ACUTE EXUDATIVE TUBERCULOSIS

by

EVELYN VEE COHEN

A THESIS SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

UNIVERSITY OF WISCONSIN

1936

ACUTE EXUDATIVE TUBERCULOSIS -

A MEDICAL, SOCIAL, ECONOMIC PROBLEM.

CONTENTS

DEFINITION

Acute exudative tuberculosis is "an intense allergic response" of the body to tubercle bacilli.[18] It is one in which "response to bacillary protein is much greater than to bacillary lipoids."[22] "These acute inflammatory phenomena must be due to either infections caused by relatively large numbers of virulent bacilli or to the fact that they occur in persons whose tissues offer a favorable medium for their growth, and whose cells are highly sensitized to bacilli and bacillary protein."[22]

ALLERGY, IMMUNITY, HYPERSENSITIVITY

In any consideration of tuberculosis, the factors of virulence of the organism and resistance of the host are of first importance. "It is possible to conceive that each type of bacillus (human, bovine, etc.) elaborates a chemically different kind of poisonous material, that a virulent bacillus elaborates more of its particular kind of poison than does an avirulent one, and that species are susceptible or resistant depending upon whether or not their tissues are chemically constituted so that they can be acted upon by the particular poison in question. Natural species and individual resistance or susceptibility will thus depend upon the degree to which the previously uninfected animal body in question is able to satisfy the growth requirements of the particular tubercle bacillus under consideration."[25]

Petroff[19] in considering the question of immunity and resistance says that there is no natural immunity against tuberculosis in the human race, but that "individuals and races differ in susceptibility to the disease is well recognized."[19]

Can immunity be inherited? It has often been pointed out that a more acute fulminating tuberculosis exists in patients with healthy parents than in those whose parents had some history of tuberculosis. This phenomena is often times attributed to an inherited resistance and decrease in the pathogenicity of the tuberculosis virus by family passage. Petroff[19] believes that the resistance noticed in such families is due probably to the periodic infections to which the children of tub-

erculous parents have been subjected.

There is also no demonstrated passive immunity that can be passed from mother to offspring, or by transfusion of body fluids, or other tissue products from an immune animal to a normal one.

It has repeatedly been observed that an animal infected with a sub-lethal dose of tubercle bacilli becomes, after some days, protected against subsequent infection with much larger doses of bacilli than the animal could have originally tolerated. Rich and Mc Cordock mention the following facts in this connection: 1. "Resistance in rabbits and guinea pigs may be acquired by infection with either virulent or avirulent human of bovine bacilli, regardless of the animal's susceptibility to the infecting organism. 2. Resistance so acquired will protect indiscriminately against subsequent inoculations of virulent human or virulent bovine bacilli regardless of the animal's original susceptibility to the organisms of reinfection, i.e. the resistance is not type specific."[25]

Thus far, we doubt if a condition of complete immunity to tuberculosis ever exists in an individual. Immunity, in this paper, will be considered as one accompanied by a lessened sensitization with an increased power.of the body to resist and destroy reinfecting bacilli. This condition usually develops in cases of tuberculosis which are running a favorable course.

"Hypersensitiveness to tuberculoprotein is due to a change wrought in the body cells by circulating bacillary protein derived either from

the destroyed bodies of bacilli or from substances produced during their growth."[22] This phenomenon appears to be protective in character, although, if excessive, it may become harmful. The hypersensitivity reaction is greatest in the early stages and in the active stages of tuberculosis.

Allergy in this paper shall be used in the same sense as Rich and Mc Cordock have defined it. "The infected body becomes changed in some manner which renders the relatively bland protein of the tubercle bacillus capable of acting upon its tissues as a powerful irritant and poison. As a result of this change, the cells of the allergic body are more extensively damaged and killed by a given amount of tuberculoprotein than are the cells of the normal body, and, furthermore, because of this enhanced irritant action of tuberculoprotein on allergic tissues and because of the resulting more extensive damage and death of cells, there appears a more violent acute inflammation at the site of action of tuberculoprotein in the allergic body. More extensive damage and death of cells and more extensive acute inflammation constitute, therefore, the local visible expression of the action of allergy. Constitutionally, the greater irritative effect of tuberculoprotein upon the allergic body is manifested by the fact that fever, malaise, prostration, and even death will ensue when an amount of tuberculoprotein which is harmless for the normal body finds its way into the blood stream of the allergic one."[25]

Zinsser and Mueller[27] and many other students of immunology believe that allergy is primarily a protective phenomenon, but this viewpoint is

not accepted by all. Clinical evidence indicates that even if it is not primarily protective, there are at least protective phases to the reaction, as, for example, the quickened reaction, the increased phagocytosis, the inflammatory response, and the heightened body temperature.

Following the primary infection with the tubercle bacilli, the cells of the body become so altered that they react in an entirely different manner to any subsequent superinfection. According to Ornstein, Ulmar and Dittler[17] this altered reaction assumes two different forms. One is a true immunity response, the reaction which prevents the spread and migration of the tubercle bacilli and results in a proliferative or fibroid process o f healing. The other is a somewhat related reaction, but does not, on superficial thought, seem to be primarily defensive. This second type of reaction, the so-called allergic or hypersensitivity reaction, a primarily exudative or serous reaction, is one which tends to result in injury to the cell. It seems to parallel the immunity reaction, but not necessarily so. Pinner and Kasper have come to the conclusion that the tuberculosis infection "tends to produce a high state of allergy in the negro, but that he, unlike the white, does not readily acquire a state of increased resistance coincidently."[21]

"It is not an accurate generalization to say that the reaction to the first contact with the bacillus is always tubercle formation and that exudative inflammation never occurs except in the allergic animal. Either the normal or the allergic animal can respond to the tubercle bacillus with either tubercle formation or exudative inflammation. Which type of

reaction will occur primarily in any instance will depend greatly upon - a. The virulence of the infecting bacillus, b. the degree of natural and acquired resistance of the individual, c. the degree of allergy, and extremely important, d. the size of the infecting dose."[25]

Pinner[20] believes that more exudate is always found in an allergic than in a non-allergic individual. This author further says that dosage of reinfection of bacilli is the factor which determines whether this allergy means immunity or hypersusceptibility, prolongation of life or shortening of life. Allergy and immunity, always co-existing, are not identical conditions. Allergy may shorten or it may lengthen life, but immunity can have only one meaning.

"Allergy,"says Blacklock,"while an important factor in determining the type of tissue reaction is, however, not an exclusive one, and there appear to be many modifying circumstances such as the amount of reinfecting dose, the virulence of the bacilli, the resistance of the patient, and the localization of the lesion. It does not explain all the factors and at the present time the relation between it and the type of tissue reaction is often obscure."[2]

It is a known fact that all allergic animals made ill by fresh reinfections come through with much less anatomical tuberculosis if they survive their acute illness than do the originally normal controls given the same infections (as first infections) and not made acutely ill by them.

"Appreciating that concepts of immunity are derived from observa-

tions of comparative immobility of foci and restriction of infection, Krause finds in allergy a force that leads tothe semblance, the effigy of immunity.

1. Only the tuberculous, therefore the immune and allergic animal, can respond with acute inflammation and necrosis to tuberculoprotein and to tubercle bacilli.

2. That the normal non-tuberculous amimal cannot be made ill by tuberculoprotein or tubercle bacilli, while the tuberculous, immune, allergic animal is almost immediately upset by either.

3. The tuberculous, immune, allergic animal subjected to reinfection and rendered acutely ill thereby will, if it survives the illness, long outlive the non-tuberculous, now immune,non-allergic animal that receives, without suffering symptoms, an identical infection by way of its first infection."[13]

PATHOLOGY

The pathological studies in the acute exudative form of tuberculosis help greatly as a means of establishing this phase as one of the acute allergic manifestations of the body.

