earliest stage, if possible.

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Chapter Five

Public Health Measures

(Chapter V--Public Health Measures)

In 1899 the first attempt in America was made to provide systemic home visiting for the tuberculous as a class. Two medical students in Baltimore under the direction of Dr. William Osler followed to their homes the patients coming to the John Hopkins dispensary, instructing them in regard to diet, fresh air and the disposal of sputum. The social aspect was recognized from the first and by working in cooperation with relief agencies the results were very striking.

In 1903-1904 special tuberculosis visiting nurses took over this work. Every where the plan was hopefully begun. It had been found that power of resistance to the bacilli was increased by healthful living conditions. The greatest difficulty we have to combat in this work is the social structure of our cities, towns, and rural communities. Every social condition which effects the living conditions of the individual also affects the problem of tuberculosis.

It was stated by the National Tuberculosis Association that in 1904 the death rate from tuberculosis in the registration area of the United States was 200 per 100,000. In 1921 the death rate had been cut in half.

These workers found an almost unsurmountable problem in the fact that with no previous knowledge of the underlying principles of the germ theory, unused to consideration for others, often untrained to habits of personal cleanliness and living under conditions that make isolation difficult, no matter how carefully given, could they expect instructions to be rigidly observed. Another problem, the majority of nurses now graduate from their hospitals either with no

experience in the care of tuberculosis or with experience only in the care of the advanced and hopeless case. In consequence their interest is not aroused and tuberculosis work in any of its forms is often repugnant to them.

A step forward in our fight of tuberculosis is teaching health in the schools. Personal hygiene has played an important factor in this campaign. The emphasis laid on nutrition has been another important factor since the greatest danger is in childhood and since the undernourished child is most susceptible to infection.

Unfortunately data regarding the results of this kind of work is not easy to obtain. Only the lowered death rate of the next decade will show the value of the work being done, and those now being saved will probably never know they were ever in danger.

In combating this disease time, money and leadership are essentials. Programs must be worked out and followed and then revised when necessary. It is necessary to discover, the contact and suspicious cases, with consequent arrangement for diagnosis and treatment; the new positive cases; prevent the spread of infection; care by instruction or nursing service those who must remain at home; co"peration of agencies for the welfare of the patients and the protection of the community; routine duties of a nurse at clinic, dispensary or fresh air school, public education; and last, record keeping. All these things must be performed in our fight of tuberculosis.

There are many hazards in the field for the tuberculosis nurse. She must be extremely tactful in dealing with

patients, families and even with the physician. She may be confronted with positive laboratory records and a physician who refuses to accept the facts or who refuses to tell the individual or cooperate in any way. The nurses greatest ally is her ability to persuade the infected individuals to protect themselves and their loved ones. Here education has played an extremely valuable part. The nurse has to work very slowly in most cases. She must have the ability to impart her knowledge to her patients in a way that is acceptable to them. The nurse must have infinite patience because her ideas will have to be suggested over and over again in most cases. An ingenious nurse will find many ways of getting her patients out of doors; of giving them work to do that will not be beyond their resources physically. Occupational therapy has played an important part in this phase. Idleness to most people is very boring and if compulsory over a long period of time is very detrimental morally.

Full and adequate records should be kept and follow up work even after evident recovery is essential. Periodic check-ups are advisable and necessary.

Tuberculosis presents no simple problem. It is confined to no sex class, race, country or age. It has existed for centuries and can only be conquered by untiring labor and a fine devotion to a human cause.

Chapter V Public Health Measures.

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Chapter Six

Bacteriology and Pathology.

Bacillus tuberculosis is the causative organism of tuberculosis. It is found in the excreta of those that have the disease, in the milk of tuberculous cows, in the rooms that have been occupied by persons with tuberculosis, and in dust that has been contaminated by material containing the bacillus.

This organism is a slender, rod-shaped, nonmotile, non-spore-forming, acid-fast bacillus. The components of the cell that render tubercle bacilli acid-fast also render them more resistant to such detolerious influence as drying, germicides, etc. Direct sunlight however destroys them in one to two hours. They are destroyed by the temperature of pasturization. Carbolic kills the bacillus in sputum in five or six hours.

Bacillus tuberculosis grows very slowly and only on certain media. Two or three weeks may elapse before the organism is demonstrable. The Bacillus grows best at body temperature but will grow in atmosphere with temperature as low as 29°C. or as high as 42°C. The bacillus must have plenty of oxygen for abundant growth.

Tubercle bacilli do not produce exotoxins, but poisonous products that are partly responsible for the symptoms of tuberculosis are liberated when the bacilli undergo disintegration. When tubercle bacilli are grown artificially, the culture medium contains a product or products known as tuberculin which is without effect on a non-tuberculous animal but produces powerful and characteristic effects when introduced into the body of a tuberculous animal. There are more

than fifty methods of preparing tuberculin, and the nature of each tuberculin depends to some extent on its method of preparation. Tuberculin and its use has been discussed in a previous chapter.