When tubercle bacilli enter the body, the body responds anatomically and "tubercle" is formed. This first response of the body usually results in nodular anatomical formations. After tubercle is formed, the body reacts to the tubercle bacilli in a new way and the result is non-nodular anatomical formations. This is known as the allergic reaction. The allergic state is usually detected three weeks after the first inoculation; but if the first infection is very large, it may be detected as early as the fifth day after inoculation.

"The allergic reaction (tissue allergy) is evoked by the contact of living or dead tubercle bacilli, or of dissociated protein derivatives (tuberculins) with the tissues of tuberculo-allergic animals. The essence of allergic reactions is exudation, appearing as typical inflammations in solid tissues and effusions in serous spaces."[12] "An exudative lesion does not develop a new tissue, but an exudate consisting of various amounts of plasma proteins and cells both of histiolytic and haematogenous origin."[20]

Krause assembles the significant anatomical features of the allergic reaction as follows:

1. The response of the tissues to tubercle bacilli is exudative, as contrasted with the native

response of proliferation. The effect is an inflammation and the visible result is a diffuse formation, again as contrasted with the nodular circumscribed structure of native anatomical tissue response.

2. The inflammation of allergy develops with amazing rapidity; visible anatomical effects are in this respect in striking contrast with those that follow primary infection and appear as nodular tubercle.

3. In the allergic animal new nodular tubercle of reinfection itself develops more promptly than in the animal of first infection.

4. Foci of tubercle existing in the body at the time of reinfection may also undergo acute inflammatory allergic reaction, and, as a result, have their fibrosis markedly stimulated."[12]

The allergic reaction may be anything from a slight hyperemia to a severe inflammation with exudation which is poured into the tissues and natural channels - it infiltrates the latter, it does not replace it. "It is a process which involves the internal surface of the lung and not its framework; structural relation is unaltered. When this lesion undergoes caseation, exudate and all the celluler elements of the in-

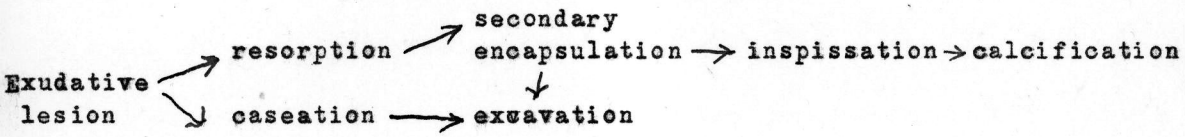
volved organ caseate, leaving only resistant elastic fibers intact."

Exudation does not necessarily mean that all the elements are derived from the blood. The cells may be proliferated alveolar, adventitial and fixed wandering cells. Points of differentiation are not in the derivation of cells, but in the fact that the productive lesion grows by intusseption (expansive growth), that it forms a true tissue, while the exudative lesion expands by infiltration and forms not a tissue but an exudate.

The degree of exudation in any lesion seems to depend on the degree of sensitization of the cells and thevirulence and dosage of bacilli, or bacillary protein which is responsible for the reaction. The greater the amount of bacillary protein set free, the greater the body's reaction; the greater the predominance of cells and fibrin in the exudate; and, up to a certain point, the greater the stimulation of the host's immunity mechanism. Should the stimulation be excessive, on the other hand, immunity may be depressed. Perhaps also the temporary or more lasting nature of the various reactions as noted in different patients is dependent upon what element, be it serum, cells, or fibrin, predominate in the exudate. "This inflammatory reaction is probably to be interpreted as being the particular specific protective agency which is necessary at the time to cope with and prevent bacilli from spreading, or to hold the amount of reinfection to a minimum until a more efficient and more complete immunity has been established. After this has been attained, evidence of desensitization appears, as is shown by the fact

that the patient suffering from advanced lesion, even when his body is capable of adequate response, reacts less violently to large reinoculations than he did to the smaller doses in the early stage of the disease."[22]

Pinner[20] has outlined the fate of an exudative process as follows:



Before healing can be accomplished, the exudate must be cleared away. "That in the air passages can be eliminated in part through cough and expectoration and in part by the same method that rids the tissues of exudative products in acute pneumonia, viz. resolution."[22] Resolution is a regular part of healing in all exudative tuberculous lesions. It consists of an enzyme action by which the inflammatory products are broken down into soluble products which are expectorated or carried off through the blood, while the debris which is left is taken up by phagocytes and eliminated or destroyed. This according to various authors seems to be the regular course followed in exudative lesions. Resolution may be so complete in many widespread exudative lesions that after the attainment of healing an x-ray film may give no conception whatever of the extent of the previous process.

The time required for resolution to take place in tuberculosis, as compared with that in pneumonia, is much longer. While acute pneumonia will resolve and heal in a few days or in a few weeks at most, tuberculosis requires days for the simplest exudates, and weeks, and more often

months, for the cellular and fibrous exudates to be removed from the lung fields. "Why it differs so much in different patients is impossible to say, but it is probably due to the peculiar chemical and physical properties of the exudate itself as well as the tissues found in different persons." In the healing of tuberculosis, one will notice that exudative phenomena resolve slowest where the greatest amount of proliferation remains, or in other words, where the greatest amount of infection exists.

Severe exudative processes may also heal by the process of fibrosis even though caseation be present. "It must not be forgotten that the allergic reaction consists of two processes which are going on in the tubercle at the same time; a more severe reaction with tendency to softening in the immediate environment of the bacilli, particularly in the center of tubercles, and a milder reaction at the periphery, which stimulates the reticular cells to proliferation, causing them to produce new fibrils which may even penetrate to the center of the foci and replace caseous material by fibrosis and convert the tubercles into scars. It must also be kept in mind that digestion of the exudate and the removal of the broken down particles from the field is always going on."[24]

Severe exudative lesions sometimes go on to necrosis and cavity formation.

"Whether the acute pulmonary infiltrations may occur in virgin soil, or whether they require an allergic territory for their development, is open to speculation. They have been interpreted as manifesta-

tions of Ranke's hyperallergic secondary stage. It is probably safe to assume that the infiltrative lesions in question are, as a rule, foci of reinfection and not primary lesions, since it has never been reported that they show the picture and course characteristic for primary lesions, namely, healing by calcification and ossification, and simultaneous involvement of regional lymph nodes."[7]

SYMPTOMS

Theoretically, the acute infiltrations may be produced by exogenous inhalation infection, by haematogenous spread from the primary focus or from reinfection foci (apical scars) if present, or by bronchogenic spread. They have been explained, too, as acute circumfocal inflammations around an old focus, caused by reinfection. "Ashoff's studies, in accordance with Huebschmann's, indicates that each focus of reinfection starts with an exudative phase, regardless of localization and size of reinfecting dose."[7] Only when for reasons in the response of the host or for reasons in the bacilli, the foci become so large as to produce clinical disease are they the potential start of progressive pulmonary tuberculosis. But even then they may completely heal by resorption or fibrosis, or both.

Pathological and roentgenological evidence suggests that the acute exudative form of pulmonary tuberculosis is usually acquired in late childhood or adolescence.