The attempt to demonstrate tubercle bacilli in the sputum from patients with pulmonary disease must never be omitted. In pulmonary tuberculosis, the sputum contains tubercle bacilli frequently in very early or minimal lesions if they are active, even when demonstrable evidence of cavity formation is absent.

One of the most important steps in the successful demonstration of tubercle bacilli is the selection of proper particles of sputum for examination. Small purulent or caseous particles should be fished out of the sputum and smeared thinly on microscope slides. After the smear has dried in the air, it is fixed in the flame in the usual way. One of the most reliable staining methods is that of Ziehl-Nelson. The slide is flooded with carbol-fuchsin and heated to steaming. Carbol-fuchsin is 3% basic fuchsin in 95% ethyl alcohol using 10c.c. with 5% Aqueores solution of phenol using 100c.c. The heating must be continued for three or four minutes. The slide is then rinsed with water and decolorized with acid alcohol until no more color is shown in the washings. Acid alcohol consists of concentrated hydrochloric acid 3cc. in solution with ethyl alcohol 95% using 97cc. The slide is then counterstained with methyline blue.

If tubercle bacilli cannot be demonstrated in repeated direct smears, the sputum should be concentrated. If the

expectoration is scant, sputum collected from 24 to 72 hours must be used. It is mixed in equal proportion with a 3 to 4% aqueous solution of sodium hydroxide and is shaken until quite homogeneous. The mixture is then cintrifuged at high speed. The sediment is spread thinly on a microscopical slide and stained by the Ziehl-Nielson method.

If tubercle bacilli cannot be demonstrated by direct smear or after concentrating the sputum, isolation by cultural methods should be attempted. Since the sputum always contains fast-growing secondary micro-organisms, it must be prepared for culture in such a way as to destroy these. This can be done by treating the sputum with alkali or acid. Various media may be used for culturing the nuetralized sputum. Corper's glyceral potato medium has given very satisfactory results. This consists of a mixture of 500cc. of whole egg, 100cc. of egg yolk. To 500cc. of a 12% volume of glycerine in water is added 125 grains of thinly sliced, freshly peeled potatoes, autoclaved at 15 pounds pressure for 30 minutes; after cooling, 400cc. of the supernatant fluid are added to the egg mixture and 12cc. of a 2% aqueous solution of malachite-green oxalate. The whole mixture is well beaten and filtered through several layers of gauze into a tubing funnel. Ten to 12cc. are filled into each culture tube. The tubes, in position to give a long slant are heated in an Arnold sterilizer at 85°C for one hour. The tubes are incubated for four days.

If all preceding methods are unsuccessful, animal inoculation should be resorted to or it may be done simultaneously with cultural methods. In this the sputum must

be prepared as a bolus and neutralized. One to three cc. of the sediment suspended in saline solution is injected into one or two guinea pigs in the groin. The inoculated animal should be killed as soon as the lymph nodes nearest the point of injection are easily palpable. If the lymph nodes do not enlarge, the animal should be killed six to eight weeks after the injection, as every inoculated animal must be autopsied. If inoculation yields a positive result, an enlarged and caseated lymph node is found near the point of inoculation. The lymph nodes in the lumbar region are enlarged and caseated, tubercles may be found in the spleen, liver and lung. The extent of the tuberculous involvement depends on the interval between injection and autopsy, and on the virulence of the bacilli.

In patients who do not raise sputum, particularly in children, tubercle bacilli can sometimes be demonstrated in direct laryngeal smears. Material obtained from laryngeal swabs should be examined in smears, cultures and animal inoculations.

For the demonstration of tubercle bacilli in other body fluids such as plural, pericardial, or peritoneal effusions, pus from cold abscess or sinuses, urine, feces, spinal fluid, excised tissue, and gastric contents, essentially the same methods are used as for sputum.

If sputa and gastric washings are carefully and repeatedly examined, negative results are of distinct diagnostic value. This patient is unlikely to have an active pulmonary tuberculosis. Negative sputum signifies that at least three adequate specimens per month have been found

negative on direct smear and on concentration.

Tubercle bacilli may be spread to different parts of the body by; the lymph stream, the blood stream, permeation of adjacent tissues, the natural passages such as the ureters from the kidneys to the bladder, and expansion over a surface. Spread by the lymph stream is the most common. When material heavely laden with tubercle bacilli breaks into a blood vessel and spreads widely over the body, forming secondary foci, this is known as miliary tuberculosis. If this affects only one organ extensively this is known as localized miliary tuberculosis.

The body does not try to destroy tubercle bacilli primarily by anti-body formation and phagocytosis as it does many other bacteria, but there is a multiplication of tissue cells which engulfs the bacilli forming restricting barriers around them. These are known as tubercles. A tubercle consists of an inner zone containing tubercle bacilli, epithelioid cells, and giant cells surrounded by a zone of lymphoid cells and an outer zone of fibrous tissue. A tubercle may enlarge singly or a number of small tubercles may merge to form a large tubercle, known as a conglomerate tubercle. This interference with the nutrition of the tubercle leads to a peculiar type of necrosis of the center of the tubercle that is characterized by the presence of a greasy lipoid material. This is known as caseation necrosis.