Clinically this exudative form is often incorrectly diagnosed. It resembles in its onset the acute cold, the grippe infections and bronchopneumonia. In order to recognize this disease in its incipiency, it is wise to consider every unduly protracted cold and every attack of grippe or influenza (especially when there is no epidemic) as a possible acute onset of pulmonary tuberculosis. In a number of cases no symptoms are noted. The temperature may or may not be elevated; the pulse rate is usually increased. The important point is that its symptomatology bears no resemblance whatsoever to the textbook symptoms of incipient

tuberculosis.[8]

At the onset there may or may not be cough. When cough is present it may not produce any expectoration, although at times for a short period there will be a small amount of sputum. If tubercle bacilli are to be found in the sputum, they must be searched for within the first few days of the onset. "Fortunately, about forty per cent (40%) of the cases have haemoptysis and tuberculosis is suspected."[16]

At the onset of the attack the patient may feel a trifle below par. This feeling of malaise, lassitude, fatigability may be no greater than that associated with an ordinary common cold. It soon disappears and with the subsidence of the cough and expectoration, the patient feels perfectly well. The entire symptom-picture usually lasts no longer than a few days to a few weeks - then the symptoms disappear and the patient feels perfectly well.

Ornstein, Ulmer and Dittler[17] have compared the symptomatology of the acute benign (exudative) to that of the acute malignant (caseous pneumonic) tuberculosis. It is as follows:

	Acute benign (exudative)	Acute malignant (caseous pneumonic)
1. Onset	1. Acute	1. Acute
2. Cough	2. Slight or absent	2. Moderately severe; always present
3. Expectoration	3. Scant or absent	3. Copious
4. Haemoptysis	4. At onset	4. May occur at any time
5. Temperature	5. Elevated only at onset	5. Irregular; elevated for a considerable time
6. Pulse	6. Elevated only at onset	6. Irregular; elevated for a considerable time

Pottenger[23] says that in the acute exudative form of tuberculosis, allergy manifests itself in the production of symptoms in three ways:

1. By producing general effects throughout the cells and tissues of the body by the toxins.

2. By producing reflex effects in different organs and tissues through impulses which are picked up in the inflamed lung and carried centralward over afferent sympathetic and vagus fibers, and

3. By producing local disturbances in the infected tissues.

He outlines the symptoms produced as follows:

Symptoms due to toxemia.	Symptoms due to reflex cause	Symptoms due to the tuberculosis process
1. Malaise	1. Hoarseness	1. Frequent and protracted colds
2. Lack of endurance	2. Tickling in larynx	2. Spitting of blood
3. Loss of strength	3. Cough	3. Pleurisy
4. Nerve instability	4. Digestive disturbances which may result in loss of weight	4. Sputum
5. Loss of appetite	5. Circulatory disturbances	
6. Digestive disturbances (hypomotility and hypo secretion)	6. Chest and shoulder pains	
7. Metabolic disturbances resulting in loss of weight.	7. Flushing of face	
8. Increased pulse rate	8. Spasm of muscles of shoulder girdle and crus and central tendon of diaphragm	
9. Night sweats	9. Diminished motion of affected side - lagging	
10. Blood changes		
11. Temperature		

In those cases in which no symptoms are present, the lesion can only be picked up by periodic examination. If the condition remains unrecognized at first, it may months later be again manifested by an

acute exacerbation with similar symptoms, as given previously. This second exacerbation marks a spread of the disease and usually does not result in complete recovery.

"The true condition is only to be vaguely suspected from the history of the illness itself, and the suspicion is made stronger if there is a family history of tuberculosis or of protracted contact with the disease."[7]

Krause[13] also believes that allergy creates symptomatology. He says: "After we artificially infect an animal for the first time, we do not find it exhibiting symptoms of illness until the allergic state is established by the infection. Or, just as significant, the only animals that can be rendered ill by inoculations of tubercle bacilli or tuberculoprotein are tuberculous, that is, allergic animals. The infected patient, long harboring tubercle and perfectly well just as long, has perhaps a few bacilli set in motion and sent to a new location, or at a long quiescent, asymptomatic focus he has an excess of bacillary protein react with focal tissue to set up an acute focal reaction. The result is an accession of malaise and fever, with perhaps cough and expectoration. These symptoms may last only for the day or for a week or less, and die down never to be repeated, or they may recur again and again until a more permanent state of illness is reached."

PHYSICAL SIGNS

Physical signs are scant. Breath sounds over the involved area are markedly diminished and are of the broncho-vesicular type. At the onset of the disease there may be a few high pitched moist rales scattered over the involved area. These rales, never numerous, seldom coarse, usually fine and high pitched, persist for a varying length of time. Dulness on percussion can usually be obtained over the involved region, provided that the area is extensive enough and sufficiently close to the chest wall to be revealed by this method of physical diagnosis.

"When one considers the exudative nature of the process, one is not at a loss to explain the pancity of physical signs. The oedematous water logged alveolar walls act as perfect felt-pad dampers and thus prevent the formation or transmission of breath sounds or rales. The percussion note, however, is not influenced by this factor, and is, therefore, dull."[17]

Ornstein, Ulmar and Dittler[17] have compared the physical findings of the acute benign (exudative) to the acute malignant (caseous pneumonic) tuberculosis. They are as follows:

	Acute benign (exudative)	Acute malignant (caseous pneumonic)
1. Breath sounds	1. Diminished in intensity; slight alteration in quality	1. Harsh and gross changes from normal quality
2. Rales	2. Few and variable; never coarse. Usually disappear early	2. Many and coarse - persistent
3. Percussion note	3. Dull	3. Dull to flat

X-RAY

A dense homogeneous shadow is seen on the x-ray in the acute exudative form of tuberculosis. This shadow may be of any size and may occupy any portion of the lung field. The shadow is very rapid in appearing. "The first change to be noted is a diffuse haze and impairment of illumination over the involved area. This is followed by a rapid increase in the density of the shadow, until the typical homogeneous opacity is obtained."[17] The shadow is "soft and flaky" when produced by an exudative inflammatory reaction.[22] Ornstein and Sampson in describing the x-ray say: "Assuming that there is no scar tissue or calcification in this area, we see mottling with decidedly ill defined margins, or a 'cotton ball' appearance as we prefer to call it, blending gradually with the surrounding lung tissue, which appears hazy and cloudy."[15]

Once the peak of the reaction has been reached and the process tends to resolve, the x-ray shadow tends to disappear. The shadowing first disappears at the periphery by means of a uniform diminution of the density - a reversal of its formation. The more usual manner of disappearance is by means of an irregular absorption of the shadowing, leaving a mottled patchy appearance with suggestion of cavitation.

Ornstein and Sampson in describing the x-ray findings when the exudate is regressing say: "Assuming that this cotton ball appearance indicates activity, as the disease retrogresses, the lung tissue surrounding the "cotton ball" appears to become better aeriated and probably at the same time we realize that the cotton ball is smaller and more

compact. Or again, we may see the cotton balls almost fade from view, leaving a more or less finely dotted appearance or little string-like shadows. As time goes on, when the patient is doing well, the mottling looses still more of its collateral haziness. The cotton balls become more compact or their appearance more discrete, the ray filtering through areas that were previously hazy."[15] Further retrogressive changes occur in this group, and the final picture is that of a well aeriated lung.

Serial x-rays, however, often after a period of no longer than six weeks to a few months, show that the entire shadowing disappears without leaving a trace.

EPITUBERCULOSIS

In recent years clinicians have described a benign type of lesion which has been named epituberculosis. It may involve extensive areas of the lung without being accompanied by symptoms. "The term has been limited to childhood, as though the process were different from that found in adult life; but I doubt very much whether epituberculosis differs in any way except in time and the extent of the exudative process from the allergic reactions which are regularly seen in plates of those suffering from pulmonary tuberculosis in the exudative form."[22] It cannot be other than an allergic phenomenon. The fact that it is so much more pronounced in childhood may be accounted for by the presence of a more highly sensitized state of thebody cells, due to the newly created property of specific reaction, and at the same time the presence of unencapsulated glands from which the amount of tuberculin necessary to cause the large reactions may escape.