This caseated tubercle may undergo calcification which is a reperative process or liquifaction which in certain parts of the body leads to the formation of cold abscesses; in the lungs it may convert a closed tuberculosis into an

open one, and may convert tubercles situated near the surface of the body into ulcers. Even in necrosis the tubercle may still contain living tubercle bacilli.

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Chapter Seven

Summary of Chapters.

(Chapter VII A Summary of Chapters)

The words used to describe the types of lesions of tuberculosis, are exudative or productive. In exudative an air saeor sacs in the lung involved becomes filled with blood serum and similar to pneumonia. In a productive lesion, the tissue which forms the frame work as the boundaries are involved. First caseation is the ingredients in the sputum which carries the tubercle bacilli. Cavitation is the breaking down of the tissues into cavities. Calcification is the depositing or laying down of lime salts in a cavity. The last stage is the fibrosing of the nodule.

The symptoms of tuberculosis are concentrated under the causes due to mal-nutrition, exposure, direct contact, and ignorance of the disease. The technical symptoms are numerous. Loss of weight over a period of time with no evidence of cessation. There is an increase in temperature and pulse and a general weakness with nervousness, irritability and general malaise. There is an instant feeling of fatigue with frequent colds and loss of appetite. Night sweats are common. There is a hoarseness of voice and dyspnea with a hacking cough. Hemorrhage brought on when coughing is extremely distressing to the patient and the relatives. Anemia usually develops unless the case is diagnosed and treated early.

The standard classification of the disease falls into there phases; the first, minimal in which there is no cavity formation with very little pain or effect; the second, moderately advanced with a cavity of maximum 4 centimetres with moderate pain and effect but with increasing symptoms

if not treated; for advanced, the cavity is over 4 centimetres in diameter and the symptoms are severe.

In diagnosing a case the doctor must have the following information. The history of contact or previous infection is very important. The symptoms of loss of weight, weakness, hemorrhage, increase in temperature, coughing with expectoration help form the framework of a diagnosis. A physical examination by a competent physician is very important and necessary. Xrays, both flat plate and fluoriscope must be obtained and carefully studied. Smear and culture examination of the sputum are definite factors in diagnosis. In blood work an increase in the staff cells is important. The sedimentation rate must be watched closely.

Medical treatment stresses rest, diet corrections, hygiene and sanitation and climate changes. Tonics to build a systemic counteraction is important, as examples cod liver oil and iron compounds. Tuberculin as a determiner of infection is used in connection with Xray.

Surgical treatment has made great advancement in the last few years. The first and most important fact to be considered is local rest of the part. This is accomplished in several different ways. Posture is invaluable and in conjunction with the use of weights to obtain mobilization, it has the greatest effect. Rest of the lung is brought about by artificial pneumothorax or oleothorax which is less common. Pneumolysis or freeing of adhesions is often very beneficial. When other methods fail a thorocoplasty may be performed, more commonly called rib resection. The phrenic nerve may be severed and allow the diaphragm to converge in

the lung and cause pressure. A scalenectomy allows the lower or first ribs to swing in and cause pressure on the lung enough to give rest to the part.

The prognosis depends upon numerous factors; the verilence of the tubercle bacilli, the resistance and cooperation of the patient, the extent location and nature of the disease and the complications that have developed.

Since Robert Kock's discovery of the tubercle bacillus in 1882 the treatment of tuberculosis has improved with an incredible degree of success. First of all people are realizing more and more, the value of frequent physical examinations. This gives the doctors a chance to determine, by means of a stethoscope, any change of sound of air through the trachea and bronchi to the lung. Also the Xray is used because of its value in detecting any changes from the normal. Doctors have found the most certain method of diagnosing tuberculosis to be the examination of the sputum in order to determine the presence of tubercle bacilli. During the past few years it has been found helpful in the diagnosis of a certain percentage of cases of tuberculosis, to examine the blood serum, employing a serological test. This test is not generally used, as it is at present of only limited value, and of a highly technical nature, and is employed only in certain sanatarium and laboratories with the hope that it may become so perfected that it will be useful as a means of diagnosis when other tests and examinations fail. At the present time, tuberculin is of considerable value as a diagnostic test but need not be tried in cases where the diagnosis may be made by other methods. A positive tuberculin test only

demonstrate the fact that infection has taken place, but it does not tell that the disease is present.

When we see and know these things about the dreadful menace tuberculosis is to society we should do all in our power to prevent it. One way for the patient himself to keep this dread disease from spreading is to cover his nose and mouth while coughing or sneezing. The hands and face should be kept clean and all utensils should be used only by himself. The patient should sleep in a bed by himself. Everyone should have frequent medical examinations and tuberculin tests. These things are safeguards for ourselves as well as others. This is a very serious problem today, for the loss of life, loss of wages, and cost of treatment of tuberculosis at the present time in the United States aggregates almost one billion dollars annually.