Goldberg and Gosul also define epituberculosis as a "specific infiltration of a large area of lung tissue on an allergic basis."[19] Blacklock in speaking about this form of tuberculosis says, "The significance of this particular type of reaction has been explained by Aschoff and Ranke as the typical response of the tissues in a non-infected, non-allergic organism. Ranke further stated that the exudative type of lesion of the primary infection was rapidly followed by productive lesions surrounding the primary focus, which became rapidly encysted,while similar changes were also present in the related lymphatic glands. On the other hand, Krause found the initial response in the subcutaneous

tissue of the non-allergic guinea pig to be formative or productive, while that of the allergic animal was exudative. It must be remembered, however, that different tissues and different animals may react differently to a first infection. Allergy, while an important factor in determining the type of tissue reaction is, however, not an exclusive one and there appears to be many modifying circumstances such as the amount of reinfecting dose, the virulence of the bacilli, the resistance of the patient, and the localization of the lesion. It does not explain all the facts and at the present time the relation between it and the type of tissue reaction is often obscure."[2]

Epituberculosis is characterized by a more or less extensive exudative or atelectatic involvement of the lung without corresponding coextensive infection, and without corresponding illness on the part of the child. The onset is subacute. It usually involves the upper lobe. When present, the physical findings are those seen in a consolidation. There is dullness of the percussion note, and bronchial breathing may be heard over the area involved, accompanied by few or no rales. The child usually shows few or no symptoms of illness, and the physical condition of the patient is good. Sometimes the breath sounds are characterized by their weakness rather than other qualities. "The temperature is usually normal or only slightly elevated, probably because the child is examined too long after the reaction has taken place for the temperature change to be detected, for we would expect to find, at least in the more severe reactions, some elevation at the height of the reaction. I recently saw such a case within a few days of the acute reaction in which

the temperature had reached 102° F."[22]

In the x-ray film there appears a more or less homogeneous shadow density over the whole or greater part of the affected lobe, indicative either of exudative lesions, or more probably of exudative lesions and atelectasis. Some of these shadows will clear quickly, while others will last for weeks and months. In the latter case it is not improbable that some infection is also present. Finally, however, most of these lesions will disappear, leaving a surprisingly clear lung field. "An enlarged caseous or partially calcified hilar gland or glands, or a caseous lymph node, may be detected as the probable source of the bacillary protein responsible for the exudative lesion."[22] When atelectasis is present, it may be caused either by pressure on a bronchus or by closure of the lumen through swelling of the bronchial structures.

This lesion possesses the same characteristic appearance as the allergic reaction which is seen regularly in the exudative forms of adult tuberculosis. However, it shows little tendency to spread further or to destroy tissue. It is surprising to find how often there is no sputum and when present that it is so generally negative for bacilli, when the lesion is so extensive. It is easy to believe that the tuberculo-protein which escapes from the primary nodule or more probably from caseous tuberculous hilar glands into a bronchus finds its way out into the air passages and aided by the respiratory act and cough, diffuses into the tissues, causing the exudative reaction.

"We are of the opinion that much of the exudative allergic reaction

with its accompanying atelectasis, which is found in the course of adult tuberculosis, also may be due to the same cause with this difference: In adult life metastases are responsible for a larger number of the reactions, and the foci from which the bacilli and bacillary protein escape are always or nearly always in the pulmonary tissues, the glands being largely out of the picture. Again, the bacillary protein diffuses readily, and while the degree of sensitivity of the tissues of the child is high the danger of necrosis is small, probably because of the absence of large numbers of bacilli to settle at some particular point."[22]

In the exudative lesions of adult tuberculosis, metastases are common, but here, too, they are not coextensive with the infiltration, as may be inferred from the limited amount of scar tissue left after the exudate has been absorbed.

Added argument which heightens the probability of this condition being an exudative response on the part of the tissues to bacillary protein rather than to metastasizing bacilli is the fact that tuberculin, when brought in contact with the abraded skin of an infected animal or man, when dropped into the normal conjunctival sac, or when instilled into the bronchi, will produce a similar exudative response.

"Tuberculous infection has not been found in some cases of so-called epituberculous lesions, which have been studied post mortem."[22] "When serial films of the chests of children who have unhealed primary complexes are taken at frequent intervals, these shadows are found quite frequently. In fact, it seems reasonable that exudative lesions of this

character should frequently accompany caseating glands."[22]

TREATMENT

In many respects the treatment of acute exudative tuberculosis is more difficult than that of the advanced case. There is little or nothing that the physician can do with his own hands. Everything depends upon the patient's natural forces of resistance and his ability to avoid future reinfection. Although a great deal of effort and discipline and sacrifice are required on the part of the patient, the disease in most cases seems to the patient hardly severe enough to warrant such expenditure. It is frequently too difficult to make the patient believe that the condition has serious significance and, therefore, it is difficult to secure the necessary cooperation on his part.

Our treatment must have aim, otherwise we can never hope to reach our goal. Brewer[4] has outlined the objectives of therapy, in a condensed fashion. They are as follows:

1. As the patient is likely to have his problem to face for many years to come, possibly for the rest of his life, his outstanding need is to understand his problem himself as thoroughly as the physician does. The treatment of these cases is an education; the physician is a teacher, and his duty to the patient is to provide a course of training.

2. Tuberculosis is the penalty paid by the human race for its civilization. Even in cases inwhich a clinical cure is possible, if the patient is then sent out to face the

same situation that causes his disease in the first place, he will pay the penalty again. The physician must seek out the faulty link or links in the patient's usual environment and must help the patient adjust himself to an environment without these.

3. There is no method of fighting tuberculosis. The physicians only hope lies in the bodys' natural defenses. His efforts are directed toward giving these an optimum chance; possibly also in stimulating them to increased activity, though the latter possibility is not admitted by everyone.

4. The physician ought to take advantage of every bit of bright prognostic evidence, and extend to the patient as much hope as possible. The hope of recovery is one of the most powerful of therapeutic agents.

5. The patient may have symptoms which annoy him greatly. It is important to relieve these symptoms because an increase in comfort means decrease in stress and stress is the chief obstacle to recovery; also, because the psychic encouragement derived from the relief of a distressing symptom quite inconsequential im its pathological origin is a powerful help toward holding the patient to his prescribed regimen and giving him valuable hope.

Brewer[4] next outlines the therapeutic thesis that every physician should keep in mind when treating tuberculosis patients.

1. It is necessary that the physician, in his initial announcement of the diagnosis, avodi conveying to the patient a distorted idea of the disease and the situation. The picture that arises in the public mind when tuberculosis is mentioned corresponds to the condition medical men knew it fifty years ago. To them it means a 'consumption', the advanced stage of the disease, with a hollow cough, a wasted body and a hopeless prognosis. To say to a person who has that conception of the term, 'You have tuberculosis' is equivalent to telling him, 'You are sentenced to be shot at sunrise.' While it is necessary to avoid frightening the patient, on the other hand the matter must not be made light of. The patient must be filled with respect for his diagnosis. He must be made to understand that he has a difficult course of treatment ahead of him and that consequences of the most serious nature will result from neglect or carelessness.

2. It is necessary that the patient be made to grasp clearly the general conception or rationale of treatment. It is essential to explain clearly that there is no specific treatment for tuberculosis, no way of 'killing the germ', that the only hope lies in building up and that the physical condition is built up by nutrition and relief of stress. Nutrition involves food, water, air and light; While the relief of stress involves both mental and physical factors, the discovery and correction of all physical defects, and the relief of as much symptomatology as possible.

3. The patient must be made to understand with absolute clearness that his condition has reduced his capacity. His recovery can come only at the price of an adjustment to his reduced capacity.

4. The utmost effort ought to be made, by a careful study of the patient's past history and present situation, to determine the precipitating factor responsible for the onset of the patient's present condition. When this can be found, its correction is the principal step toward recovery.

5. It is absolutely essential to secure the patient's thorough and willing cooperation. Unless he can under-

stand the reason for every step, unless he is enthusiastic about it and heartily in accord with it, he will pursue it half heartedly and without benefit.

a. A thorough understanding on the part of the patient of what he is expected to do and why it must be done is essential to his cooperation.

b. The patient must be impressed with the physician's understanding and knowledge of the subject. There is only one way for the physician to make this impression effectively and that is actually to know the subject thoroughly.

c. A constant check must be kept on the patient by means of periodic visits, examinations, and follow-up questions. The physician should keep the patient posted on his progress, point out to him what items of the regimen have helped him improve and explain cause of failure to improve and cause of relapses. It is necessary to emphasize to the patient constantly that it is his own efforts that are causing his improvement and that everything depends on himself.

6. About half the battle lies in keeping up the patient's morale.

a. The patient must be made to believe that it is possible for him to get well.

b. It is necessary that the desire to get well be aroused and maintained in the patient.

c. The patient must understand thoroughly how he can get well.

d. The problem of morale furnishes an additional reason for checking up the patient frequently and keeping full and accurate records.

e. The physician's personality is, of course, an essential factor in maintaining the patient's morale.

f. One of the most difficult as well as the most necessary tasks of the man who treats these cases is to convince the patient that the treatment is going to require a long time." 4

The patients having acute exudative tuberculosis fall into three

groups:

1. The thin and undernourished, weak and asthenic type, which progresses favorably under rest and nutrition, the latter being the principal problem in this type of case.

2. The fat, well nourished patient, who is weak, febrile and toxic. He is hard to convince that he has tuberculosis and nutrition is not the principal problem. His manifestations are often largely allergic and many of these people improve quite satisfactorily under tuberculin treatment.

3. The patient, either thin or well nourished, with severe reflex symptoms. Sedatives of the antispasmodic, analgesic and hypnotic type help these patients considerably.

The above are the general measures that one must follow in the treatment of acute exudative tuberculosis. But what are the most specific measures to be followed? Ornstein and Ulmar[16] have divided the acute forms of pulmonary tuberculosis into three types, as follows: 1. The exudative type. 2. The exudative productive type, and 3. The caseous pneumonic type. In the exudative type of tuberculosis Ornstein and Ulmar[16] say that there should be no interference with the normal return to status quo by any operative procedure such as pneumothorax, and that these patients must be protected from reinfection. They also advise

bed rest until all trace of the lesion has disappeared. This usually means a period of six weeks to three months. "Any form of therapy in this group would have been successful. This miracles usually explained by phrenicectomy belong to this group."[16] This treatment is also advocated for the exudative productive type of acute pulmonary tuberculosis.

In the acute stage of the caseous pneumonic type of acute pulmonary tuberculosis, there should be no interference. "It is only after the temperature has subsided and the sloughing has taken place that any form of compression therapy should be attempted. Lung compression and mot lung rest should be the aim. If pneumothorax cannot be induced because of adhesions, or fails because of persistently positive sputum, a thoracoplasty or apicolysis must be tried. The index of success is the disappearance of tubercle bacilli from the sputum. Phrenic neurectomy is useless."[16]

Certainly the treatment in all cases of acute exudative tuberculosis needs more than just the simple measures of treatment as mentioned above. Let us look into some of these measures more carefully as follows:

1. Rest. This is important in order to check the spread of the disease and for this purpose rest is essential. By resting the diseased part, the lymph flow is impeded and the disease becomes localized.[5] It exerts a beneficial effect on the patient by aiding him in putting up a more efficient defense, and on the disease process by causing a minimum amount of toxins to be given off from the active foci. Psychical rest

must also be maintained. The adjustment of the patient to his disease and to his social, economic and domestic problems are just as necessary a part of his rest treatment as the adjustment of his physical machine to the demands made for the natural body repair and those made by the disease. While prolonged bed rest is necessary during the acute phase, it does not follow that it is the most desirable state for maintaining physiologic balance in a healthy person. Exercise in the normal healthy man increases muscle tone, strengthens heart action, increases appetite, favors digestion, produces normal tiring, stabilizes the nervous system and promotes sleep. It is desirable for the patient to change from his passive condition of continuous forced rest to that of a carefully prescribed amount of activity, just as soon as conditions will permit, but not before.

2. Fresh Air. The benefit that is to result is not from putting the patient in the open air, but from the reaction on the part of the patient when placed in the open air. Fresh air is a stimulant to the skin and temperature is a stimulant to metabolism. "Air carries not only oxygen and carbon dioxide, but also water vapor and it is always in motion when unobstructed. Air is cleansed and purified by movement. Stagnant, confined air contains a much higher bacterial content than open air. Then, too, the bacteria live much longer in the stagnant air of the closed room than in the open, for in the latter the wind movement keeps the organisms moving and the sunlight destroys them."[22]

3. Nutrition. Undernourished patients must gain weight. It has

been stated that fat eaters are much less liable to become tuberculous than are those who dislike fat. Fatty foods, such as milk and cod liver oil, should be encouraged as much as is possible. The diet must be well balanced and contain the necessary amount of proteins, carbohydrates, fats and vitamins. During the acute febrile stage the diet should be as generous as is consistent with the patient's powers of digestion. Milk is often well tolerated, but may lead to diarrhea with offensive stools in which case a more solid diet should be given and milk stopped. Salts in sufficient amount must also be included in the diet. The presence of salts of sodium potassium and calcium are essential to activity of the cell. Iron, phosphorous, iodine and copper are also essential to the animal economy.

Sauerbruch, Hermannsdorfer and Gerson have proposed a salt free diet. This diet has been proved to be of definite value in the treatment of skin tuberculosis. Recently this diet has been used in the treatment of pulmonary tuberculosis. The purpose of this diet is to withdraw the chlorine and dehydrate the tissues and at the same time flood them with vitamins A and D, through foods and the administration of cod liver oil and phosphorus. Sodium chloride is withheld from the diet. They diet which they recommend consists of about 3,000 calories per day, made up of about 90 grams of protein, 1600 grams fat, and 220 grams carbohydrate, which is taken salt free. This diet is to be seasoned with a salt mixture known as "Minerologen", which consists of calcium, aluminum, strontium, bismuth, sulfate, silicate, potassium, sodium, magnesium, bromine, phosphate and lactic acid. Each patient is advised to eat from three-

fourths to one pound of raw fruits and 100 grams of raw vegetables per day. "The great difficulty with this diet is its lack of the salt flavor, which has its one drawback."

4. Gold Therapy. Sanocrysin is a double thiosulphate of gold and sodium and is valuable in the treatment of acute exudative tuberculosis. This drug is best administered in sterile normal salt solution and is given intravenously. Sanocrysin brings about a rather sudden cessation of symptoms and shortens the convalescent period. There is a decrease in sputum and disappearance of bacilli, a favorable change in the blood proteins and a marked stimulation in fibrogenetic tissue. "Although there is no doubt a slight bacteriostatic effect that cannot well be demonstrated, the principal effect seems to be the indirect one as a stimulant to the reticuloendothelial system and fibrogenetic tissue."[10]

5. Calcium. This has been advocated in the treatment of tuberculosis. Calcium is given in the form of collosol calcium subcutaneously or intravenously. Even with daily intravenous doses of 5 c.c. of a 10% solution, there is usually no change in the condition of the patient suffering from acute exudative tuberculosis.

6. Sodium Morrhuate. This is a sodium salt of cod liver oil, which has been advocated in the treatment of acute exudative pulmonary tuberculosis. It is given by subcutaneous injections in doses of from 0.1 to 1 c.c. of a 3% solution. There is no evidence of any more gain in weight in patients receiving this treatment than in those who have not received sodium morrhuate.

7. Sanatorium Treatment. It may be said that there are two main factors in sanatorium treatment: a. To teach the patient how to live under his altered circumstances, and b. To train the body to as high a condition of physical fitness as possible. Of the many factors which go to make up this treatment may be mentioned rest, exercise, fresh air, diet, routine life with regular habits, treatment of symptoms, etc. A patient in a sanatorium is encouraged to ask, "How well can I get", rather than "how soon can I get home".[5]

8. Climate. A great deal of harm has been done by the failure to appreciate that the best climate for a patient varies according to the individual and to the stage of the disease. A patient will often have a fixed belief that if he goes to a certain place he will be cured. The truth is there is no place that is good for every patient with tuberculosis, and how a patient lives is far more important than where he lives. Contentment and reasonable comfort are essential and the patient must also be able to afford to stay a sufficient time and have the necessary medical supervision. Acute cases are better in an institution at home where they can be under medical supervision and any complication or new development in the disease can have the appropriate treatment. There should be good ventilation with an abundance of fresh air, but it is not necessary for the patient to be uncomfortably cold and some patients, especially those who are thin or have poor circulation, may suffer from too much exposure to fresh air. In this acute stage it is usually a mistake to send the patient abroad and he should certainly not be sent to a high altitude. For the patient with acute exudative tuberculosis,

the important question is, "How shall I live?" not "Where shall I live?"

SURGICAL TREATMENT

If rest under the best conditions does not create a tendency to resolution or healing of the exudative process, other means must be tried. The only other treatment we have available at the present time is surgery. Surgery has as its main purpose relaxing and cmmpressing the lung. By so doing, the oxygen is reduced in the diseased area and the carbon dioxide and pH is increased. The aim of all surgical procedures is:

1. To relax or compress the diseased pulmonary tissues so as to put the lung at relative rest by relieving it of the movement incident to respiration, thus reducing the danger of reinoculation and metastases.

2. To lessen the flow of the circulatory blood and lymph in the infected area; reducing the harm from the absorption of toxins.

3. To remove tension so that less strain will be put upon the healing tissues, thus facilitating relaxation of the pulmonary tissues and approximation of the walls of cavities, should they be present.

The surgical procedures are:

1. Induced pneumothorax. Artificial pneumothorax consists in the introduction of air or some other gas between the parietal and visceral layers of the pleura. A pleural pocket is formed, and eventually the lung is collapsed, thus being put at surgical rest. In favorable cases the activity of the disease is quickly brought under control. "Probably

one of the real advantages of pneumothorax as compared with other surgical procedures in the treatment of the preponderantly exudative type of lesion is an immunologic one based on the fact that this particular type of pathology is found in patients who are building up an immunity, but who have not yet attained it. Patients with early exudative lesions react to every new reinoculation of bacilli or bacillary protein more markedly than those who have their immunity better and more completely established, as is evident from the comparatively lesser degree of reaction which they show when the disease becomes more extensive and of longer duration."[22]

Pneumothorax helps greatly in the treatment of acute exudative tuberculosis in preventing the lesion from spreading to the contralateral lung. Bilateral compression is usually not recommended in the treatment of acute exudative tuberculosis, because the contralateral lung is involved secondarily by means of bacillary protein which gains access to the blood stream from foci in the more seriously involved lung. The exudative reaction in the contralateral lung will clear up when the foci in the other lung from which the reinoculations are taking place is compressed.

There are certain dangers of incuding pneumothorax which should be mentioned. These complications are - gas embolism, pleural shock, puncture of the lung, puncture of a large blood vessel or of the heart, surgical emphysema, pain, dyspnoea, displacement of the mediastinum, pleural hernia, febrile reactions, infection of the track of the needle, perforation of the visceral pleura, thickened pleura and tuberculous pleurisy.

2. Operations on the phrenic nerve. The clinical results from phrenic paralysis are notoriously unpredictable. In some cases expected success is not realized, while in others healing of the lesion occurs when only improvement had been anticipated. This operation relieves tension from the tissues and allows them to relax. Unfortunately this operation reduces the patient's vital capacity from 1/8 to 1/6. In order to understand the effect of diaphragm paralysis, one must remember that anything that reduces the size of the thoracic cage must effect the entire lung.[22]

There are two types of phrenic operations, one is the temporary phrenic, in which the nerve is merely crushed or cut or partially excised amd the permanent phrenic, in which the nerve is resected or evulsed and is then unable to regenerate. The temporary phrenic may be used in the treatment of acute exudative tuberculosis, but the permanent phrenic should not be used until the patient is given an adequate amount of bed rest and pneumothorax, for there is always a possibility that the exudative process will resolve itself.

Stanbury[26] has made a complete anatomic study of the diaphragm and viscera following section of the phrenic nerve. His work shows that atrophy of the diaphragm is evident as early as the third week after section of the phrenic nerve and is complete by the fourth month. After paralysis one-half of the diaphragm is elevated and eventrated into the thorax. With stretching it becomes a thin whitish membrane of parchment-like thinness. There is also marked distortion of the abdominal viscera

following phrenicectomy. This may be due to the increase in the size of the abdominal cavity. The readjustment of the viscera may produce a tension and even torsion of their mesenteric attachments. Visceroptosis is also one of the complicating sequelae to paralysis of the diaphargm. All these facts should be considered before a permanent phrenic is done.

The chief objection that has been offered against the temporary operation is that the period of paralysis is highly variable and that usually it is too short to have the desired effect on the lesions, but a second operation may readily be used to paralyze the diaphragm permanently or temporarily, according to the effect of the initial operation.[1]

"In the light of our experience it would seem that there is a distinct indication for a temporary crushing operation as a trial procedure in a more or less acute spreading, unilateral disease where there is reason to suspect poor resistance and a liklihood of the contralateral lung becoming involved."[6]

3. Thoracoplasty. This operation consists in the resection of the ribs for the purpose of producing collapse of the lung. Brown and Sampson speaking about the treatment of the acute exudative phase of tuberculosis, especially in girls and negroes between the ages of 12 and 19 years, say "As artificial pneumothorax has often so greatly increased the chances of recovery in such patients, it seems justifiable, if pneumothorax cannot be produced, to resort to thoracoplasty, in hopes that extension to the opposite lung may be prevented and that the collapse produced may act in a way similar to that brought about by artifi-

cial pneumothorax."[3]

In summarizing the treatment of acute exudative tuberculosis, it is important to remember that: "All patients with the exudative type of disease should be considered to be in a more serious, a more rapidly changing, a more acute condition. In a certain number the condition improves very rapidly, but in others it progresses just as rapidly. Hence it is highly important to watch these patients more closely than those with proliferative disease. Cavities frequently appear with startling rapidity and may enlarge very suddenly. On account of the rapid changes that often occur in patients between the ages of 12 and 19 especially in girls it is well to place such patients under this head. For similar reasons all negro patients fall into this group. In this group artificial pneumothorax is the operation of choice. The phrenic operation is often of little aid."[3]

Exudative lesions should not be collapsed simply because they are exudative and because the patient is having fever from which he is not relieved after a few weeks of sanatorium treatment. If the patient is holding his own and the disease is not showing any great tendency to spread, and he is having only the symptoms that must be expected from the condition present, we infer that he is building up a satisfactory defense which will probably in time enable him to overcome his present condition.

"Early exudative lesions, with or without cavity, according to our experiences, will usually heal by resolution and proliferation if treated by the usual hygienic-dietetic regimen, plus weights to the chest

wall and tuberculin, in from one year to eighteen months. This is a much shorter period than is required for bringing about a result by pneumothorax."[22]

A SOCIAL AND ECONOMIC PROBLEM

Tuberculosis and civilization go hand in hand. It is not a new disease; it is as old as civilization itself. Egyptian mummies have shown that tuberculosis existed as far back as 1600 B.C. Even Chinese history dating back to 550 B.C. mentions it. In the course of years, it has taken more lives than any other disease. It has challenged youth; it has fought youth and it has won the battle. Tuberculosis has been the most deadly weapon that civilization has had to contend with. Youth today stands up and demands that something be done, and something must be done to help youth.

In the fifty-six years since Koch discovered the tubercle bacillus methods for combating tuberculosis have crystallized into a fairly well standardized program, the basic elements of which are control of the carrier and education of the public. Control of the carrier has always been difficult because the disease is of long standing and often not manifest, and also because case reporting and follow-up have never been fully achieved.

Laws requiring the reporting of tuberculous cases are premised on the assumption that the tuberculous individual sooner or later consults a physician. But early cases of tuberculosis do not often come to the attention of the physician. However, our more recent knowledge of early tuberculosis in children opens up a new approach to the problem, and at least suggests means of supplementing the accepted measures which have been employed in the past.

Searching for tuberculosis among apparently healthy children is valuable from two standpoints, namely the individual welfare of the child and the protection of the public health. Obviously, when a child is found to be infected a careful investigation of his environment is indicated in order to discover if he is living in contact with an open or a suspicious case. By breaking that contact a great service is rendered the child. Furthermore, much can be done to build up his resistance and to train him in proper health habits.

Quite as important is the utilization of a case of first infection or of childhood type of tuberculosis as a clue by means of which we may trace the whereabouts of active cases of adult type of tuberculosis. In the control of tuberculosis, the household and not the individual patient is the unit. Having knowledge of a case of tuberculosis it becomes our obligation to search for the source of infection. The search often leads to an obvious open case which menaces the entire community. A systematic search for tuberculosis is, therefore, valuable as a comprehensive case-finding measure. The most simple and most satisfactory plan for discovering tuberculosis among children seems to be to make tuberculin tests of all children of a given group and to examine all positive reactors with the x-ray. Two circumstances, the expense of making x-rays and the objection of some parents to the tuberculin test have hindered the more rapid development of group case-finding among children.

Early diagnosis is essential in the eradication of tuberculosis and in the treatment of the patient, and yet the physician who recogniz-

es the early case of tuberculosis and who comes into a community with the intention of treating pulmonary tuberculosis as such and telling the truth about it is not going to have smooth sailing. He is going to be disbelieved by patients and laughed at by confreres and considered a monomaniac by both.

The man who has the correct idea of the early case and is able to recognize it will, if he comes into a community that already has a good medical personnel, see an astonishing number of cases. The early cases go the rounds. They have tonics and surgical operations and diagnoses. The real "appendices" and heart cases and peptic ulcers are filtered out by competent men before they reach him. The genuine cases of tuberculosis, because of their chronic nature, does not respond permanently to tonsillectomies or to treatment of some sort, and they eventually find their way to him, one and all.

"At the present time the greatest obstacle to the eradiaation of tuberculosis lies in the mental attitude of the public, both lay and medical, toward the disease. Making the diagnosis is a comparatively simple task compared with that of telling the patient and his relatives about it. The associations of the word tuberculosis in the popular mind constitutes a vague though powerful and terrifying complex, founded on the medical profession's conception of the disease of forty years ago. They include cachexia and fever, a hollow cough, germs in the sputum, infectiousness, hopelessness, if these are not present, the case is not tuberculosis. Tuberculosis, in the popular mind, is of a different order from other diseases. The name strikes into people a qual-

ity of terror and hopelessness that no other disease carries with it, except possibly syphilis. People who would readily accept a grave verdict of heart disease, Bright's disease, or peptic ulcer, resist a diagnosis of tuberculosis, unreasonably and desperately. Even though one assures them that a diagnosis of early tuberculosis carries with it far more hope of recovery and health than the others, they continue to tremble and weep in terror at the word 'tuberculosis'.

"In their unwillingness to accept the terrible stigma of tuberculosis, the early cases go from one physician to amother to find one whose diagnosis will have a more pleasing sound, and in the hope that they can get away from the dread condition by denying its existence. They believe statistics; when a lecturer or a magazine states that tuberculosis is so common that one person in ten in their community is diagnosable as tuberculosis of some type, they accept it. But if you tell John Smith that he is one of the ten per cent, that is impossible. The attitude that I have observed in the majority of laymen toward tuberculosis is classifiable phychologically with dragons, sea-serpents and Dante's Hell and is quite as firmly fixed.

"The great white plague cannot be tamed until we reach such a state of affairs that the first medical man who sees the early case can recognize it, and either treat it or refer it for treatment."[4]

Recent studies have shown that the incidence of infection is less than it was twenty-five years ago. Now about thirty five per cent of individuals are infected by the age of fifteen years. Negro children

between the ages of 5 and 15 years show only a slightly higher percentage of infection than white children, but their tuberculosis death rate is six times as high. The Negro tuberculosis death rate is generally speaking two and one-half to three times that of the whites. According to conditions prevailing in 1930, out of every 1,000 males born, 42.5 will eventually die of tuberculosis in the white population and 96.7 im the colored population.

Let us stop for a moment and consider the negro and tuberculosis. If there is no lack of response to primary infection, if there is an immunity conferred on the negro child by having had this infection, we must look further to find the essential differences in the life of the negro child which causes him to succumb later to the malady. When we consider the various influences, there is one outstanding environment. It is readily conceded that the location, sanitation, and the size of the average negro dwelling does not compare favorably with the most humble provided for his white neighbor. We know that the wage scale does not offer favorable comparison. We also know that the foods furnished are not those necessary to healthful living. In most negro districts in the South, the greatest average consumption of milk per person is two pints per week. Milk, eggs, red meats and vegetables form a negligible part of the dietary of the rural and urban negro. The average family subsists on a diet of sweet potatoes, turnips, greens, corn bread and salt meat. How can strong bodies be built from this kind of a diet? Unfortunately the negro has a decreased sensitivity to pain symptoms and does not consult a physician until the symptoms are severe and the di-

sease is far advanced.

The tuberculosis death rate has declined in the United States from 202 per 100,000 living persons in 1900 to 60 in 1933. In 1933 there were in the United States 74,836 deaths from tuberculosis. Deaths from pulmonary tuberculosis constitute about 90% of all deaths from tuberculosis. In 1900 more than three times as many people relatively died of tuberculosis as in 1933. The tuberculosis death rate has continued to decline throughout the depression years. The reduction in the tuberculosis rate since 1904 is equivalent to a saving every year of 175,000 lives.

Tuberculosis is the leading cause of death between the ages of 15 and 45 years. The tuberculosis death rate among infants is two-thirds as high as the rate in the general popylation. Forty per cent of the tuberculosis deaths among infants are due to tuberculous meningitis.

Males in the United States have a higher tuberculosis death rate than females. The tuberculosis death rate for young females is declining more slowly than that for young males. Among females the tuberculosis death rate in 1932 was highest between the ages of 20 and 30 and in the years beyond 70. Among males they were highest between the ages of 50 and 80. In the age group of 15-24 years, the death rate for females is one and one-half times that for males. Recent studies of tuberculosis among young women seem to discredit the idea that the higher death rate among them is attributable to their increasing industrialization. The desire for a slender figure, flimsy dress, cigarette smoking or excesses of the "jazz age". It seems more probable that biological factors play more significant part than environmental influences. The higher death rates among

young women suggest that a more intensive effort should be made to find cases, through tuberculin testing and x-raying, especially in prenatal clinics, industrial groups, schools, colleges and nurse training schools.

Although heart disease and other diseases such as cancer, nephritis and cerebral hemorrhage are responsible for more deaths than is tuberculosis, well in excess of 50% of such deaths occur after the age of 60 years. Tuberculosis takes its greatest toll between the ages of 15 and 45 years, the most productive period of life.

About 60% of all deaths from tuberculosis occur during that period of life from 15 to 45 when a person is of greatest economic value. Tuberculosis cuts off an average of one and one-half years from the complete expectation of life of the people of the United States. Tuberculosis costs the United States government more than $46,000,000 in 1932 for service connected compensation alone. The sum of $400,090,176 is the total paid out in compensation to World War veterans afflicted with tuberculosis for the period 1923 to 1932. This is 33 1/3 % of the total amount paid on compensation and it does not include hospitalization.

There are in the United States now more than 650 sanatoria with more than 86,000 beds. There are more than 7,000 public health nurses engaged in tuberculosis work. There are 3,600 traveling and permanent clinics for the free diagnosis, treatment and education of adults and children. At least 1,000 open air schools and classes, more than 100 preventoria and as many summer camps are provided for children who are physically sub-standard or were in contact with tuberculosis.

Only twenty states in the United States have reached the National Tuberculosis Association's standard of one bed to each annual death from tuberculosis. These beds are distributed as follows: In public or tax-supported institutions (including federal, state, county and municipal, 68,371 beds; in private institutions (those supported only by fees of patients) 4,344 beds; in semi-private institutions (those that are not included in the other two groups), 14,202 beds. Altogether it is estimated that 1,371,000 persons have been admitted to all civilian sanatoria in the United States from the time they opened im 1885 through 1931. The total amount invested in civilian tuberculosis sanatoria andhospitals is approximately $212,535,000. The average cost of maintenance per person is $17.50 weekly. The total amount of yearly maintenance im the United States is $58,000,000. The average length of stay in a sanatorium is six and one-half months. Sixteen per cent of the patients admitted to civilian sanatoria with pulmonary tuberculosis are in the minimal stage, 30% moderately advanced and 54% far advanced. Of sanatorium beds in the United States, 79% are public or tax supported, 5% private (for profit only) and 16% semi-private (all not included in the two other designations.[14]

Tuberculosis is a communicable, hence preventable,disease. It is not incurable. This disease should be added to the list of diseases that can be successfully approached in the schools. Control of the infection before it has undermined health gives promise of preventing the disease in childhood and consequently in early adult life, when it is most frequent. Every case of tuberculosis comes from another and every case may lead to another.

BIBLIOGRAPHY

1. Alexander, John: Temporary Phrenic Nerve Paralysis. Jour. Amer. Med. Assn. CII, 1934, page 1552.

2. Blacklock, John W.S.: Tuberculous Diseases in Children, His Majesty's Stationery Office, London, 1932, page 46.

3. Brown, Lawrason and Sampson, Homer: A Tentative Working Classification to Facilitate the Treatment of Pulmonary Tuberculosis. Amer.Jour. of Med. Sc., CIXC, 1935, page 325.

4. Brewer, Miles J.: Thoughts on the 'Early Case' of Tuberculosis. Amer. Rev. of Tuberculosis, XXI, 1930, page 151.

5. Burell, L.S.T.: Recent Advances in Pulmonary Tuberculosis. Blakiston's Son and Co., Philadelphia, Pa. 1929.

6. Decker, H. Ryerson: The Results of Phrenic Nerve Operations in 222 Cases. Jour. Thor. Surg. II, 1932, page 538

7. Douglas, Bruce H. and Pinner, Max: Acute Subapical versus Insidious Apical Tuberculosis. Amer. Rev. of Tuberc. XXI, 1934,p. 305.

8. Douglas, Bruce H. and Pinner, Max and Wolepar, Ben: Acute Subapical versus Insidious Apical Tuberc. Am. Rev. of Tuberc. XIX, 1928, page 153.

9. Goldberg, B. and Gasul, B.M.: Epituberculosis. Amer. Rev. of Tuberc. XXIV, 1931, page 285.

10. Henricksen, Karl and Sweany, Henry and Hruby, Allan: Sanocrysin Treatment in Tuberculosis. Amer. Rev. of Tuberc. XXVIII, 1933, page 1.

11. Hudson, Bernard: The Modern Treatment of Pulmonary Tuberculosis. Jonathan Cape Co., London, 1930.

12. Krause, Allen K.: The Evolution of Tubercle. Jour. of the Outdoor Life. New York, 1927.

13. Krause, Allen K.: Immunity and Allergy in the Pathogenesis of Tuberculosis, Tubercle, 1928, page 1.

14. National Tuberculosis Association Statistics.

15. Ornstein, George and Sampson, Homer: The Determination of Clinical Activity in Pulmonary Tuberculosis from Roentgenograms. Amer.Rev. of Tuberc. V, 1921, page 842.

16. Ornstein, George and Ulmar, David: The Treatment of Acute Forms of Pulmonary Tuberculosis. Amer. Rev. of Tuberc. XXVIII, 1933, page 484.

17. Ornstein, George and Ulmar, David and Dittler, Edgar: Benign Acute Pulmonary Tuberculosis. Amer. Rev. of Tuberc. XXVIII, 1931, page 223.

18. Ornstein, George and Ulmar, David and Dittler, Edgar: A Clinical Classification of Pulmonary Tuberculosis. Amer. Rev. of Tuberc. XXIII, 1931, page 248.

19. Petroff, S.A.: Immunology of Tuberculosis. The Cyclopedia of Medicine, Piersol.

20. Pinner, Max: Modern Concepts in the Pathology of Tuberculosis. Amer. Rev. of Tuberc., XVII, 1928, page 601.

21. Pinner, Max and Kasper, J.A.: Pathological Peculiarities of Tuberculosis in American Negro. Amer. Rev. Tuberc. XXVI, 1932, page 463.

22. Pottenger, Francis Marion: Tuberculosis in The Child and the Adult. The C. V. Mosby Company, St. Louis, 1934.

23. Pottenger, F.M.: Early Diagnosis of Tuberculosis. Amer. Rev. of Tuberc. XXI, 1930, page 159.

24. Pottenger, F.M.: Exudative and Proliferative Processes. Amer. Rev. of Tuberc. XVIII, 1928, page 580.

25. Rich, Arnold and Mc Cordock, Howard: An Enquiry Concerning the Role of Allergy, Immunity and other factors of importance in the Pathogenesis of Human Tuberculosis. Bull. of Johns Hopkins Hospital, XLIV, 1929, page 273.

26. Stanbury, W. Stuart: Anatomical Changes in the Diaphragm Following Phrenicectomy. Amer. Ref. of Tuberc. XXIX, 1934, page 528.

27. Zinsser, Hans and Mueller, J.H.: Nature of Bacterial Allergies. J. Exper. Med. XXXXI, 1925, page 159.

Approved by

W. E. Evans

Professor of Medicine

Date

February 20th, 1936